Hypertension in Pregnancy Research File

1. What is hypertension?

This could be a very simple or very complex answer.  Hypertension is a condition where the blood flows through the blood vessels with more force than normal.  But what is normal?  What this means as far as diagnosis, let alone risk and outcomes is up for debate. The general diagnosis of hypertension is based on having a blood pressure above 140/90 or being on antihypertensive medication.  One study that had limitations, but was still interesting, showed that mortality for those under 50 years old didn’t increase until the systolic number was about 200 or the diastolic was over 100, and over the age of 50 that changed to a systolic greater than 140.

<http://www.ncbi.nlm.nih.gov/pubmedhealth/PMHT0024199/> - basic definition of hypertension from trusted sources

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3138596/> - very interested article on rethinking hypertension.  the information is admittedly limited and not high quality but the concepts are interesting and point to needing more research in this area.

1. How should blood pressure be taken in pregnancy?

Korotkoff phase V sounds are most precise, so when the sound disappears instead of muffles.  “Controlling Chronic Hypertension in Pregnancy” Sibai

ACOG Practice Bulletin No 29: Chronic Hypertension in Pregnancy - Should use Korotkoff V sounds, a cuff that is 1.5 times the upper arm circumference, patient should be sitting upright or in the left lateral recumbent position, and the reading should be taken after at least 10 minutes of rest with her arm supported at the level of her heart, and should be taken with a mercury sphygmometer ideally rather than an electronic machine.  No caffeine of tobacco in the 30 minutes prior to the reading.

Automatic machines that take measurement without anyone in the room can help reduce white coat effects and observer errors.  The averages of readings taken that way and at 1 to 2 minute intervals correlates well with the mean value while awake with ambulatory readings.  MAJOR issues that I’ve seen very commonly are not waiting long enough for the person to rest before the reading, talking to the person while doing the reading, not supporting the forearm fully and at heart level, and rounding off by large amounts.  There are studies that how shown that 24 hour ambulatory monitoring and home measurement more accurately predict the risk of future cardiovascular events, as opposed to in office readings.  “Taking blood pressure: Too important to trust to humans?”

Blood pressure in the left arm in the right lateral position (this position had the most significant difference) is about 10mmHg or more lower than BP in other positions, like the right arm in a supine position, and this should be noted.  The upper arm in the lateral positions gets a lower reading than that same arm in a supine position.  Using the legs should really not be done.  “Blood Pressure Variation on Each Measuring Site in the Right Lateral Position” - very small study but interesting research.

The center of the cuff should be at the level of the right atrium.  Reading should be done on both arms at the initial visit at least.  They recommend a mercury sphygmomanometer over any automatic device.   The IDEAL cuff should have a bladder that has a length of 80% and a width of 40% of the arm’s circumference.  Arm above the right atrium will give too low of readings and below the right atrium will give too high of readings.  Sitting pressure is about 5 mmHG higher than supine.  Diastolic may be 6 mmHg higher if the back is not supported, and systolic may be 2-6 mmHg higher if the legs are crossed!  The tech needs to palpate the brachial artery and center the bladder over it.  The bottom of the cuff should be 2-3 cm from the antecubital fossa.  The cuff should be inflated 30 mmHG over the point where the radial pulse disappears, and then deflated at 2-3 mmHg per second.  NO ONE SHOULD TALK DURING THE READING.  At least two readings should be taken, at intervals of at least a minute, and then they should be averaged.  It there is a 5 mmHg or more difference between the first two readings another reading or two should be done and then averaged.  - “Recommendations for Blood Pressure Measurement in Humans” - VERY specific and one of the best resources for taking blood pressure.  Not cited well though.

There is debate about using Korotkoff 4 or 5 sounds.  It is the only time the phase 4 sounds are suggested for use since some pregnant women have sounds all the way down to zero, but it is rare (not even once in 197 women in one study).  Since diastolic readings are thusly difficult/controversial in pregnancy, it makes sense that systolic readings should be of more concern, though studies are inconclusive about which number or average, etc, is most predictive of risk.  “How Should Blood Pressure Be Measured During Pregnancy?”

1. How many pregnant women have hypertension?

It is the most common medical disorder seen in pregnancy.  About 70% is due to pre-eclampsia, and about 30% is due to chronic hypertension or renal disease.  It is estimated by the National Blood Pressure Education Program Working Group that up to 5% of pregnancies are affected by chronic hypertension, which is my focus.  About 8% of women have hypertension issues during pregnancy, which includes gestational hypertension and pre-eclampsia as well.

Chapter 5, Sibai - My favorite source.  Well researched, comprehensive, and one of the few that focuses on chronic hypertension alone and therefore has more pertinent information for that specialty/topic.  This author is the leading expert on the topic as well.

Pregnancy induced hypertension (not chronic hypertension), is estimated to effect 6-8% of US pregnancy according to Ribowski in “Pregnancy Induced Hypertension”.

1. What kinds of chronic hypertension are there?

90% of chronic hypertension is "primary" or "essential", meaning there is no underlying

cause that we know of.  The other 10% is secondary, or due to other issues that came first : renal diseases, lupus/collagen vascular diseases, endocrine diseases like diabetes, hyperthyroidism, pheochromocytoma, and vascular disease (aortic coarctation, vasculitis).

Chapter 5, Sibai

1. How is chronic hypertension in pregnancy diagnosed?

It is diagnosed with a documented history of hypertension prior to pregnancy, or persistent elevation of blood pressure on two occasions, more than 24 hours apart, and BEFORE the 20th week of pregnancy.  Elevated blood pressure is defined as being above 140/90.

Chapter 5, Sibai

1. How is chronic hypertension in pregnancy classified?

Mild - traditionally under 160/110.  ACOG redefined as less than 180/110

Severe - over 160/110 or 180/110 depending on the source

Low risk - mild and uncomplicated chronic essential hypertension

High risk - secondary hypertension, severe hypertension, complicating factors in mild hypertension (presence of hypertension for more than 4 years, renal disease, cardiomyopathy, coarctation of the aorta, retinopathy, diabetes class B to F, collagen vascular disease, antiphospholipid antibody syndrome with perinatal loss, previous severe pre-eclampsia with perinatal death)

Chapter 5, Sibai

Pregnancy Induced Hypertension, Jennifer Ribowski

1. What types of hypertension are there in pregnancy?

Chronic hypertension - see above ^^ Greater than 140 systolic and/or 90 diastolic before 20 weeks of pregnancy, or a known history. Either primary/essential or secondary.

Pregnancy Induced Hypertension/Gestational Hypertension - New onset hypertension after 20 weeks of pregnancy (above 140 systolic and/or 90 diastolic); no other features to indicate pre-eclampsia; normalization of BP by 3 months postpartum

Pre-eclampsia - New onset of hypertension after 20 weeks of pregnancy, plus proteinuria (edema is NOT used as a diagnostic characteristic) and other metabolic derangements of varying severity. Normalization of BP by 3 months postpartum

Superimposed pre-eclampsia - Pre-eclampsia symptoms and signs superimposed over pre-existing hypertension, so hypertension prior to 20 weeks of pregnancy.  Proteinuria greater than or equal to 500 mg/24 hours or thrombocytopenia or abnormal liver enzymes.

Pregnancy Exacerbated Hypertension - This is not a common term but I have found mention of this classification.  It is for women who have preexisting hypertension, which is then made worse by pregnancy, but is unrelated to pre-eclampsia.  It is said to be due to a sensitivity to aldosterone and progesterone.

It is not well understood if PIH is a separate entity at least some of the time, or if it is always an early form of pre-eclampsia.  I think there are clear cases where it is not related to pre-eclampsia, but this is why so much more research needs to be done or revisited.  Jennifer Ribowski, “Pregnancy Induced Hypertension”

<http://www.moldiag.com/moldiag/en/id/D0280> - no citation, but from a website that appears to be legitimate and professional/scientific

<http://www.ncbi.nlm.nih.gov/pubmed/10884226> - trustworthy source, one of the few mentions of pregnancy-exacerbated hypertension

“Hypertensive Disorders of Pregnancy” - Very thorough source outlining the types of disorders, their risks, and basic management strategies.

“Controlling Chronic Hypertension in Pregnancy” Sibai

“Pregnancy exacerbated hypertension” Gelle DS - an Abstract about a genetic mutation that causes progesterone triggered hypertension.

1. What about white coat hypertension in pregnancy?

This is absolutely fascinating.  It was found that when BP is measured in the conventional clinical way (in other words, VERY POORLY), a huge amount of the women diagnosed with gestational hypertension actually have benign white coat hypertension (up to 60% possibly) and are normotensive in all other respects (babies are similar in size, rates of pre-eclampsia are similar).  The difference is that women who have white coat hypertension (24 hour monitoring shows normal BP when office visits showed hypertension) were MUCH more likely to have a cesarean, many of which were clearly unnecessary, and were a result of the OB using office data that was flawed (they didn’t know about the 24 hr data).  In one of the studies, 41.1% of true hypertensives had a cesarean, 45.2% of the white coat hypertensives did, and only 12.4% of the normotensives.  The question that lingers for me then is, how many of the true hypertensives had unnecessary cesareans too then, just because of a number on a piece of paper?   “How should blood pressure be measured during pregnancy?” Pickering

1. Are there unique issues for researching hypertension in pregnancy?

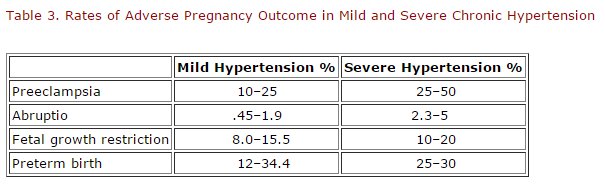
Yes!  Studies are largely unable to sort out the different types, severity, and “chronicity” of the hypertension women have, particularly early onset pregnancy related hypertension versus pre-existing hypertension. **This is comparing apples to orangutans!!!** “Inability to sort hypertensive pregnancies accurately into pathophysiological categories complicates and invalidates the results of many clinical studies of pregnancy and hypertension”.  Rigorous study is also difficult since there is so little understanding of the “natural history” of these issues.  Of 215 studies that one paper looked at, only 46 includes information about the risks of chronic hypertension in pregnancy. The majority of the 215 lacked sufficient definitions about blood pressure, consistency in measurement, failure to account for for confounding variables (smoking, previous pre-e, age, parity, thrombophilic states, renal disease, etc).  Since poor outcomes are relatively rare, studies have to be large to reach adequate “power”.

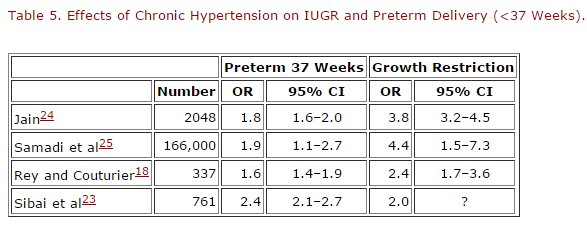
“Treatment for chronic, mild-to-moderate hypertension and pregnancy: what does the evidence say?” Rosene-Montella - An excellent source that brings up the issues with research and therefore what we can or should suggest for therapies.  Good quality paper.

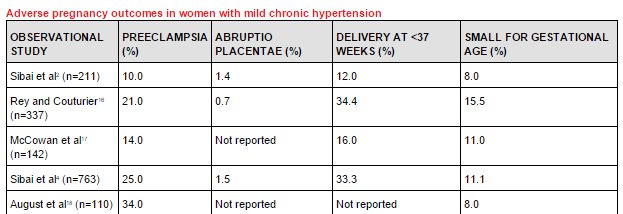
I would add that the inability to get accurate readings and the effects of white coat hypertension make it doubly hard to do good research in this area.  Almost all of the research being done assumes that the common information about hypertension in pregnancy is correct, and almost no one is looking at how nutrition impacts the risks of hypertension in pregnancy (those that are are not doing so holistically, but with a focus on micro and macronutrients rather than a more complex and well rounded perspective).

1. What are the risks of chronic hypertension in pregnancy?

These women have an increased risk of superimposed preeclampsia, placental abruption, and poor perinatal outcome but there is a wide range as far as how large of an increased risk researchers think there is.  Most of the increased risk is from superimposed preeclampsia, so for women who don’t develop that, the statistics are not that shocking (in my opinion).  The risk of superimposed preeclampsia is tricky since it is diagnosed in many different ways, some stricter than others, so it ranges from .47%-52% depending on the source.  I prefer the criteria that includes significant proteinuria, significant symptoms and/or laboratory derangements.  Placental abruption risk is .47%-5% depending on the severity and duration of hypertension - .45-1.9% in mild uncomplicated hypertension and 2.3-5% for severe hypertension with no difference when antihypertensives are used.  These charts are helpful.







“The outcome of uncomplicated essential hypertension, otherwise called low risk chronic hypertension, in pregnancy with adequate prenatal care should probably not be any different than the outcome of any other pregnancy.” Sibai - “Essential Hypertension During Pregnancy”

I would love if there were a chart that included the rates of these complications for women without hypertension at all to compare.  The overall risk numbers on the second graph do help, but don’t include abruption or preeclampsia.  The general population rate (so not excluding hypertensives) is .0065% and preeclampsia is 3-6%.

“Maternal Complications in women who have chronic hypertension” states that chronic hypertensive women have an increased risk of preeclampsia (OR 3.8), gestational diabetes (OR 1.8), and placental abruption (OR 2.3).  “Maternal Complications in women who chronic hypertension” - large study of 3300 women.  Want to find the full text though.

Chapter 5, Sibai

<http://www.aafp.org/afp/1998/1015/p1384.html> - Basic, reliable, used for stats

<http://www.cdc.gov/reproductivehealth/maternalinfanthealth/pretermbirth.htm> - reliable, used for stats

<http://aje.oxfordjournals.org/content/153/4/332.full> - a very large sample size and straightforward study

“Essential Hypertension During Pregnancy”, Chapter 44 Sibai - Similar in information to his other information, with some unique features.  I would again like more in text citations though.

ACOG Practice Bulletin No 29 - Studies differ slightly but perinatal mortality risk is cited as 28/1000 (including superimposed pre-eclampsia), 5/1000 (not including superimposed preeclampsia), 45/1000 (not sure what is included or not) versus 12/1000 in the general population.  Chronic hypertension “doubled the risk for placental abruption and tripled the risk for perinatal mortality”.

In a study of women with severe hypertension after 20 weeks of pregnancy 72% had preexisting chronic hypertension (so this is sort of a misleading article I think, since it is mixing chronic and gestational hypertensions), 78% developed preeclampsia, 71.4% had cesareans, 8.4% had placental abruptions, 8.4% developed HELLP (the same 8.4% from the abruptions perhaps??), 3.9% had acute renal insufficiency, 1.3% pulmonary edema.  THERE WERE NO MATERNAL DEATHS, DIC, OR ECLAMPSIA.  “Pregnancy and Severe Chronic Hypertension: Maternal Outcome” Vigil-De Gracia - 154 women in the sample, retrospective study over 5 years

In another study of women with chronic hypertension, the women who had not expanded their blood volume were more likely to deliver growth restricted babies, or have their babies die, regardless of the severity of their hypertension.  Chronically hypertensive women had lower blood volumes as a group, and had smaller babies.  The lack of expansion was demonstrated by creatinine clearance levels at nonpregnant levels for moms who had SGA babies, and levels FAR below prepregnant levels in moms whose babies died.  Creatinine clearance levels may help identify which women with chronic hypertension will have a poor fetal outcome.   “Expansion of intravascular volume and fetal outcome in patients with chronic hypertension…” - Small study of 20 women with chronic hypertension.

This study looking at placental abruption in chronic hypertensive is really interesting.  Rates were 15.6 and 5.8 per 1000 pregnancies for chronic hypertensives and normotensives respectively.  PIH and fetal growth restriction modified the risk for chronic hypertensives.  The HIGHEST risk was found in women with chronic hypertension, superimposed PIH and large for gestational age babies.  Strangely, chronic hypertension with a large for gestational age baby (but not PIH) is not associated with placental abruption.  So for chronic hypertensives, the bigger the baby, the better prognosis unless they have superimposed PIH.  “Chronic hypertension and risk of placental abruption….” Ananth - Retrospective study from 1995-2002

Women with mild chronic hypertension (diastolic between 90-110) have similar outcomes to the general population if they do not develop superimposed preeclampsia.  5.3% of these women (without preelampsia) had small for gestational age babies, and 1 of the 190 babies died.  A separate subgroup of 21 women who developed superimposed preeclampsia had 32% growth restricted babies, and 5 of the 21 babies died.   “Pregnancy Outcome in 211 Patients with Mild Chronic Hypertension” Sibai - Abstract for a study of 211 women. Reputable source

The rates of stillbirth for women with chronic hypertension vary so wildly.  In the best source I found, though, chronic hypertension had a stillbirth rate of 1.7 per 1000 births, while their classic “low-risk” category (white, 25-29, normal weight, multip, no chronic hypertension, no preexisting diabetes), had a rate of .8 per 1000.  “Prepregnancy risk factors for antepartum stillbirth in the United States” - Reddy, Very large retrospective study. 155,629 singleton pregnancies.

Low serum albumin levels are associated with increased perinatal mortality in hypertensive pregnancies.  “Hypertension in pregnancy: maternal and fetal outcomes according to laboratory and clinical features” Brown - Does not distinguish between types of hypertension.  Large study with 1183 women in Sydney hospital.

1. How does chronic hypertension change the risk of placental abruption?

On the general population rates - “Placental abruption complicates approximately 1% of all singleton pregnancies, and the incidence is at least doubled in twin gestations. A recent, population-based epidemiologic study in the United States comprising 7,465,858 singleton and 193,266 twin births found that abruption was recorded in 0.59% and 1.22% of singleton and twin births, respectively.[17](http://www.glowm.com/section_view/heading/Placental%20Abruption/item/122#r17) For the incidence of placental abruption, the twin to singleton relative risk was 2.1.”

Table 1. Evidence and strength of association linking major risk factors with placental abruption based on published studies

|  |  |  |
| --- | --- | --- |
|  | **Evidence** | |
| **Risk Factors** | **Strength** | **RR or OR** |
| Maternal age and parity | + | 1.1–3.7 |
| Cigarette smoking | ++ | 1.4–2.5 |
| Cocaine and drug use | +++ | 5.0–10.0 |
| Multiple gestations | ++ | 1.5–3.0 |
| Chronic hypertension | ++ | 1.8–5.1 |
| Mild and severe preeclampsia | ++ | 0.4–4.5 |
| Chronic hypertension + preeclampsia | +++ | 7.8 |
| Premature rupture of membranes | ++ | 1.8–5.1 |
| Oligohydramnios | + | 2.5–10.0 |
| Chorioamnionitis | ++ | 2.0–2.5 |
| Dietary/nutritional deficiency | +/− | 0.9–2.0 |
| Male fetus | +/− | 0.9–1.3 |

RR, relative risk; OR, odds ratio.

\*These estimates are the ranges of RR or OR found in independent studies.

“We recently set out to evaluate whether the increased risk of placental abruption among women with chronic hypertension is modified by PIH and fetal growth restriction.[39](http://www.glowm.com/section_view/heading/Placental%20Abruption/item/122#r39) Compared to normotensive women with appropriately grown babies, the risk for abruption was 2.4-fold higher (95% CI, 2.3, 2.5) among women with chronic hypertension as an isolated condition. However, if women experienced either fetal growth restriction and/or PIH, the risks were substantially higher (RR 3.8; 95% CI, 3.6, 4.1) and (RR 7.7; 95% CI, 6.6, 8.9), respectively.[39](http://www.glowm.com/section_view/heading/Placental%20Abruption/item/122#r39)”

http://www.glowm.com/section\_view/heading/Placental%20Abruption/item/122#14871

**A study on placental abruption showed that chronic hypertensive women had no greater risk of placental abruption** (relative risk of 1.4) but women with superimposed preeclampsia (2RR 2.8) and severe preeclampsia (RR 3.8) were associated with higher rates of abruption.  Smoking combined with hypertension raised the risks of abruption more than expected.  “Influence of hypertensive disorders and cigarette smoking on placental abruption and uterine bleeding during pregnancy” Ananth - very large study, and very interesting results.  Study done in Nova Scotia.

<http://www.sciencedirect.com/science/article/pii/S0002937894703280> -

CONCLUSION: Although hypertensive women experiencing abruptio placentae are more likely to have higher-grade abruptio placentae and lower umbilical cord pH values, the overall perinatal outcome was not significantly different from that of normotensive women experiencing abruptio placentae. (AM J Obstet Gynecol 1994;170:1595-9.)

1. Does anything reduce the risk of uncomplicated chronic hypertension in pregnancy?

It seems that L-arginine supplementation may help prevent some complications (superimposed preeclampsia, neonatal complication) and make it so less antihypertensives are required, but more research is needed.  “L-arginine supplementation in women with chronic hypertension” Isabella Neri - small study

1. What are the risks of gestational hypertension?

It is difficult to get a clear answer to this since there are different severities, and it overlaps in many cases with pre-eclampsia at some point.  Mild gestational hypertension is said to need additional monitoring; severe gestational hypertension has outcomes worse than mild pre-eclampsia.

<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3279097/> - Excellent, trust worthy source, good information, conprehensive.

Mild PIH usually develops after 37 weeks, and in those cases the outcomes are similar to those who have no blood pressure issues.  The risk of developing pre-e is between 15-25%, or 35% if PIH developed before 37 weeks.  The risk of PIH recurrence is about 26%.  - Ribowski “Pregnancy Induced Hypertension”

Gestational hypertension may be PROTECTIVE for twin pregnancies.  http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3986365/

1. What lab work shows signs of active hypertensive disorders or risk of developing them?

Increased pulse pressure before pregnancy may suggest increased risk for complicated hypertension in pregnancy.  “Pulse pressure and arterial compliance…” Sarah Hale - interesting abstract.  Very small study

Women with low circulating vitamin D are more likely to have hypertension in pregnancy. “Vitamin D and hypertension in pregnancy” Ringrose - well done paper

Maternal serum hcg can be used to help diagnose and manage hypertension disorders in pregnancy.  Very high hcg levels are seen in preeclampsia and superimposed preeclampsia.  “Can serum HCG Values be Used in the Differential Diagnosis of Pregnancy Complicated by Hypertension” Ayse Gurbuz - very interesting and well documented study

C-reactive protein levels are raised significantly in hypertensive disorders of pregnancy and can be used as an early marker for diagnosis and intervention. “C-reactive protein levels in women with pregnancy induced hypertension” Ghosh - interesting abstract, small study.

Uric acid elevation is likely a result of reduced glomular filtration and is a phenomenon of pre-e.  An increase in insulin resistance, and a beyond average increase in circulating lipids (triglycerides and fatty acids, LDL) as well as a reduction in HDL cholesterol also are seen in pre-e. Also reduced renin, angiotension II and aldosterone.  Different peptides are also being looked at as possible early markers for pre-eclampsia.  Magon, “Hypertension in Pregnancy”

Serum creatinine above 90 micromol/L Australian Prescriber

About 10% of patients with severe preeclampsia also have HELLP, though not all women with HELLP have hypertension.  Hemoglobin and hematocrit may be elevated due to blood volume constriction, or low due to hemolytic anemia as in HELLP.  Decreased coagulation factors, uric acid above 6mg/dL, creatinine may be normal or may be elevated in severe preeclampsia, alkaline phosphatase may increase 2-3 fold and lactate dehydrogenase may be quite high.  Proteinuria is “the last sign to develop” and is an “indicator of fetal jeopardy”.  “Preeclampsia” Courtney Reynolds

Looking at doppler uteroplacental flow-velocity waveforms in early pregnancy can identify women who are at higher risk of developing preeclampsia. “Doppler ultrasound and aspirin in recognition and prevention of pregnancy-induced hypertension” McParland - interesting small study that showed benefits of aspirin therapy for women at higher risk of developing pregnancy related hypertension.

Serum albumin less than 3g was associated with higher odds of severe proteinuria, and below 2.5g was associated highly with HELLP and perinatal mortality. “Clinical significance of serum albumin level in pregnancy related hypertension” Won Joon Seong.  Interesting abstract, study was on 394 women.

1. What causes pregnancy induced hypertension?

This remains one of the “most significant and intriguing unsolved medical problems complicating pregnancy”. Pre-eclampsia seems to be a combination of maternal, placental and fetal factors.  An interesting perspective is to approach pre-e with an understanding of endocrine and metabolic disorders being the most common risk factors - diabetes, renal disease, chronic hypertension, obesity.  Magon “Hypertension in Pregnancy”.

It has been shown that women carrying female babies are more likely to have PIH and preeclampsia.  The theory is that this is due to changes in the immunological tolerance.  “Impact of fetal sex in pregnancy-induced hypertension…” Shiozaki - abstract; study was large and retrospective

The “disease of theories”.  There are many theories, and possibly different types of preeclampsia that would make it so multiple theories are possibly true.  Theories about what causes preeclampsia include endothelial cell injury, rejection phenomenon, **compromised placental perfusion**, altered vascular reactivity, imbalance between prostacyclin and thromboxane, decreased glomular filtration rate with retention of salt and water, **decreased intravascular volume,** increased central nervous system irritability, dissemintated intravascular coagulation, uterine muscle stretch, **dietary factors**, and genetic factors.  The current most prominent theory is that immune system issues cause poor placentation, resulting in decreased placental perfusion, which stimulates the creation of substances with activate or damage endothelial cells throughout the body.  “Preeclampsia” by Courtney Reynolds

Dr Brewer’s theories about blood volume are the most interesting to me.  This article showed that primiparas with a hemoglobin below 10.5 had a 7% chance of developing hypertension whereas primparas with hgb above 14.5 had a 42% chance of developing hypertension.  There were significant differences especially when looking at the 13-19 week readings.  Brewers theory makes the most sense to me for many cases of preeclampsia, although perhaps there are cases where the above theories are also true.  He believed that preeclampsia was the end result of blood volume constriction which resulted from a poor diet.  Unfortunately, his research has been poo pooed and there isn’t much being done to further it, except some studies I found which were seemingly unrelated but were proving pieces of his argument - studies that show blood volume is indeed restricted in cases of pre-eclampsia, that hemoglobin numbers don’t drop the same way, etc.  “Preeclampsia” by Courtney Reynolds is a great source for the details (which honestly are above me) but she notes that “plasma volume is reduced in patients with preeclampsia”, though she seems to think it is a symptom rather than a cause.

Another study showed that women who developed hypertension in pregnancy started out expanding their blood volume, but it was followed by significant contraction in the third trimester, typically before the blood pressure went up (but not always).  The same went for women with chronic hypertension - blood pressure was inversely related to plasma volume and to fetal growth (which relies on plasma volume).  “Plasma Volume Contraction: A Significant Factor in Both Pregnancy Associated Hypertension and Chronic Hypertension in Pregnancy” Eileen Gallery - rather small study but very convincing results.

Metabolic Toxemia of Late Pregnancy by Dr Tom Brewer - Excellent source.  Old and cites much of his own work and case studies but gives an extremely important alternative perspective.  I wish there was something updated but there isn’t yet.

In “Not All Infants…” they pose the hypothesis that there must then be at least two types of preeclampsia, since the majority of babies born to preeclamptic mothers are NOT suffering from reduced blood flow to the placenta as evidenced by the fact that they are the same weights as babies born to normotensive moms.  One type of preeclampsia perhaps restricts fetal growth and leads to preterm delivery and the other does not.  “Not All Infants Born to Women With Preeclampsia Are Low-Birth Weight; Gestational Age is a Key Factor”.  A similar article “Rethinking IUGR in Preeclampsia: Dependent or Independent of Maternal Hypertension?” elaborate a bit and says normotensive women have their risk of IUGR go up when they get pre-e, but chronically hypertensive women do not have their risk go up when they get superimposed preeclampsia (but had higher risk to begin with).  In other words, they are suggesting that we untangle chronic hypertension, preeclampsia and IUGR and start questioning the relationships that have been taken for granted for a long time now.  It seems that IUGR, like preeclampsia, may have many different etiologies, and it is likely more complex than we have described previously.  IUGR is of course also associated with a low BMI, for example.

Maternal factors that increase the risk of preeclampsia other than chronic hypertnesion are first pregnancy, first pregnancy with a new father, older than 35 or moreso 40, family or personal history of preeclampsia, diabetes, lupus, antiphospholipid antibody syndrome, renal disease, and obesity.  “How should hypertension in pregnant patients be managed?” Hayes - Not sure about the reputability of this source, though the information seems in line with other good sources.  Good overview of management.

1. What are signs and symptoms of pre-eclampsia?

Clinical signs of severe PIH or pre-eclampsia may include generalized edema, rapid weight gain, blurred vision or diminished vision, severe headaches, epigastric pain, oliguria, nausea with or without vomiting, hyperactive reflexes, chest pain or tightness, shortness of breath. Ribowski

1. How can we be reasonably certain that someone with hypertension does not have pre eclampsia?

Lab work should include a 24 hour urine test, and over .3g of protein is considered proteinuria.  Spot protein samples with 2+ or greater is highly suggestive of proteinuria too.  ACOG also recommends assessing for end organ damage to assess for severe pre-e - high or low hematocrit, low platelets, elevated liver enzymes, elevated urea nitrogen and creatinine, and an increase from first trimester serum uric acid or elevated serum uric acid levels.

Holistic Midwifery Volume 1 - Anne Frye says the first thing to check is the woman’s diet, to see if it is a sufficient quantity and variety of food.

It may not be possible to distinguish between worsening chronic hypertension and superimposed severe pre-e late in pregnancy (with no history in particular) and may not be possible at all if she has renal disease.  Oliguria and hemoconcentration are indicative of preeclampsia.  Serum creatinine levels may be elevated.  ACOG Practice Bulletin No 29

1. Can we reduce someone’s risk of developing pre- eclampsia?

Baby aspirin daily until 37 weeks and 1.5 g/day calcium may be beneficial. Australian Prescriber

Looking at doppler uteroplacental flow-velocity waveforms in early pregnancy can identify women who are at higher risk of developing preeclampsia.  In one study of such women, 25% developed gestational hypertension, 19% developed proteinuric hypertension and 17% developed hypertension before 37 weeks.  The risk was lowered with aspirin use to 13%, 2% and 0% respectively. “Doppler ultrasound and aspirin in recognition and prevention of pregnancy-induced hypertension”

Aspirin appears to reduce the risk of preeclampsia, particularly for women at higher risk of developing it.  One case of preeclampsia is prevented for every 114 women treated with antiplatelet agents.  It also appears to reduce the risk of preterm birth before 37 weeks.  “High risk” in this context meant having chronic hypertension, or normotension but with previous severe preeclampsia, diabetes, kidney disease or autoimmune disease.  They recommend that women at high risk for preeclampsia and women with more than one moderate risk factor (first pregnancy, over 40, last pregnancy more than 10 years ago, BMI 35 or more at first visit, family history of pre-e, multiple pregnancy)  for preeclampsia take 75mg aspirin from 12 weeks until birth.  “Reducing the Risk of Hypertensive Disorders in Pregnancy” NICE Clinical Guidelines - Extremely good, well researched and cited source.  Probably the best that exists as far as these recommendations go.

There is no evidence that magnesium, folic acid, vitamin C or E, fish oil, garlic, low sat diet, maintaining a “healthy” BMI, or exercise reduce the risk of preeclampsia. “Reducing the Risk of Hypertensive Disorders in Pregnancy” NICE Clinical Guidelines

Rest may have some benefit (study looked at 4 hours of daily left side rest). Lycopene should be researched more, as one study found a large reduction in risk of developing pre-e when taking it.  Calcium supplementation has shown varying results and should have more research done as well (unlikely to HARM, so may be a good choice for those at high risk, especially if they don’t get much dietary calcium).  “Reducing the Risk of Hypertensive Disorders in Pregnancy” NICE Clinical Guidelines

Contrary to the NICE guidelines - Magnesium supplementation may improve lipid status and decrease the risk of developing preeclampsia.  “Study of Serum Lipid Profile and Magnesium in Normal Pregnancy and in Pre-Eclampsia: A Case Control Study” - very well documented study and report.

Consumption of large amounts of cod liver oil or other sources of marine fatty acids in early pregnancy may increase the risk of developing hypertensive disorders in pregnancy. This is especially interesting in relation to the use of aspirin to prevent pre-e since both act as blood thinners.  “Relationship between high consumption of marine fatty acids in early pregnancy and hypertensive disorders in pregnancy” Olafsdottir - 488 women in this study in Iceland.  Interesting.

One study found that supplementing with MORE salt (NaCl) helped slightly reduce the risk of preeclampsia, and there is also a case study showing that salt supplementation helped reduce blood pressure in a pregnant patient.  They used 20g a day of supplemental salt in the original study.  They hypothesize that adding salt helped due to a lack of aldosterone in some pregnant women, which leads to blood volume depletion.  So more salt is a good idea if a woman fits this characterization - a blood pressure that doesn’t fall in the first half of pregnancy, and low tetrahydroaldosterone excretion in the urine (if tested).  “Blood Pressure reduction in pregnancy by sodium chloride” Stefan Farese - Very interesting case study that also references older studies on salt in pregnancy.  Well documented, detailed reporting.

Sabine Kuse has worked with over 20,000 women with high risk pregnancies, and has seen that additional salt is helpful for many women showing signs of preeclampsia, and has never had a negative effect.  Dr. Margaret Robinson also showed the important of salt in her studies in 1950’s - low salt diets led to higher rates of preeclampsia, placental abruption and perinatal death.  These studies were recreated but using rats instead of humans, and the results again showed that low salt diets profoundly affect pregnancy, leading to low birth weight and organ damage.  “Salt is vitally important for a healthy pregnancy” Brewer Site - really great source with references to many interesting studies and physiology information

It seems obvious, but physical activity, especially during pregnancy but also before (and best combined), is protective and lowers the risk of developing hypertensive disorders in pregnancy.  Some women get nervous about being active if their blood pressure is already high or getting higher, but staying active is important.  “Physical Activity and Hypertensive Complications During Pregnancy” Chantel Martin - Large study of over 3000 women in North Carolina.  Showed clear benefit.

1. What are the risks for someone that has preeclampsia?

Infants born at term to women with preeclampsia are similar in weight to those born to mothers without preeclampsia, but preterm babies born to preeclamptic mothers are significantly lower in weight than preterm babies born to non-preeclamptic mothers.  In this particular study, 61% of preeclamptic women gave birth at term, so most of these babies are a very normal birth weight.  This leads to the author’s hypothesis that there must then be at least two types of preeclampsia, since clearly the majority of babies born to preeclamptic mothers are NOT suffered from reduced blood flow to the placenta.  One type of preeclampsia perhaps restricts fetal growth and leads to preterm delivery and the other does not.  “Not All Infants Born to Women With Preeclampsia Are Low-Birth Weight; Gestational Age is a Key Factor” - Very interesting.  Looks at one study.  Not a lot of information about the quality of the study.

1. What are the risks for someone who has both chronic hypertension and preeclampsia?

Interestingly, these women seem to do better in some respects than women who have preeclampsia without ever having had chronic hypertension.  An electron-optical study on placentas showed that chronic hypertensives had similar changes to their placentas as preeclamptic women, but that women with superimposed preeclampsia had less signs of ischemia than women who were normotensive before developing preeclampsia; the point being that the placenta is able to adapt, and having hypertension prior to preeclampsia gives the placenta some amount of protection if the woman does develop preeclampsia as well. “An ultrastructural and ultrahistochemical study of the human placenta in maternal essential hypertension” CJP Jones - An older abstract, nto sure how large the study was.  Interesting findings and I would like a more up to date version.

1. How does chronic hypertension relate to preeclampsia?

Pre-eclampsia is thought to be a disease of endothelial dysfunction, so perhaps pre-existing hypertension damages the endothelial health, making pre-eclampsia more likely as regulatory control of blood pressure is already diminished.  “Treatment for chronic, mild-to-moderate hypertension and pregnancy: what does the evidence say?” Rosene-Montella

Not all women with preeclampsia or even eclampsia have high blood pressure.   **40% of women with eclampsia have seizures at normal blood pressure and without proteinuria!!!!!!** Cipolla “Seizures in women with preeclampsia”

1. What is the rate of eclampsia for those who have pre-eclampsia, and what are the risks of full blown eclampsia?

Nearly 1/50 women with eclampsia die, as well as about 1/14 of their babies (much higher in developing countries). Eclampsia occurs in 1 in 2000-3000 pregnancies in the US.  “Women who develop eclampsia exhibit a wide spectrum of signs and symptoms ranging from severe hypertension and proteinuria to mild or absent hypertension with no proteinuria.”  In a small study, only 13% of eclamptic women could have been considered to have severe preeclampsia prior to seizure.  Another study showed only 20% of patients with eclampsia had a diastolic above 120.  In other words, eclampsia is not always a progression from severe preeclampsia, so it is interesting that that is the focus of management still as people grasp for straws trying to deal with this phenomenon.   **40% of women with eclampsia have seizures at normal blood pressure and without proteinuria!!!!!!**Placental abruption happens after 20-50% of prolonged seizures.  Seizures cause trauma to the mother’s brain, hemorrhage, hypoxia, and cause fetal heart rate changes due to hypoxia as well.

Cipolla “Seizures in women with preeclampsia”

“Uncomplicated eclampsia causes no permanent neurologic deficit”.  Also interesting to note that one set of researchers found that 29% of eclamptic patients DID NOT HAVE PROTEINURIA!  Fascinating.  - “Preeclampsia” Courtney Reynolds - very interesting and thorough source about the theories of what causes preeclampsia.  Brings up controversies and alternative perspectives somewhat.

1. What is the proposed physiology of why hypertensions make pregnancy higher risk?

1. What is the protocol for monitoring and managing chronic hypertension in pregnancy?

There is no consensus.  Most issues are from superimposed preeclampsia and IUGR, so ruling that out by monitoring fetal growth via ultrasound and pre-e workups is suggested. “There is no conclusive data to address either the benefits or the harms of various monitoring strategies for pregnant women with chronic hypertension”  ACOG recommends baseline ultrasound at 18-20 weeks, repeat at 28-30 weeks, and monthly after to monitor fetal growth.  IF growth is restricted, then they recommend frequent NSTs and BPPs (otherwise, not indicated).

Uncomplicated, mild chronic hypertension usually can be delivered at term, vaginally with good maternal and neonatal outcomes.  Induction is indicated perhaps if the woman has a prior adverse outcome.  There are no RCTs that look at the timing of delivery for women with chronic hypertension or superimposed preeclampsia.  Superimposed, severe preeclampsia should be considered for induction after 28 weeks, even earlier for HELLP, after 37 weeks for mild superimposed preeclampsia.    ACOG Practice Bulletin No 29

<http://www.sciencedirect.com/science/article/pii/S0020729214002938>  - Inducing is not beneficial in mild chronic hypertension- “There were no differences in superimposed pre-eclampsia (SPE), severe hypertension, preterm delivery, placental abruption, oligohydramnios, intrauterine growth restriction, or perinatal mortality between the groups. Group B had higher gestational age at delivery (*P* = 0.001) and birth weight (*P* = 0.01), but lower cesarean (OR 3.4; 95% CI, 1.2–10.3;*P* = 0.03) and neonatal care unit admission (OR 5.4; 95% CI, 1.4–21.0; *P* = 0.01) rates. More women with SPE were diagnosed before than after 37 weeks in group B (*P* = 0.01). Overall, patients who developed SPE had more adverse pregnancy outcomes than those who did not.”

For chronic hypertensive, fetal monitoring is recommended to include a ultrasound for fetal growth, amniotic fluid volume and umbilical artery doppler velocimetry between 28-30 weeks, and between 32-34 weeks.  If results are normal, don’t repeat.  Same recommendations if gestational hypertension is discovered prior to 34 weeks, and no fetal monitoring recommended if discovered after 34 weeks unless otherwise indicated.  Their guidelines are extremely medical and don’t appear to have evidence backing most of it up unfortunately.  NICE Guidelines

Monitoring uric acid levels may be of particular benefit for chronically hypertensive women who do NOT have proteinuria since uric acid is a strong predictor for poor outcomes even without proteinuria present.  “Poor pregnancy outcomes linked to increased uric acid” Pittsburgh Medical Center - medium sized study

Albumin/creatinine ratio in a random urine sample may be a reliable alternative or complementary method of monitoring the severity of preeclampsia as well, since 24 hour urine collection is difficult. “Albumin to creatinine ratio in a random urine sample: Correlation with severity of preeclampsia” Fady Moiety - a fairly small sample with a sort of strange design.  Good information but more studies are needed.

1. What is the target blood pressure level in pregnancies that are complicated by hypertension?

Expert opinion is that hypertension shouldn’t be medicated until it is severe since there is little evidence showing benefit from tight control, and there is some evidence showing harm. Most suggest aiming for 140-160/90-100 since lowering more than that can cause fetal growth restriction. Australian Prescriber, “Hypertensive disorders of pregnancy” -another good general resource that outlines the classifications of disorders well.

ACOG Practice Bulletin No 29 - No evidence that medications decrease the incidence of IUGR, superimposed pre-e, placental abruption or perinatal mortality.  May decrease the incidence of severe hypertension.  Also, treatment may INCREASE the risk of SGA babies.

No difference in outcomes when target diastolic is 100 or 85 - only difference was less cases of severe maternal hypertension in the tightly controlled group.  “Less Tight versus More Tight” - <http://www.nejm.org/doi/full/10.1056/NEJMoa1404595> - good abstract detailing a study on different target bps for chronic hypertensives in pregnancy.

Treating with antihypertensives halves the risk of severe hypertension (8-13 women treated to prevent 1 episode of severe hypertension).  Does not change the risks of fetal-neonatal death, preterm birth, or SGA babies.  “Antihypertensive drug therapy for mild-to-moderate hypertension during pregnancy” WHO; really excellent resource, reputable, well cited, large studies.

1. What drugs are used to control hypertension in pregnancy?

Labetalol - beta blocker; Oxprenolol - beta blocker; Nifedpine - calcium channel antagonist; Methyldopa - centrally acting; Hydralazine - vasodilator; Prazosin - alpha blocker. Australian Prescriber.

1. What are the pros and cons of each antihypertensive that is used in pregnancy?

Labetalol - bradycardia, bronchospasm, transient scalp tingling.  Oxprenolol - bradycarida, bronchospasm.  Nifedipine - Severe headache, peripheral edema.  Methyldopa - sedation, light-headedness, dry mouth, nasal congestion, hemolytic anemia, depression.  Hydralazine - flushing, headache, lupus-like symptoms.  Prazosin - postural hypotension.  Australian Prescriber

Beta-blockers were more effective in preventing severe hypertension, otherwise there are no clear differences as far as risk and outcome go from the group that is used in pregnancy.  “Antihypertensive drug therapy for mild-to-moderate hypertension during pregnancy” WHO

1. Are there different recommendations for how to manage labor and birth in someone with hypertension?

No difference in labor management for women with uncomplicated, mild chronic hypertension.  Severe hypertension or secondary hypertension may need medications which need to be titrated/dosed appropriately.  Generally recommended for women with preeclampsia to have blood pressure kept below 160/110.  Little information about using analgesia or anesthesia.  Higher cesarean rate for epidural (no surprise).  ACOG Practice Bulletin No 29

Continuous fetal monitoring has not been shown to improve outcomes other than seizure reduction, even for “high-risk” women, like those with chronic hypertension. For high-risk women, continuous EFM leads to one additional cesarean for every 12 who have continuous EFM.   “How does electronic fetal heart rate monitoring affect labor and delivery outcomes?” - Detailed and concise information.  One of my favorite sources.

Expectant management at least to 41 weeks is best for mild chronic hypertension that doesn’t escalate to superimposed pre-e - “<http://www.sciencedirect.com/science/article/pii/S0020729214002938>  - Inducing is not beneficial in mild chronic hypertension- “There were no differences in superimposed pre-eclampsia (SPE), severe hypertension, preterm delivery, placental abruption, oligohydramnios, intrauterine growth restriction, or perinatal mortality between the groups. Group B had higher gestational age at delivery (*P* = 0.001) and birth weight (*P* = 0.01), but lower cesarean (OR 3.4; 95% CI, 1.2–10.3;*P* = 0.03) and neonatal care unit admission (OR 5.4; 95% CI, 1.4–21.0; *P* = 0.01) rates. More women with SPE were diagnosed before than after 37 weeks in group B (*P* = 0.01). Overall, patients who developed SPE had more adverse pregnancy outcomes than those who did not.””

1. What is the general pattern of blood pressure change in pregnancy?

There is an across the board fall in blood pressure in the first and second trimesters which is generally even greater in chronic hypertensives.  That makes it difficult to diagnose chronic hypertension if it isn't diagnosed prior to the pregnancy.  Chesley and Annitto noted that 39% of chronic hypertensives had a marked decrease in blood pressure in mid pregnancy, with severely hypertensive women being in the normal range.

Sibai, Chapter 5

Hypertensive women have a drop in BP in the first half of pregnancy and may become normotensive during that time.  Then BP rises progressively to pre-pregnancy levels.  “Home Blood Pressure Levels” Evelyne Rey - Interesting study on the difference between home and office readings.  Small but well crafted study.

Normotensive women have their bp drop until 20 weeks, and then rise until the day of delivery, with an average of 8% increase from the middle of pregnancy to the end.  In pregnancies complicated by hypertension, the BP stays the same until 22 weeks and then goes up by 9% systolic and 13% systolic by term.  Systolics were elevated in the first half of pregnancy for women who developed pre-e, whereas those who developed gestational hypertension did not exhibit this.  Women who developed preeclampsia had  their BP rise faster or on a “higher slope’ than those who had gestational hypertension.  All women showed a pulse increase up until the end of the second trimester, and then a slight decrease until term. “Predictable BLood Pressure Variability in Health and Complicated Pregnancies” by Ramon Hermida, et al - Great study that looked at the patterns of bp in different types of pregnancies.  Most useful and from the source description of this.

1. How does labor affect the blood pressure?

1. What happens to blood pressure in the postpartum?

1. What tools do we have to manage blood pressure naturally in pregnancy?

This is not “natural” but giving women with pregnancy induced hypertension a volume expanding infusion leads to a reduction in blood pressure for up to 72 hours.  This points again to Dr Brewer’s approach and theory about blood volume constriction being the cause of preeclampsia rather than a secondary symptom.  The authors of this study think that the blood pressure reduction is related to more than just the volume expansion, and is perhaps related to a fall in prostaglandin levels triggered by the volume expansion.  “Fall in blood pressure in response to volume expansion in pregnancy associated hypertension (pre-eclampsia): Why does it occur?” - interesting but small study

Chinese medicine sees hypertensive disorders as an issue of kidney yin deficiency, and corresponds to the Chinese disease categories slippery fetus and fallen fetus.  This is not my area of specialty at all, but wanted to note that they have treatment protocols and they seem quite promising.  One small study with 16 women with hypertension in pregnancy had 14 of them “cured” with a treatment of a special formula.  “Lei Wei Di Huang and Hypertension in Pregnancy” - very small study but good starting point for further research on this area someday.

Hypnosis and self hypnosis can be useful for reducing blood pressure.

\*“Why hypnosis can reduce high blood pressure and how to do it” - Good basic article.  Not well cited but an excellent resource for clients who may have this going on.

“Hypnosis for high blood pressure” - a script to use for hypnosis, would be great for clients who may have rising bp

There are many herbs which can be useful for managing blood pressure in pregnancy.  Cramp bark, black haw, garlic, hawthorne, reishi.  One daily protocol outlined is 5 ml twice daily of a hawthorne, crampbark or black haw, and passionflower tincture, 1 clove of garlic, 2 ml reishi tincture twice a day, 2 g calcium citrate, 5000 mg magnesium citrate, 1000 mg Vit C, 400 iu Vit E, 30 minutes of walking, daily yoga, biofeedback, meditation, and warm baths 2-3 times a week with epsom salts and lavender essential oil. “botanical treatment of hypertension in pregnancy” - Nice concise source, some studies cited, but not well many as is the case with most herbal sources. It is conservative though and seems in line with other sources I’ve seen.

Susun Weed’s suggestions include avoiding stimulants, drink nettle and red raspberry leaf infusions, exercise, managing emotional stress, maintaining a healthy weight but also not obsessing over weight, biofeedback, garlic (oil capsules are effective for some), parsley, onions, a cucumber a day (or juice), juice of half a lemon + 2 tsp cream of tartar in some water for three days at a time with a rest of two days between, hops tea, passionflower tincture, skullcap tea (1-2 cups a day), hawthorne infusion or tincture (15 drops 2-3 times a day). “Preventing and managing high blood pressure during pregnancy: The Wise Woman Way” Susun Weed - not cited at all, but from one of the big names of herbalism, whom I respect.

Dr Tori Hudsons’s supplement recommendations (NOT SPECIFICALLY FOR PREGNANCY SO SAFETY SHOULD BE CONFIRMED) are calcium, magnesium (400-1200 mg a day), potassium, CoQ10, fish oils, garlic, hawthorne, mistletoe, and Rauwolfia (more on that next). “High blood pressure - lifestyle considerations” Tori Hudson - great resource from a respected naturopath.  Mention of studies but not well cited, but her info is very detailed and she is an expert.

Rauwolfia is a great treatment option for non-pregnant people, and one midwife-herbalist I know has used it successfully for pregnant women as well. Most sources state not to use in pregnancy, but it appears that is founded on a lack of studies rather than studies which has shown problems.  For mild cases they start with 2 drops, 3 times a day, and adjust up until they reach the blood pressure target or they reach the maximum dosage (12 drops 3 times a day according to this source).  I wouldn’t use it as a first line option, but think it is worth considering later in pregnancy if medication is the next option.  Doctor’s have isolated the elements in Rauwolfia to create pharmaceuticals (Reserpine).  “Rauwolfia Root” - Great source on this herb with history, how it works, dosage and side effects.

**HUGS!!!** Frequent hugging with a partner/spouse was associated with lower blood pressure and higher oxytocin levels in one small study.  I think prescribing more hugging for women in healthy relationships is a great idea.  “More frequent partner hugs and higher oxytocin levels are linked to lower blood pressure and heart rate in premenopausal women” Kathleen Light - small study, shows need for more research, but is exciting to see this sort of work being done.

Eating watermelon and taking valerian tincture/capsules is also recommended.   
“Remedies for High Blood Pressure” - source geared towards midwives, not cited, but info is similar to other sources and it is a nice concise source to share.

Essential oils can be used safely to help manage blood pressure, though the extent to which this is possible is debated. At the very least, inhalation is a safe and effective method, and which oil doesn’t seem to matter much, though recommended oils include lavender, ylang ylang, and bergamot. One study on essential hypertensives found once daily inhalation caused significant decreases in blood pressure, pulse, subjective stress, state anxiety, and serum cortisol levels.  “The effects of inhalation method using essential oils on blood pressure…” JH Hwang - Small study, basic abstract.

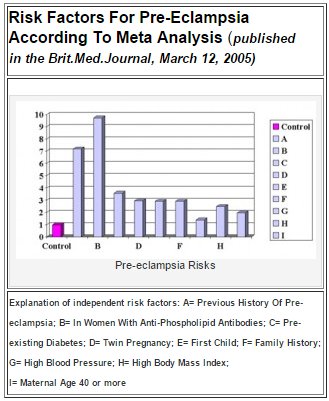
1. Are there techniques for labor that can minimize any blood pressure increases?

1. Are there studies on how nutrition mitigates the potential risks of placental abruption for hypertension in pregnancy?
2. Are there studies that negate the theory that hypertension in pregnancy is higher risk? (Anne Frye mentions one)

1. What is the history of hypertension management in pregnancy?

1. What are some perspectives on the appropriateness of homebirth for women with chronic hypertension?

I found this graph to be useful in conceptualizing the risk of developing pre-e, as related to other risk factors.  Chronic hypertension is G on the graph.  E is first child, and H is high BMI which most midwives have no issue with taking, despite their higher likelihood of developing pre-e.



In another study of women with chronic hypertension, the women who had not expanded their blood volume were more likely to deliver growth restricted babies, or have their babies die, regardless of the severity of their hypertension.  Chronically hypertensive women had lower blood volumes as a group, and had smaller babies.  The lack of expansion was demonstrated by creatinine clearance levels at nonpregnant levels for moms who had SGA babies, and levels FAR below prepregnant levels in moms whose babies died.  Creatinine clearance levels may help identify which women with chronic hypertension will have a poor fetal outcome.   “Expansion of intravascular volume and fetal outcome in patients with chronic hypertension…” - Small study of 20 women with chronic hypertension.

1. What are some perspectives on the appropriateness of homebirth for women with gestational hypertension?

1. How does (or DOES) hypertension effect the placenta?

1. How does maternal hypertension effect a baby's birth weight?

1. How does maternal hypertension affect the baby long term? Does drug use to “control” blood pressure change this?

An increase in blood pressure in the first trimester may be protective, but hypertension the continues or gets worse in pregnancy, or starts at the end of pregnancy may negatively affect the child in the long term with more diagnosis of multiple diseases. “Pregnant mother’s blood pressure may affect future health of children” - interesting journalism piece.  Seems reputable.

1. What are the risks of hypertension OUTSIDE of pregnancy?

It seems from my research that these risks are wildly overstated.  The risks are higher for stroke, heart attack, heart failure, and kidney disease.  But the CDC states that more than half of all adults over the age of 54 have hypertension, so when controlled for age, it seems like the correlation or magnitude of the problem is less clear.  The older people get, the more likely they are to die.  We all have to die from something!!!  So let’s look more at the liklihood.

“The risk of stroke is directly related to the height of the blood pressure. For example, the Framingham indicate that the incidence of stroke in men age 45 to 54 years was 10/100,000 with a systolic blood pressure of 110 to 119 mm Hg and 37/100,000 with a systolic blood pressure of 160 to 169 mm Hg. With respect to diastolic blood pressure, the incidence rose from 17/100,000 with a diastolic blood pressure of 80 to 84 mm Hg to 29/100,000 at a diastolic level of 100 to 104 mm Hg. In women of similar age the difference was even greater, the incidence of stroke being three times greater in the group with the higher diastolic level.” - stroke.ahajournals.  I think this is fascinating.  In the men aged 45-54 years group, the control (systolics 110-119) experienced strokes in 1/10,000 cases, and the matched group with systolics from 160-169 experiences strokes in 3.7/10,000 cases. It was unclear if this mean these were the rates in a year, or what, so here are some more numbers.  The Framingham study site has lots of interesting tables and calculators.  If a woman under the age of 56 has a pretty high untreated systolic number of 156-167, but no other risk factors (smoking, diabetes, prior cardiovascular disease, atrial fibrillation, left ventricular hypertrophy) her risk of having a stroke in the next 10 years is 3%.  If that same woman had a very normal systolic blood pressure of 107-118 her risk would be 1%, and a systolic of 119-155 her risk is slated at 2%.  The risk goes up by age as well, regardless of blood pressure.

<https://www.framinghamheartstudy.org/risk-functions/stroke/stroke.php> - Excellent source, very interesting, well cited, and reputable.

<http://www.cdc.gov/bloodpressure/facts.htm> - information feels basic and oversimplified.

<http://stroke.ahajournals.org/content/5/1/76.long> - very interesting.  one of the original pieces that set the stage for hypertension to be highly managed.  good information that is helpful for getting perspective in an era that now assumes all of this to be true.

Also interesting to note is that cerebral perfusion is maintained by autoregulation, but at too high of pressures, this doesn’t function appropriately.  The interesting part though is that each person upper limit before autoregulation is taken off course is highly individual, which is why some people may have a seizure at 180/120 while others are asymptomatic or may have a slight headache.

* “Preeclampsia” Courteny Reynolds

Usually night time blood pressure drops by about 10% from day time levels, but people who don’t have this drop (non-dippers) may be at increased risk of BP-related complications.  Some evidence has shown that night BP is the best predictor of risk.  “Recommendations for Blood Pressure Measurements in Humans”

1. What is the history of hypertension?

First published BP measurement was in 1733. As a clinical entity it started in 1896 with the creation of the cuff based sphygmomanometer. In 1905 the technique improved with NIkolai Korotkoff describing the sounds heard with the stethoscope when using the sphygomomanometer.  Essential hypertension was introduced as an idea in 1925.  Until the 1970’s, it was generally accepted that hypertension was not something to be treated unless very severe (over 200/100).  Studies in the 1970’s started showing benefit to treatment, and randomized controlled trials were stopped when those given placebos had more problems and it was deemed unethical to withhold the treatment then (reminds me a lot of the Rhogam studies, and a similar time frame).  I wonder if things would be very different if the research didn’t veer this direction and had taken the time to learn more about how treatment did and did not benefit, since now researchers are hesitant or adamantly against withholding treatment to see the effects.

wikipedia “History of Hypertension” - very interesting and relatively well cited source