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ABSTRACT

Objective: Gestational hypertension (GH) and mild preeclampsia (PE) represent the most common medical complications of pregnancy, with the majority of cases developing at or near term. There is little knowledge of the course of blood pressure over time in these women. We explored the pattern of systolic and diastolic blood pressure over time in women with GH or mild PE at term participating in the HYPITAT trial, and we attempted to identify clinical factors influencing these blood pressure patterns and the impact of severe hypertension on clinical management.

Study design: We used data from the HYPITAT trial, that included women with a singleton pregnancy with a fetus in cephalic position between 36 and 41 weeks of gestation with the diagnosis of GH or mild PE. Blood pressure measurements were performed from randomization or admission until delivery or discharge from the hospital. We included the highest blood pressure of each day.

We evaluated systolic and diastolic blood pressure change over time, as well as the influence of clinical characteristics and laboratory findings on the course of blood pressure. We used univariate and multivariate regression analysis with a backward stepwise algorithm for the selection of variables. The model with the best fit (lowest AIC) was selected as the final model. We also compared mode of delivery for women with and without severe hypertension.

Results: We studied 1076 women who had 4188 blood pressure measurements done. The systolic blood pressure showed a significant non-linear increase over time and for the diastolic blood pressure the pattern was also non-linear. In the multivariable model of systolic blood pressure change over time, nulliparity, ethnicity, systolic blood pressure (at baseline), BMI and LDH at randomization influenced the course of blood pressure. In the diastolic blood pressure model ALT and the baseline diastolic blood pressure had a significant influence. When we explored the association between blood pressure and mode of delivery, it appeared that development of severe hypertension was a risk factor for Caesarean section.

Conclusion: The blood pressure in patients with GH or PE at term showed a non-linear increase with time, which was aggravated by clinical characteristics. Development of severe hypertension was a risk factor for Caesarean section, which may explain the elevated Caesarean section rates in the expectant monitoring group in the HYPITAT trial.

Introduction

Gestational Hypertension (GH) and mild Preeclampsia (PE) represent common medical complications of pregnancy. The majority of cases develop at or near term. And even though these disorders are associated with minimal to low maternal and

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neonatal morbidity and mortality, complications are seen, especially in women who progress to severe disease. The HYPITAT trial showed that induction of labour reduces the risk of clinical progression to a high risk situation compared to expectant monitoring in women with GH or mild PE at term. This reduction occurred without significantly increasing the caesarean section rate and with similar neonatal outcomes [1]. We subsequently evaluated whether it is possible to predict which woman will progress to severe disease (a combination of high blood pressure, severe proteinuria, HELLP syndrome or eclampsia) based on her clinical characteristics. It was possible to distinguish women with a low risk and women with a high risk for progression to severe disease [2]. The systolic and diastolic blood pressure played a role in these prediction models. Since there is little knowledge of blood pressure patterns over time in women with GH or mild PE at term, we wanted to evaluate this in the HYPITAT cohort.

In uncomplicated pregnancies there is a steady decrease in blood pressure up to a mid-pregnancy drop and then it increases up to delivery. In general, a woman's final blood pressure measurement is similar to the blood pressure found early in her pregnancy [3]. In contrast, the blood pressure in women with GH or PE is generally stable during the first half of pregnancy and then increases up to delivery [4]. Several studies have demonstrated that factors such as maternal characteristics and serum markers can predict the occurrence of GH and/or PE and maybe also the course of blood pressure during pregnancy [5–8].

The present study explores the pattern of systolic and diastolic blood pressure over time from randomization to delivery. We also tried to identify factors influencing the course of systolic and diastolic blood pressure in women exhibiting signs of GH or mild PE at term. Finally, we tried to identify whether the course of blood pressure was related to the clinical management (caesarean delivery).

Patients and methods

We used data from the HYPITAT trial, a randomized clinical trial in the Netherlands that was performed between October 2005 and March 2008, comparing induction to expectant management in women whose pregnancy was complicated by GH or mild PE at term (36–41 weeks gestation) [1]. GH was defined as diastolic blood pressure (DBP) ≥95 mmHg measured on two occasions at least six hours apart. Mild PE was defined as DBP ≥90 mmHg measured at two occasions at least six hours apart combined with proteinuria. Proteinuria was defined by local protocol as ≥2+ protein on dipstick, >300 mg total protein in a 24 h urine collection or protein/creatinine ratio >30 mg/mmol. Severe GH or PE (DBP ≥110 mmHg and/or systolic blood pressure (SBP) ≥170 mmHg) was an exclusion criterion as well as proteinuria ≥5 g in 24 h and pre-existing hypertension. Patients were induced within 24 h after randomisation or were treated according to local protocol in the expectant management group. Monitoring of the patients consisted of frequent maternal blood pressure measurements, assessments of proteinuria, laboratory tests and regular assessment of foetal condition.

In the 1153 women who were eligible for the study, blood pressure measurements were performed from randomization or admission until delivery or discharge from the hospital. Some women had more than one blood pressure measurement per day. In these cases, the maximum SBP and DBP of the day were used for the analysis. Because the main objective of the study was evaluation of blood pressure changes over time, we were restricted to using data from women who had one baseline measurement at randomization and at least one follow-up measurement for this analysis. The analyses were performed separately for SBP and DBP.

Data analysis

First we modelled values of SBP and DBP separately over time. In view of the variable timing and number of measurements per woman, the modelling was performed using a repeated measures, linear mixed-effects model to make optimum use of all available data. The dependent variable was blood pressure and the repeated measure was time in days after randomization. The model was specified in terms of random intercept and random slopes for individual participants. To evaluate possible non-linear time course of blood pressure over time, we tested if adding time-squared terms (for more than linear increase) or time-cubic terms (for S-shaped increase) to the model improved the model fit as judged by the Akaike Information Criterion (AIC). In the second step we evaluated whether a series of baseline factors influenced the change of blood pressure over time. The studied factors were maternal age, Caucasian versus non-Caucasian ethnicity, educational level, nulliparity, smoking during pregnancy, the diagnosis (gestational hypertension or preeclampsia) and a series of baseline clinical and laboratory findings at randomization including body mass index (BMI), gestational age, systolic and diastolic blood pressure, serum haemoglobin, creatinin, uric acid, aspartate aminotransferase (ASAT), alanine amino transferase (ALAT), lactate dehydrogenase (LDH), platelet counts and any proteinuria (++ or

Table 1
Baseline characteristics.

Variable	Value n (%)/median (IQR)[a]	Available data n (%)
Clinical characteristics		
Nulliparous	793 (74)	1076 (100)
Maternal age (year)	30.1 (27–33)	1076 (100)
Gestational age (weeks)	38.4 (37–39)	1076 (100)
Previous abortion	254 (24)	1076 (100)
Maternal smoking	119 (11)	1013 (94)
Body Mass Index (kg/m2)	32.3 (28–36)	579 (54)
Ethnic origin		990 (92)
Caucasian	869 (81)	
Non-Caucasian	121 (11)	
Education level		630 (59)
High	220 (20)	
Low	410 (38)	
Blood pressure (mmHg)		
Systolic	143.7 (140–150)	1076 (100)
Diastolic	96.9 (95–100)	1076 (100)
MAP	113.5 (110–117)	1076 (100)
Diagnosis		1076 (100)
Gestational hypertension	733 (68)	
Pre-eclampsia	324 (30)	
Unknown	19 (2)	
Laboratory findings		
Dipstick		877 (82)
Negative	500 (46)	
+	230 (21)	
++	101 (9)	
+++	46 (4)	
Hemoglobin (gr/L)	7.5 (7.0–8.0)	1015 (94)
Hematocrit (SI unit)	0.36 (0.34–0.38)	916 (85)
Platelets ($\times 10^9$/L)	236 (192–275)	1007 (94)
Uric acid (mmol/L)	0.32 (0.27–0.37)	974 (91)
Creatinine (μmol/L)	62.4 (53–70)	956 (89)
Aspartate aminotransferase (U/L)	21.4 (20–25)	835 (78)
Alanine aminotransferase (U/L)	14.8 (10.0–17.0)	869 (81)
Lactate dehydrogenase (U/L)	300 (207–373)	730 (68)
Mode of delivery		1076 (100)
Spontaneously	736 (68)	
Vacuum/forceps extraction	167 (16)	
Caesarean section	173 (16)	

[a] Data are number of patients (%) or median (interquartiel range; IQR).

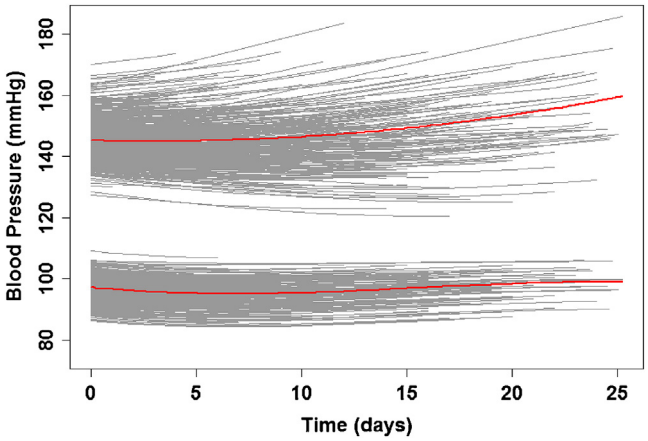

Fig. 1. Pattern of systolic and diastolic blood pressure over time in individual patients. The red line shows the average value. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

Table 2
Modelling of systolic and diastolic blood pressure changes over time.

Blood Pressure	Coefficient	SE	p-value
Systolic			
Time	−0.17	0.11	0.13
Time-square	0.03	0.007	<0.001
Diastolic			
Time	−0.71	0.12	<0.001
Time-square	0.067	0.02	<0.001
Time-cubic	−0.001	0.0005	0.004

SE: standard error.

more protein on a dipstick, >300 mg total protein within a 24-h urine collection, or ratio of protein to creatinine >30 mg/mmol).

In a series of univariate analyses, we studied the relationship between each factor and time course of blood pressure. The models consisted of SBP or DBP as the outcome of interest, the time, time-squared, the prognostic factor, and interaction terms between the factor and time variables. All factors which had a significant ($p < 0.1$) main effect or interaction with time were selected for multivariable modelling with backward stepwise variable selection. The model with highest fit (lowest AIC) was selected as the best model. To illustrate the impact of the interaction effect between time and some of the predictors graphs were created showing the course of systolic or diastolic blood pressure at predictor values corresponding to the 25th, 50th and 75th percentile.

Finally, we evaluated whether women who progressed to severe disease had different intervention rates from women in whom the blood pressure remained stable. To do so, we stratified the women included in the randomized clinical trial into those in whom disease progressed to severe disease, and those in whom disease remained stable, and then assessed the impact of the randomized allocation (induction or expectant management) on caesarean section rates. Progression to severe disease was defined as the occurrence of any of the following: a diastolic BP ≥110 mmHg, a systolic BP ≥170 mmHg and/or proteinuria ≥5 g in 24 h, maternal complications: eclampsia, HELLP-syndrome (platelet count ($<100 \times 10^9$/L and AST >70 U/L or ALT >70 U/L) and mortality.

Statistical analyses were conducted with SPSS version 21 (IBM Corp, Armonk, USA) for Macintosh and R for Windows and for Macintosh (R-Project Software, Vienna, Austria, version 3.1.2)

Results

There were 1153 women who fulfilled the inclusion criteria. Since the main objective of the study was evaluation of blood pressure changes over time, we restricted this analysis to data from 1076 women (99.3%) who had one baseline measurement at randomization and at least one follow-up measurement for this analysis. Overall, 4188 individual blood pressure measurements were included in this analysis. The median number of follow-up measurements per patient was 4 (range 1–56). The maximum follow-up duration was 25 days. Baseline patient characteristics and the amount of available data for these women are presented in Table 1.

Fig. 1 shows the smoothed patterns of systolic and diastolic blood pressure changes over time in the individual patients with

Table 3
Univariate analysis of parameters influencing systolic and diastolic blood pressure.

Variable	Systolic blood pressure		Diastolic blood pressure	
	Coefficient (SE)	p	Coefficient (SE)	p
Clinical characteristics				
Parity: Nulliparous vs Multiparous	0.87 (0.74)	0.241	−0.48 (0.42)	0.25
Maternal age (year)	0.17 (0.07)	0.012	−0.02 (0.04)	0.69
Gestational age (weeks)	−1.10 (0.26)	<0.001	−0.38 (0.15)	0.01
Maternal smoking	0.4 (1.04)	0.70	−0.59 (0.59)	0.31
Body Mass Index (kg/m2)	0.22 (0.08)	0.008	0.02 (0.03)	0.60
Ethnic origin: non-Caucasian vs. Caucasian	0.78 (1.02)	0.45	0.44 (0.58)	0.44
Education level: high vs. low	0.51 (0.88)	0.56	0.33 (0.51)	0.51
Diagnosis: Pre-eclampsia vs. Gestational hypertension	2.30 (0.70)	0.001	−0.29 (0.40)	0.46
Laboratory findings				
Proteinuria	2.35 (0.70)	<0.001	0.37 (0.40)	0.35
Hemoglobin (gr/L)	−1.19 (0.47)	0.011	−0.13 (0.27)	0.64
Platelets ($\times 10^9$/L)	−0.005 (0.005)	0.357	−0.002 (0.003)	0.46
Uric acid (mmol/L x 10)	14.28 (4.49)	0.001	5.31 (2.59)	0.04
Creatinine (μmol/L)	0.020 (0.02)	0.273	0.003 (0.01)	0.75
Aspartate aminotransferase (U/L)	0.096 (0.038)	0.013	0.01 (0.02)	0.54
Alanine aminotransferase (U/L)	0.016 (0.036)	0.653	−0.01 (0.02)	0.56
Lactate dehydrogenase (U/L)	−0.004 (0.003)	0.205	0.0005 (0.002)	0.82

All factors which had a significant ($p < 0.1$) main effect or interaction with time (see Table 4) were selected for multivariable modelling.

Table 4
Multivariable model assessing the factors influencing systolic blood pressure over time.

Systolic blood pressure Variable	Coefficient	SE	p-value
Time			
Time	1.171	0.708	0.098
Time^2	−0.019	0.018	0.304
Clinical characteristics			
Maternal age	0.148	0.070	0.036
Gestational age	−1.084	0.259	0.000
BMI	0.162	0.054	0.003
PE vs PIH	2.096	0.709	0.003
Non-Caucasian ethnicity	−1.209	1.034	0.242
nulliparity	−0.962	0.832	0.248
LDH	0.002	0.003	0.611
Interaction terms			
Time × BMI	0.016	0.008	0.058
Time × Non-Caucasian	0.722	0.191	0.000
Time × nulliparity	0.351	0.138	0.011
Time × LDH	−0.002	0.001	0.046
Time^2 × LDH	0.0002	0.000	0.004
Time × Baseline SBP	−0.011	0.004	0.002

Table 5
Multivariable model for assessing the predictors of diastolic blood pressure over time.

Diastolic blood pressure Variable	Value	SE	p-value
Time			
Time	3.498	0.873	<0.001
Time^2	−0.222	0.056	<0.001
Time^3	0.002	0.001	0.051
Clinical characteristics			
Gestational age	−0.435	0.151	0.004
ALAT	−0.024	0.024	0.314
Interaction terms			
Time × Baseline DBP	−0.037	0.009	<0.001
Time^2 × Baseline DBP	0.002	0.000	<0.001
Time × ALAT	−0.041	0.014	0.003
Time^2 × ALAT	0.007	0.002	<0.001
Time^3 × ALAT	−0.0002	0.0001	<0.001

the smoothed average blood pressure course included as the red line. Overall, the SBP values showed more inter-individual variability than DBP. The mean SBP increased from 145 to 160 mmHg over 25 days, while DBP showed a much milder increase from 95 to 97 mmHg. Table 2 shows the uncorrected modelling of systolic and diastolic blood pressure changes over time. For SBP, there was a significant non-linear increase over time. DBP had a significant positive effect of time-square, but also a significant negative effect of time-cubic. The slightly S-shaped red line for DBP in Fig. 1 reflects the combined effects of time-square and time-cubic. Table 3 shows the results of the univariate analyses for SBP and DBP. All factors which had a p-value below 0.1 or a significant interaction with time were selected for multivariable modelling.

Table 4 shows the result of the multivariable modelling of systolic blood pressure changes over time. The analyses demonstrated that nulliparity, ethnicity, SBP at baseline, BMI and LDH (at randomization) showed interaction with time or time-squared or both, indicating that they influenced the course of blood pressure. Fig. 2 shows the impact of the interaction between LDH and time and time-square on the course of SBP. At high levels of LDH, systolic blood pressure increased more rapidly than at low LDH levels.

Table 5 shows the factors that were selected for the multivariable modelling of DBP. Here, ALAT at randomization had a significant negative interaction with time and time-cubic, and a significant positive interaction with time-square. DBP at baseline also influenced the course of BDP over time, by a significant

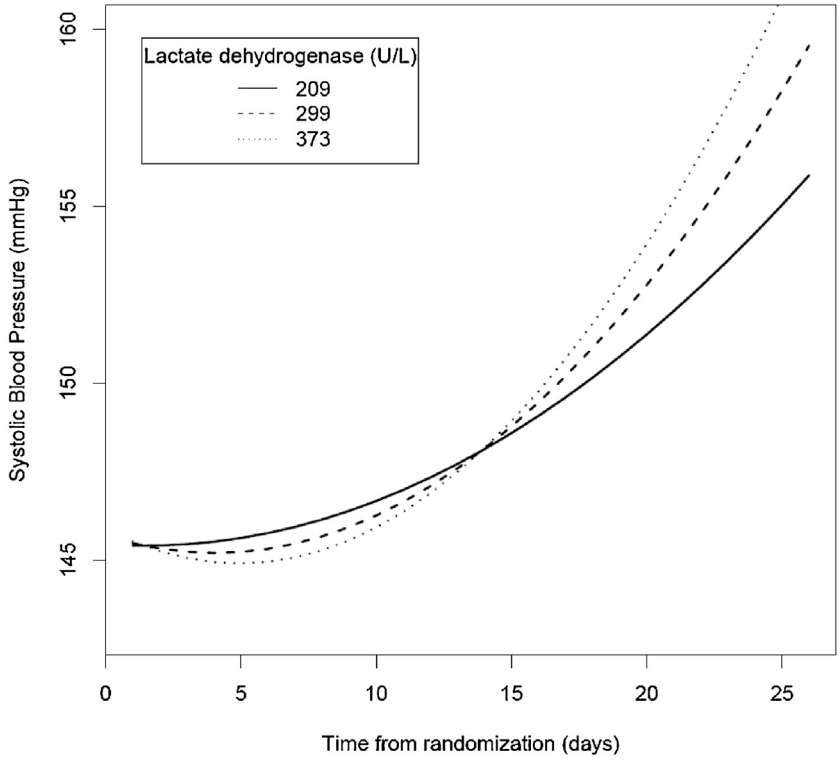

Fig. 2. Course of systolic blood pressure for the 25th. 50th and 75th percentile of the LDH values to illustrate the interaction effect of LDH and time.

Table 6
Intervention rates from women who progressed to severe disease compared to women in whom the blood pressure remained stable.

	CS	No CS	Odds ratio	95% CI
All women	N = 126	N = 630		
Progression to severe disease	64 (28%)	162 (72%)	3.0	2.01–4.42
No progression to severe disease	62 (12%)	468 (88%)		
Patients with progression to severe disease	N = 64	N = 150		
Induction of labour	20 (23%)	68 (77%)	0.63	0.34–1.16
Expectant monitoring	44 (32%)	82 (68%)		
Uncomplicated patients	N = 62	N = 468		
Induction	34 (12%)	255 (88%)	1.01	0.60–1.73
Expectant monitoring	28 (12%)	213 (88%)		

negative linear interaction with time and a positive interaction with time-square.

Finally, we also studied whether women who progressed to a higher blood pressure and/or severe disease had different intervention rates from women in whom the blood pressure remained stable. Table 6 shows two by two tables of progression versus intervention for all randomized women. The CS rate was 28% among women who had progression to severe disease versus 12% among women who did not develop severe disease during pregnancy. We subsequently stratified these data according to induction of labour and expectant monitoring. In both strata, with and without progression to severe disease, there was no impact of induction of labour on the CS rate.

Discussion

In this observational study, we elaborate on the recent findings of the HYPITAT trial, which revealed that labour should be induced in women with mild PE or GH at term [1]. We explored the pattern of systolic and diastolic blood pressure over time in these women, and we attempted to identify clinical factors influencing the pattern of blood pressure. Knowledge of factors influencing blood pressure patterns over time is potentially important in the clinical management of women with GH or mild PE at term. The overall results of the HYPITAT trial resulted in the adoption of induction of labour as the standard of care and an increase in inductions in the Netherlands [9]. The present results might help answering the question whether clinicians should manage all women in the same way.

Variables influencing the systolic blood pressure, causing a significant non-linear increase over time were parity, ethnicity, systolic blood pressure at baseline, BMI and LDH. The diastolic blood pressure pattern was also non-linear with time, which was influenced by ALT and the baseline diastolic blood pressure. Compared with the literature there is an overlap between the parameters influencing blood pressure and risk factors for GH and PE, such as nulliparity, advanced maternal age, multiple pregnancies, diabetes, chronic hypertension, and obesity [7,10,11].

Several studies have described that casual time-unspecified measurements of blood pressure are suboptimal for predicting the development of preeclampsia [3,12]. However, the diagnosis GH or PE still relies on one or sometimes two measurements. Our data showed a difference in the pattern of systolic and diastolic blood pressure. The SBP increased over time, whereas the DBP leveled off after a specific period of time. Given the observational nature of this study, this could be related to the fact that clinicians were more focused on the DBP or that women were induced or got antihypertensive medication.

The fact that we do not have precise data on the use of antihypertensive medication can be considered a limitation of our study, but it is inherent to the observational nature of the study. Another limitation of the study is that we did not have information about the blood pressure devices that were used for the measurements. We assumed that manual blood pressure measurements were performed according to the Dutch guidelines.

In the HYPITAT trial, as in any clinical situation, treatment was allowed and given to a substantial proportion of women. Approximately 25% of women received oral antihypertensive medication after randomisation and 7% received intravenous antihypertensive medication after randomisation. However, the exact moment and dosage of the medication was not recorded, so it was not possible to incorporate medication in our calculations as a time-dependent variable. For SBP with the increasing gradient our data suggest that the increase occurs despite treatment. For DBP with the s-shaped gradient the effect is less clear (Fig. 1).

We also noticed that the CS rate was higher among women who had high blood pressures. This increase can be explained by the disease progression and is not due to the fact that women were induced. We previously described a subgroup analysis of the randomized patients of the HYPITAT trial, showing that an unfavourable cervical examination (dilatation, consistency, position and length) also predicts progression to a high risk situation [2,13]. So even though most clinicians would traditionally choose expectant management in this situation, we can conclude that women with an unfavourable cervix are good candidates for induction of labour. This decreases the risk of progression to a high risk situation without increasing the caesarean section rate. Magee et al. also described this as a result of their CHIPS Trial. They found that lack of control of severe hypertension during pregnancy is a risk factor for adverse pregnancy outcomes [14].

In conclusion, blood pressure pattern in patients with GH or PE at term follows a non-linear course over time and is influenced by clinical characteristics and the baseline blood pressure. Moreover, high blood pressure is a risk factor for caesarean section.

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