Postpartum management of the hypertensive disorders of pregnancy: a systematic review and meta-analysis

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OBJECTIVE: To assess the effectiveness and safety of management strategies for postpartum hypertension.

DATA SOURCES: We searched the Cochrane Pregnancy and Childbirth's Trials Register in collaboration with their Information Specialist, on October 20, 2022. As the Pregnancy and Childbirth Review Group closed (2023), we updated our literature search on September 17, 2024 (topped up on September 25, 2025), using a strategy developed with an information specialist from the Royal College of Physicians, United Kingdom.

STUDY ELIGIBILITY CRITERIA: We included randomized controlled trials assessing any intervention (pharmacological, surgical, or models of care) used to reduce maternal blood pressure in participants with postpartum hypertension.

STUDY APPRAISAL AND SYNTHESIS METHODS: Search results were screened independently by 2 authors, with any disagreement resolved by consensus. Data were extracted independently, onto a Cochrane-based bespoke form which included Cochrane's Trustworthiness Screening Tool. Random-effects meta-analysis was performed in RevMan.

RESULTS: Of the 944 studies identified, 40/44 included had informative data. Certainty of evidence was low or very low. There were no safety concerns. In 7 trials (n=1113 participants) of diuretics (primarily furosemide) vs placebo/no therapy, blood pressure control was better with diuretics when administered alongside antihypertensive. In 3 trials (n=96) of antihypertensive vs placebo, data were insufficient to inform effectiveness. In 9 trials (n=865) of antihypertensive (4 types) vs another (3 types) for nonsevere hypertension, additional antihypertensive need was similar in comparisons with either nifedipine or methyldopa, but greater when amlodipine or either enalapril or lisinopril/thiazide were compared with nifedipine. In 8 trials (n=403) of antihypertensive vs another for severe hypertension, blood pressure was lower with diltiazem (vs nifedipine). In 4 trials (n=668) of uterine curettage vs usual care, observed improvements in laboratory parameters were of unclear clinical significance. In 9 trials (n=1263) of models of postnatal care (usually blood pressure self-monitoring/management, N=6) vs usual care, blood pressure was lower 8 months postpartum following blood pressure self-monitoring/management or lifestyle change.

CONCLUSION: While diuretics may aid in blood pressure control, they cannot be recommended as monotherapy. Evidence guiding the optimal choice of antihypertensive agents remains limited. Of greatest relevance to practice is the effectiveness of: enalapril or amlodipine (vs nifedipine) in controlling blood pressure; and blood pressure self-measurement/management or lifestyle change (vs usual care) in preventing longer-term cardiovascular outcomes.

Key words: antihypertensive, diuretic, home blood pressure monitoring, hypertension, postpartum, randomized trial, uterine curettage

Introduction

Hypertension after birth may represent continuation of an antenatal hypertensive

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A.K. is the inventor of HAMPTON for home BP monitoring antenatally. A.E.C. and R.J.M. were involved in SNAP-HT (included in this review). R.J.M. has received grants (NIHR, Stroke Association/British Heart Foundation) to study BP self-monitoring, worked with Omron on BP telemonitoring, and Sensyne developed telemonitoring based on his work in pregnancy; all fees, honoraria, and licensing are held by the University of Oxford. I.D.T., J.N.B., P.v.D., and L.A.M. report no conflict of interest.

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AJOG at a Glance

Why was this study conducted?

Most reviews address only antenatal management of hypertension.

Key findings?

Our findings are based largely on very low certainty evidence. Diuretics cannot be recommended as monotherapy, as coadministered antihypertensives appear responsible for any favorable effects on blood pressure (BP). There is insufficient evidence to recommend one antihypertensive over another, but individual trials of nifedipine vs. amlodipine or angiotensin-converting enzyme inhibitors (with/without thiazide diuretic) suggest nifedipine is more effective. Lifestyle change and BP self-measurement/management appear promising in reducing longer-term outcomes.

What does this add to what is known?

We apply trustworthiness criteria and comprehensively review trials of pharmacological and nonpharmacological interventions to reduce postnatal BP. Research gaps of greatest relevance are the effectiveness of: enalapril or amlodipine (vs. nifedipine) in controlling BP; and BP self-measurement/management or lifestyle change (vs. usual care) in preventing longer-term cardiovascular outcomes.

disorder of pregnancy (HDP) (eg, chronic hypertension), postnatal progression from antenatal chronic or gestational hypertension to preeclampsia, or development of hypertension for the first time. As blood pressure (BP) peaks on days 3 to 6 postpartum, when most women have returned home and are transitioning back to their primary healthcare provider, postnatal hypertension is a significant contributor to maternal morbidity[1] and postnatal hospital readmission.[2]

Given the impact of postpartum hypertension on short- and longer-term health outcomes, it is important to know how to optimize management. Despite increasing recognition of the importance of BP control postpartum,[3] most reviews focus on HDP care antenatally. To date, there is no comprehensive systematic review addressing the full range of postpartum hypertension management strategies. This includes pharmacological interventions beyond antihypertensive medication (eg, loop diuretics to reduce intravascular volume[4]), surgical interventions (eg, uterine curettage to remove all trophoblastic tissue[5]), and models of care (eg, BP self-measurement and titration of antihypertensives, which may lower BP in the short- and longer-term[6,7]). Therefore, this systematic review aimed to assess the effectiveness and safety of interventions for postpartum HDP management, to guide clinical practice, guidelines, and future research.

Methods

Our protocol was prospectively registered, peer-reviewed, and published by the Cochrane Editorial Service.[8]

Information sources and search strategy

We searched the Cochrane Pregnancy and Childbirth's Trials Register in collaboration with their Information Specialist, on October 20, 2022. As the Pregnancy and Childbirth Review Group closed (2023), we updated our literature search on September 17, 2024, with an additional top-up search on September 25, 2025, using a strategy developed with an information specialist from the Royal College of Physicians, UK. Search terms covered "postpartum" AND "hypertension" AND "randomized trial" (as detailed in Methods Appendix).

Eligibility criteria

We included: randomized controlled trials (RCTs), accepting abstracts if authors confirmed analyses were final; participants with an HDP postpartum; any intervention to reduce maternal BP; and comparisons of interventions with placebo, no therapy, or another intervention. Postpartum hypertension was systolic BP ≥140 mmHg or diastolic BP ≥90 mmHg,[3] ideally measured twice, ≥4 hours apart in a clinic/ward setting, from 0 to 6 weeks postpartum. Interventions were antihypertensives or nonpharmacological approaches, which included uterine curettage, models of care, or others to lower BP.

We excluded quasi-randomized controlled trials

Study selection.

We assessed the following outcomes, marked with an asterisk as being of primary interest in our protocol,[8] and with a cross to indicate core outcomes for preeclampsia[9]: BP control*, maternal mortality† and morbidity*†, breastfeeding* (at hospital discharge), postnatal length of hospital stay, postnatal readmission to secondary care, longer-term cardiovascular outcomes, and patient satisfaction*. BP control was: poor BP control* (systolic BP ≥160 mmHg or diastolic BP≥110 mmHg) or unacceptably high BP as defined by authors; need for additional antihypertensive; and BP values* before and following discharge. Maternal mortality† was while pregnant or within 42 days postpartum. Maternal morbidity was: early stroke*†; blood transfusion*†; and safety (maternal adverse events). Longer-term cardiovascular outcomes* included those from 6 weeks postpartum.

Data extraction and analysis.

Search results were screened independently by 2 authors.

Eligible studies were screened using Cochrane's Trustworthiness Screening Tool.[10] Potentially "high risk" studies were classified as "awaiting classification," until authors could provide explanation/reassurance.

Data were extracted using a form based on Cochrane standards, by 2 authors independently, and discrepancies resolved through discussion, involving a third reviewer if necessary. Authors were contacted for clarification, as needed.

Assessment of risk of bias.
Data included the Cochrane risk-of-bias assessment,[11] deemed to be high if at least 1 bias domain was high-risk or ≥3 unclear risk, and low if all domains were low-risk; otherwise, risk-of-bias was unclear.[11]

Outcomes were excluded when attrition was ≥20%, entered into Review Manager,[12] and checked for accuracy.

Data synthesis.
Analyses were by intention-to-treat whenever possible. Outcome denominators were the number randomized, minus participants whose outcomes were missing. For dichotomous data, we calculated summary risk ratios (RRs) with 95% confidence intervals (CIs), and added 95% prediction intervals (PIs) for overall effect post hoc, when events were reported in at least 2 RCTs. For continuous data, we used mean difference (MD). We used random-effects meta-analysis for combining data, anticipating underlying treatment effects may differ between trials, and presented estimates of Tau^2 and I^2. By outcome, we assessed reporting biases using funnel plots if ≥10 studies. We regarded heterogeneity between-studies as substantial with I^2>30% and either Tau^2>0, or P<0.10 in chi-square test for heterogeneity; substantial heterogeneity was investigated using subgroup and sensitivity analyses, including use of risk of bias.

Certainty of evidence was evaluated using grading of recommendations assessment, development and evaluation.[13] We used grading of recommendations assessment, development and evaluation profiler guidance development tool to create Summary of Findings tables.

Subgroup analyses were planned by: antihypertensive type, route of administration, and timing of onset after birth; HDP type; ethnicity; parity; gestational age at birth; and/or BP level at randomization. Subgroup differences were examined by interaction tests, quoting the chi-square statistic and p, and the interaction I^2.

Sensitivity analyses were planned to explore the effect of high risk of bias for allocation concealment and attrition, by excluding these studies. Also, we performed a post hoc network meta-analysis (NMA) for pharmacological interventions and the outcomes of poor BP control and additional antihypertensive, the impact of all interventions vs placebo/no therapy or one intervention versus another; any 2 interventions were compared by combining direct estimates, obtained by pooling data from head-to-head studies that compared those interventions, while indirect estimates were obtained by pooling data from studies through all common comparators. We used a Bayesian random-effects model with a logitlink function, using the getmc package in R statistical software version 4.4.2.

Results

Study selection

Of the 944 citations assessed, we included 44 trials, of which 4 did not report outcomes of interest and were not considered further. For full details, see Supplemental Figure 1 (PRISMA Diagram).

The 40 included trials had a median of 92.5 participants (range 6−480). Further details are discussed by interventions: diuretics (N=7 trials),[4,14−19] antihypertensives for nonsevere (N=12)[20−31] or severe (N=8)[32−39] hypertension, uterine curettage (N=4)[5,40−42], and models of care (N=9).[6,43−50]

Study characteristics

Supplementary tables detail individual trial characteristics, including drug dosage (Supplemental Table 1), and summarize pharmacological interventions (Supplemental Table 2).

Risk of bias of included studies

Risk of bias was high for most trials (33/40, Supplemental Figure 2, a), primarily due to the lack of blinding; ≈20% of trials reported outcomes for which loss to follow-up was >20%, primarily for longer-term outcomes (Supplemental Figure 2, b). Three trials did not report outcomes for postpartum participants separately from antenatal participants but were included for outcomes for which there were no events.[32,34,35] Most outcomes were based on VERY LOW certainty evidence, due to high-risk-of-bias, between-trial heterogeneity, and small sample sizes. Only descriptive analysis of heterogeneity was possible.

Outcomes for which data were available are discussed below; all are detailed in Summary of Findings Supplemental Tables 3−7.

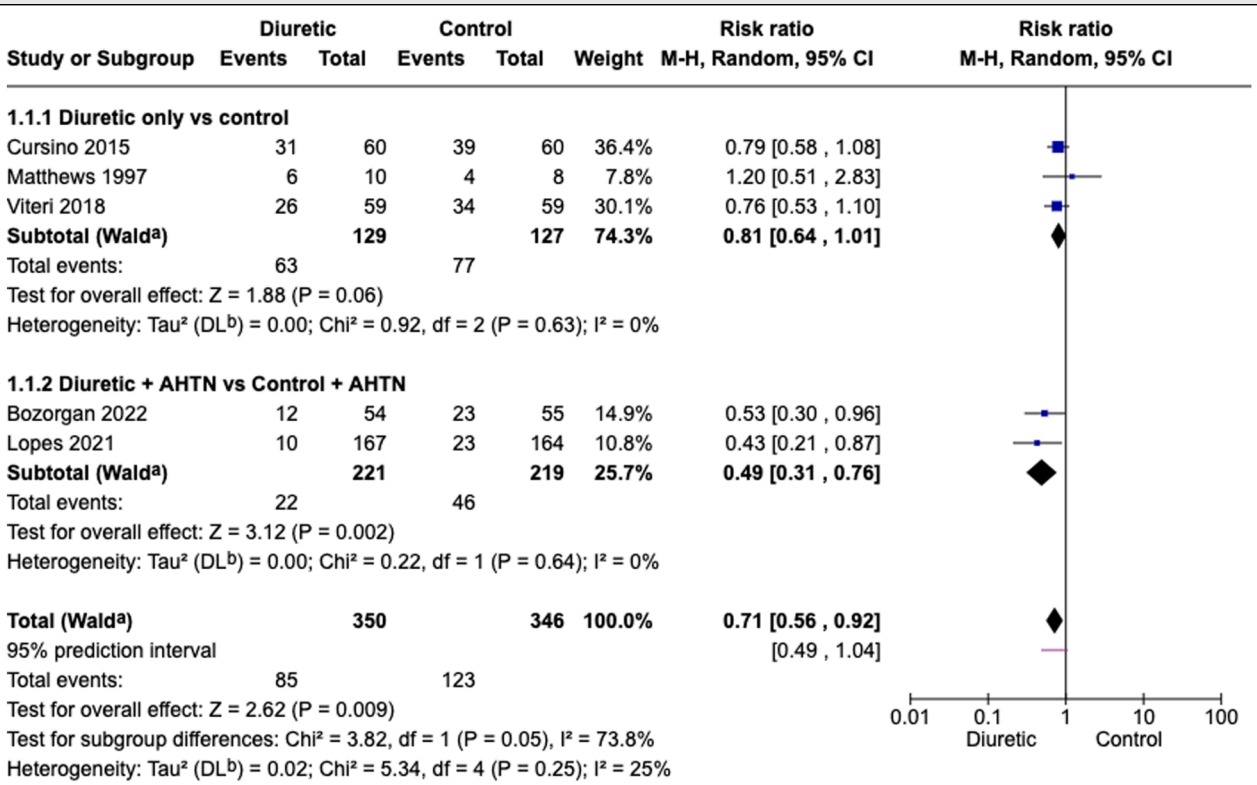
Synthesis of results

Diuretics.
Seven trials (median 118 women) compared diuretics with placebo/no therapy.[4,14−19] Women had preeclampsia[14−18] or any HDP.[4,19] Daily oral furosemide (20 mg[14,16,19] or 40 mg[15,18]) or torsemide (20 mg[17]), with supplemental potassium in 1 trial.[4] Controls received placebo[15,17,18]/no therapy.[4,14,16,19] To both groups, 3 trials administered antihypertensives (nifedipine[14,16] or unspecified preexisting medication[19]).

All outcomes were based on VERY LOW to LOW certainty evidence (Supplemental Table 3).

Poor BP control (BP≥150/100 mmHg by discharge on days 2−3 postpartum,[17] or persistent hypertension by days 5[14], 7−8,[15,19] or 10[18]) was less frequent with diuretic (vs control) (85/350 vs 123/346; RR=0.71, 95% CI [0.56−0.92], 95% PI [0.49−1.04]; I^2=25%; N=5 RCTs),[15,17,18] which appeared due to trials that administered antihypertensive to both groups[14,19] (Figure 1). Need for additional antihypertensive did not differ between groups (133/557 vs 140/556; RR=0.94, 95% CI [0.66−1.33], 95% PI [0.46−1.93]; I^2=61%; N=7); between-trial heterogeneity appeared due to trials that administered antihypertensives to both groups (Figure 2). Of 2 trials that administered nifedipine to both groups; the one that found furosemide reduced additional antihypertensive was of lower quality[14] than the trial that found furosemide increased such need.[19] In leave-one-out analyses, I^2 remained 0 within diuretic vs placebo/no therapy when additional antihypertensive was not administered to both groups, and was substantial (>80%) when additional antihypertensive was administered (Supplemental Table 3). One trial (102 women) reported BP in a format usable for meta-analysis.[14] Systolic BP (but not diastolic BP or mean arterial

FIGURE 1
Poor blood pressure control: diuretic vs placebo/no therapy trials



[a]CI by Wald-type method. [b]Tau[2] by DerSimonian and Laird method.

CI, confidence interval.

pressure [MAP]) on day 5 postpartum was lower with diuretic (vs control, MD −4.00 mmHg [−6.22 to −1.78]). BP information in other trials, reported graphically or descriptively, did "not differ" between groups.

No maternal death,[17] stroke,[17,19] or blood transfusion[17] events were reported. There was no between-group difference in maternal adverse events reported as delayed postnatal complications[4] (6/433 vs 8/433; RR=0.75 [0.27−2.10]; N=4), with no events when defined as maternal morbidity.[16,17,19]

Length of hospital stay ranged from 2 to 7.9 days in each group.[4,14−17,19] Postnatal readmission did not differ between groups (12/251 vs 17/251; RR=0.93 [0.21−4,18], 95% PI [0.10−8.43]; I^2=48%; N=2; subgroup interaction *P*=0.17).

Longer-term cardiovascular outcomes (BP at 6 weeks postpartum) did not differ between groups (3/10 vs 2/8; RR=1.20 [0.26=5.53]; N=1).

Antihypertensive therapy

Vs placebo/no therapy.

Three trials (median 31 women, all with preeclampsia) compared antihypertensive with placebo,[20−22] 2[20,21] regardless of postnatal BP level (L-arginine[21] started before birth or oral nifedipine started immediately after birth[20]). Another trial evaluated intravenous (IV) ketanserin for diastolic BP >95 mmHg.[22]

All outcomes were based on VERY LOW certainty evidence (Supplemental Table 4).

Additional antihypertensive was not needed with nifedipine vs placebo (31 women).[20] MAP at 18-36 hr postpartum was lower (MD −6.30 mmHg [−7.83 to −4.77]) with antihypertensive (vs placebo/no therapy) with nifedipine,[20] but not with L-arginine (MD −1.00 mmHg [−8.01−6.01]).[21]

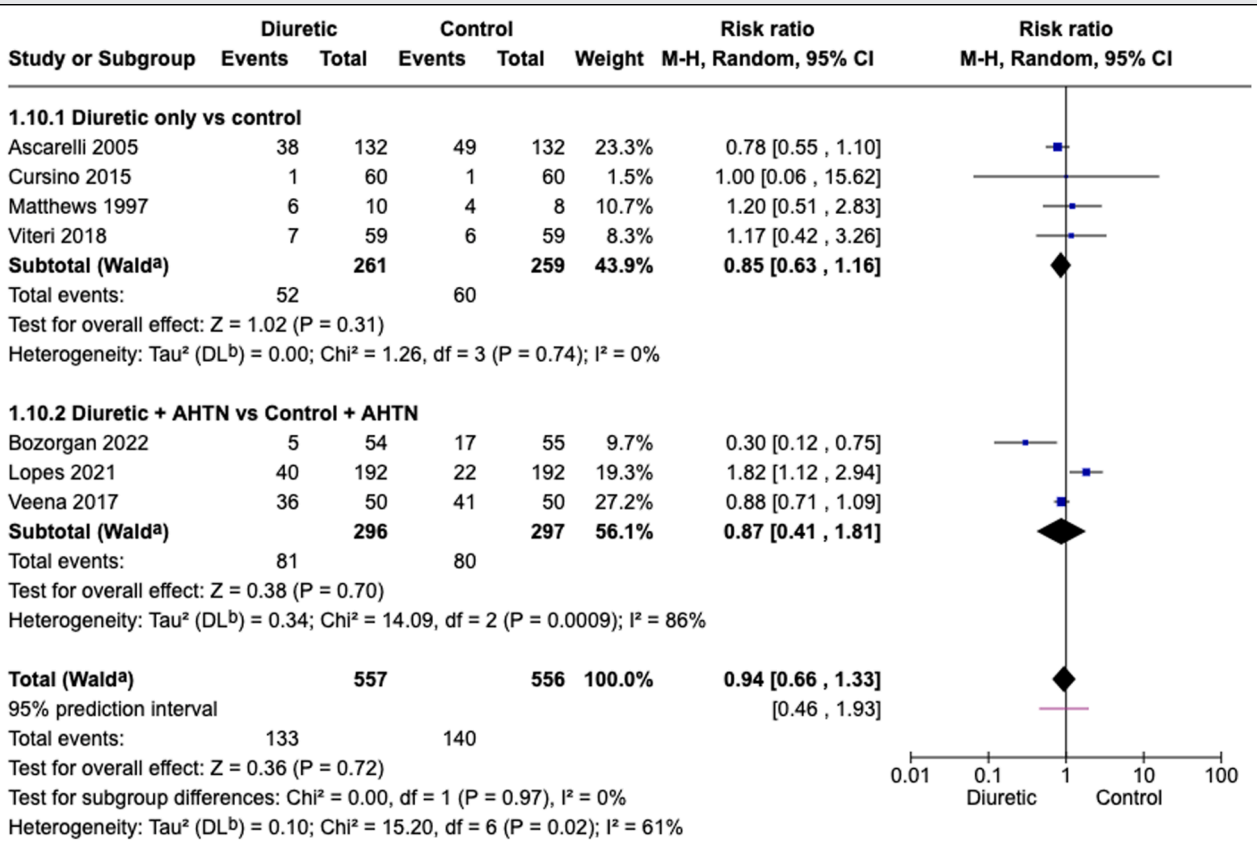
There was no difference in adverse events (4/42 vs 1/43; RR=4.00 [0.49−32.72]; N=2), defined as reversible side effects (eg, headache),[21] or need to stop the drug due to side effects.[22]

One trial reported postnatal discharge at a "similar time."[20]

Vs other antihypertensive (for non-severe hypertension).

Nine trials (median 88 women) compared one antihypertensive with another,[23−31] for any HDP[23−27,31] (including *de novo* postpartum hypertension[24]), gestational hypertension,[28] preeclampsia,[29] or either.[30] Eligible BP was ≥140/90 mmHg,[30] diastolic BP 95-105 mmHg[28] or ≥96 mmHg,[29] BP≥150/100,[24,25,27,30] or not stated (presumed to be 140/90 mmHg).[23,26,31] Trials compared labetalol,[24,25] amlodipine,[26] enalapril,[23] or hydrochlorothiazide+lisinoproil[27] with nifedipine, or labetalol with amlodipine[30] (all oral). Methyldopa, administered orally[28,31] or intramuscular,[29] was compared with oral timolol,[28] IV hydralazine,[29] or oral captopril.[31] The trial

FIGURE 2
Need for additional antihypertensive: diuretic vs placebo/no therapy trials



Leave-out-one analyses, failed to identify the source of between-trial heterogeneity. [a]CI by Wald-type method. [b]Tau[2] by DerSimonian and Laird method.
AHTN, antihypertensive; *CI*, confidence interval.

studying parenteral antihypertensive recruited women with postpartum diastolic BP ≥96 mmHg.[29]

Outcomes were based on primarily VERY LOW certainty evidence (Supplemental Table 5a).

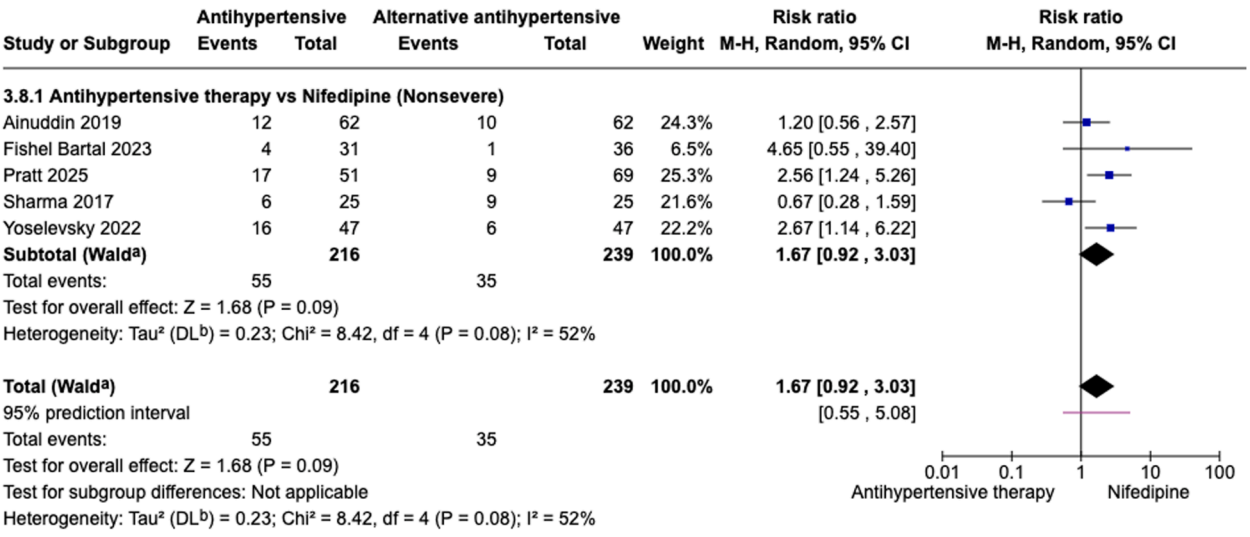
Poor BP control (severe hypertension) did not differ with labetalol vs amlodipine[30] (5/65 vs 2/65; RR=2.50 [0.50−12.42]; n=130 women), hydrochlorothiazide/lisinopril vs nifedipine[27] (6/31 vs 5/36; RR=1.39 [0.47−4.13]; n=67), or captopril vs methyldopa (continued from before birth; 80/84 vs 81/88; RR 1.03 [0.96−1.12]; n=172). There was no difference in need for additional antihypertensive between other antihypertensives (labetalol,[24,25] hydrochlorothiazide/lisinopril,[27] amlodipine,[26] enalapril[23]) vs nifedipine (55/216 vs 35/239; RR=1.67 [0.92−3.03], I^2=52%; N=2) (Figure 3); in leave-one-out trial analyses, between-trial heterogeneity was reduced to 6% following exclusion of Sharma et al[25] (and RR 2.07 [1.32−3.27], 95% PI [1.24−3.46]. Two of the 4 other trials administered angiotensin converting enzyme inhibitors,[23,27] and another evaluated amlodipine[26]; 2[23,26] were significantly less effective than nifedipine in the individual trials. With methyldopa vs other antihypertensive, there was no difference in need for additional antihypertensive (3/142 vs 6/136; RR=0.50 [0.12−1.97]; I^2=0%, N=3), with no events reported in the trial with timolol as the comparator.[28] The latest BP values reported were at day 1 (12 hours),[29] day 6[28], hospital discharge,[30] or following discharge on days 7-10[27,31]; standard deviation values were often unavailable, but mean BP was consistent with no between-group differences.

There were no maternal deaths[27,29,30,51] (317 women), but 1 stroke with labetalol[30] (vs amlodipine, 130 women). Side effects were: "major"[25]; "adverse events"[23,27]; bradycardia, asthma, or hyperglycemia for labetalol, or tachycardia, headache, leg swelling, or orthostatic hypotension for amlodipine[30]; or need to change drugs due to side effects[28,29]; the few side effects were similar between groups (4/222 vs 3/225; RR=1.44 [0.33−6.32], 95% PI [0.33−6.32]; I^2=0%; N=6).

Postnatal length of stay was ≈3 to 4 days and similar between groups (MD 0.05 days [−0.32−0.42; n=455). Most were breastfeeding at discharge on hydrochlorothiazide/lisinopril vs nifedipine[27] (21/31 vs 19/36; RR=1.28 [0.87−1.90]).

Postnatal readmission with amlodipine,[26] enalapril,[23] and

FIGURE 3
Need for additional antihypertensive: antihypertensive vs nifedipine trials



By leave-out-one analysis, heterogeneity was attributable to the Sharma 2017 trial. For details, see Supplemental Table 5, a. [a]CI by Wald-type method. [b]Tau[2] by DerSimonian and Laird method.

CI, confidence interval.

hydrochlorothiazide/lisinopril[27] vs nifedipine (7/129 vs 6/152; RR=1.34 [0.47−-3.87], 95% PI [0.47−3.87]; I^2=0%; N=3).

Vs other antihypertensives (for severe hypertension).

Eight trials (median 42 women) compared one antihypertensive with another.[32−39] Most women had preeclampsia, but 2 trials specified only severe hypertension.[37,38] Three trials enrolled antepartum and postpartum women, without reporting outcomes specifically for postnatal recruits.[32,34,35] Antihypertensives were: IV labetalol vs IV hydralazine[32−34]; IV labetalol,[35] oral diltiazem,[36] oral hydralazine,[37] or oral methyldopa[39] vs oral nifedipine; or oral clonidine vs oral captopril.[38]

All outcomes were based on VERY LOW certainty evidence (Supplemental Table 5, b).

BP control (persistent severe hypertension) did not differ between groups in the IV labetalol vs IV hydralazine trial that reported this outcome[33] (1/40 vs 0/42; RR=3.15 [0.13−75.05]); most women required 1 to 2 doses of antihypertensive, as in the captopril vs clonidine trial.[38] Need for additional antihypertensive was similar with other antihypertensives vs nifedipine (25/70 vs 14/72; RR=2.02 [0.74−5.55], 95% PI [0.49−8.29]; N=3) and clonidine vs captopril (35/43 vs 37/45; RR=0.99 [0.81−1.21]), although event rates were low and high, respectively. The latest BP values were reported on days 2[36] and 4[38] and were lower (by 14 mmHg systolic, 9 mmHg diastolic, or 10 mmHg MAP) with diltiazem vs nifedipine,[36] but similar with hydralazine vs nifedipine,[37] or clonidine vs captopril[38] (Supplemental Table 5, b).

There were no maternal deaths[32,33,39] (171 women). Maternal adverse events were similar between groups (22/123 vs 31/129; RR=0.74 [0.46−1.21], I^2=0%; N=3), defined as any symptoms,[35] need to change drugs due to side effects,[33] or "adverse reactions" (eg, dry cough, rash).[38]

Postpartum length of stay was 4 to 5 days, and similar with clonidine vs captopril[38] (MD 0.70 days [−0.12−1.52]).

Network meta-analysis

The NMA failed to run in R because of disconnected networks, given the small numbers of reporting trials and the list of antihypertensive therapy approaches: placebo/no therapy, furosemide or torsemide, nifedipine, furosemide+nifedipine, antihypertensive not otherwise specified (="placebo+existing antihypertensive'"), furosemide + antihypertensive not otherwise specified, labetalol, amlodipine, hydrochlorothiazide+lisinopril, captopril, methyldopa, timolol, hydralazine, dihydralazine + nifedipine, clonidine. We did not combine nifedipine and amlodipine, given their difference half-lives.

Uterine curettage

Four trials (median 78 women) compared uterine curettage with usual care.[5,40−42] All participants had preeclampsia (defined as proteinuric gestational hypertension) deemed "severe." Curettage was undertaken immediately after delivery of the placenta, following maternal sedation, and under ultrasound guidance for women who had a vaginal birth.

All outcomes were based on VERY LOW certainty evidence (Supplemental Table 6).

The only measure of BP control was MAP at 24 hours postpartum, which was similar between curettage and usual

care[5,40,41] (MD −7.12 mmHg [−15.01−0.78]; 95% CI, PI [−22.71 to +8.47]; I^2=97%; N=3), but substantial heterogeneity was unexplained in leave-one-out analyses (Supplemental Table 6).

No mortality[41] was reported (100 women). Most outcomes were about safety, related to surgical or medical complications (eg, acceleration of maternal preeclampsia). There was no uterine perforation (0/50 [0%−7.1%]).[41] With curettage (vs usual care), preeclampsia-related laboratory values at 24 hr postpartum were similar (ie, platelet count,[5,40,41] serum creatinine,[5,41] lactate dehydrogenase[40−42]) or slightly more favorable (ie, lower uric acid,[5,41] aspartate aminotransferase,[5,40−42] and alanine aminotransferase[5,40−42]), but any differences were very small (Supplemental Table 6).

Length of stay was shorter (MD −5.50 days [−6.58 to −4.42]; N=1[41]).

Models of care

Nine trials (median 120 women) from Europe and North America, compared novel models of care with usual care.[6,43−50] Participants had gestational hypertension or preeclampsia, with 4 trials[44−46,48] also including women with chronic hypertension, and 1 trial not specifying HDP type.[47] Six trials evaluated BP self-monitoring, with[6,43−45,47]/without[46] patient guidance about medication down-titration,[6,43] down or up-titration,[44,45] or titration unspecified[47]; all compared the intervention with usual care, except for the artificial intelligence (AI)−guided BP self-monitoring/management, which compared with BP self-monitoring/management with manual instructions. Other interventions studied were: BP treatment thresholds of 140/90 vs 150/95 mmHg[48], nutrition and exercise (vs usual care),[49] and an intervention aimed at improving neonatal sleep (vs usual care).[50] Diagnostic criteria for hypertension (140/90 mmHg) and preeclampsia (hypertension and new-onset proteinuria from 20 weeks) were similar. Follow-up was for days (eg, 7 days[44,46]), weeks (eg, 2[48] or 6[45]), months (eg, 4[50], 6[6,49], or 9[43]), or was unspecified.[47]

Outcomes were based on primarily VERY LOW certainty evidence (Supplemental Table 7).

Need for additional antihypertensive did not differ between groups in small trials of: differential BP control (44/128 vs 32/128; RR=1.38 [0.94−2.02]; N=1), BP self-monitoring/management (vs usual care; 47/136 vs 45/143; RR=1.05 [0.93−1.19], I^2=0%; N=2), or AI-guided BP self-monitoring/management (vs BP self-monitoring with instructions; 3/59 vs 5/60; RR 0.61 [0.15−2.44]; N=1). BP was similar with BP self-monitoring/management (vs usual care) (Supplemental Table 7), but diastolic and mean arterial BP were lower with improved neonatal sleep (vs usual care; MD −4.00 mmHg [−7.33 to −0.67]).

No maternal mortality or adverse safety events were reported[45] (119 women).

Postnatal readmission did not differ between groups, according to differential BP control, BP self-monitoring/management (vs usual care), or AI-based BP self-monitoring/management (vs manual instructions) (52/580 vs 58/588; RR=1.12 [0.53−2.35], I^2=71%; N=7). Between-trial heterogeneity was attributed to the BP self-monitoring/management (vs usual care) comparison, and in leave-one-out analyses, 1 trial[43] that was similar methodologically to another trial from which it differed in effect[6] (Figure 4). However, BP (systolic, diastolic, MAP, and/or 24-hour ambulatory BP monitoring) was lower at: 6 weeks after birth following BP self-monitoring/management (vs usual care); and 6 to 9 months postpartum following BP self-monitoring/management or a lifestyle intervention (vs usual care), but not with improved neonatal sleep (Supplemental Table 7).

No trial found differences in satisfaction with care, but different measures precluded meta-analysis: health-related quality of life (but with>20% loss to follow-up),[43] median Likert scale scores for overall experience and decisional regret,[45] and satisfaction as a theme in qualitative analyses of interviews with half of participants.[46]

Comment

Principal findings

This systematic review identified 40 RCTs of interventions to lower BP postnatally in women with HDPs, providing primarily VERY LOW quality evidence, with single trials informing most outcomes, usually BP control or safety.

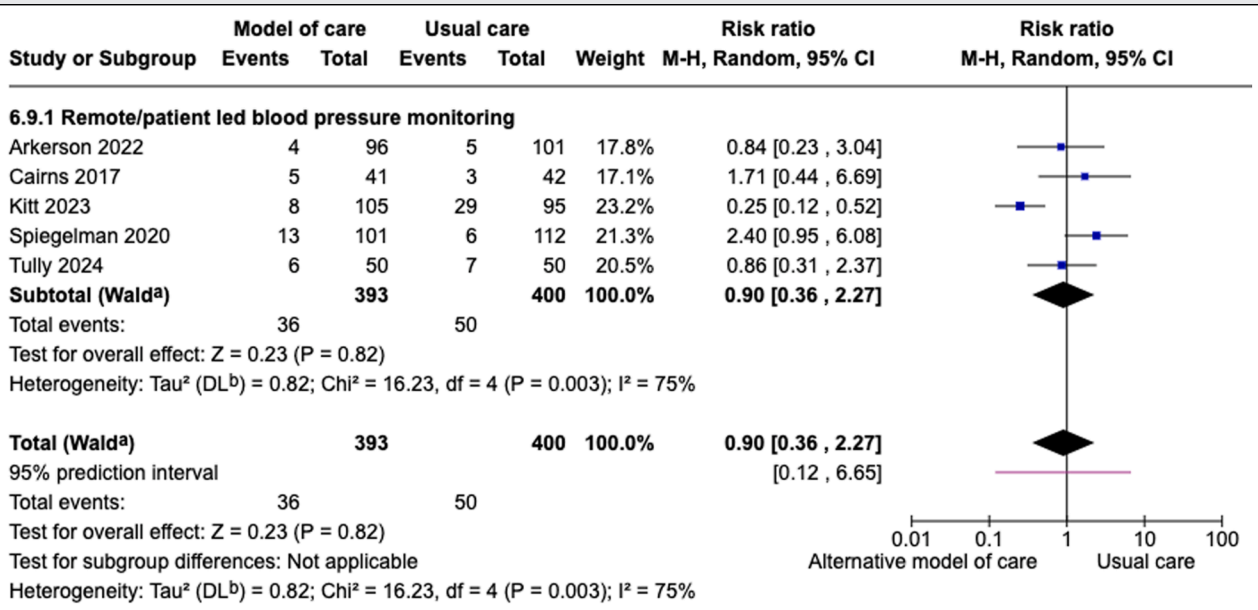
Women with HDPs who received loop diuretics, primarily furosemide (vs placebo/no therapy) postnatally, had better BP control; however, the effect appeared attributable to trials that administered additional antihypertensive medications to diuretic and control groups and the 95% PI was not significant. No safety concerns were raised, although data were limited on breastfeeding.

For antihypertensive (vs placebo/no) therapy, there was no reliable evidence to guide postpartum management. Trials enrolled women with preeclampsia before birth, to either prevent or treat postpartum hypertension. BP was lower with IV ketanserin, a drug not widely used in maternity. There were no safety concerns.

There was little to guide choice of one antihypertensive over another. Given multiple comparisons made, no reliable evidence was available for treatment of nonsevere hypertension, and NMA was not possible because of disconnected networks. Of note, in single trials vs nifedipine for nonsevere hypertension, amlodipine and enalapril or lisinoproil/thiazide were associated with more additional antihypertensive use. There were no safety concerns. For treatment of severe hypertension, there was no clear impact of one antihypertensive vs another.

There was scant evidence available to evaluate the risks and benefits of uterine curettage. No BP outcomes were reported. Most outcomes were about safety, showing only slight improvement in laboratory results, reflective of preeclampsia resolution, and of uncertain clinical significance. While no

FIGURE 4
Postnatal readmission: remote/patient-led blood pressure monitoring



Leave-out-one analyses identified Kitt 2023 as the source of between-trial heterogeneity (RR, 1.39; 95% CI, 0.79–2.40; I^2=0%). [a]CI by Wald-type method. [b]Tau^2 by DerSimonian and Laird method.

CI, confidence interval; *RR*, risk ratio.

uterine perforation was reported, data were consistent with rates up to 7%.

Models of postnatal care focused on BP self-monitoring and antihypertensive titration, which was associated with numerically lower BP at 6 weeks to 8 months after birth (after antihypertensive therapy had stopped); there was no evidence of harm. Improved neonatal sleep and lifestyle change were also associated with lower BP.

Comparison with existing literature and interpretation

This review updates previous systematic reviews of RCTs,[18,52–54] or randomized and observational literature.[55–57] To our knowledge, ours is the only review to apply trustworthiness criteria, and comprehensively review RCTs of pharmacological and nonpharmacological interventions to reduce postnatal BP, covered variably by previous reviews of: loop diuretics,[18,52–57] antihypertensives,[52–55,57] uterine curettage,[55] and models of care.[55,57] Of note, some previous reviews evaluated magnesium sulfate which is not administered to lower BP.[52,57] To the collective literature from these reviews, we found 2 additional trials of antihypertensives[26,30] or evaluating models of care,[46–49] and excluded 4 trials not meeting trustworthiness criteria and one that was withdrawn (Supplemental Figure 1).

For diuretics, our synthesis aligns with prior results, in finding no significant improvement in postpartum BP control, based on weak evidence. Although not eligible for this review, a furosemide vs placebo trial for prevention of postnatal hypertension found no impact on BP before hospital discharge or initiation of antihypertensive.[58]

Here as in previous reviews, there remains sparse information about restarting women's antenatal antihypertensive after birth. Also, evidence remains inconclusive to recommend one antihypertensive over another, regardless of hypertension severity. The quantity of information remains small in contrast to antepartum use of antihypertensives. For nonsevere hypertension, NMA (61 trials, 6923 women) demonstrated all commonly used antihypertensives are more effective than placebo for BP control.[59] For severe hypertension, there have been two NMAs. A 2018 NMA (32 trials, 3236 women) found no difference in effectiveness between IV labetalol and either oral nifedipine or IV hydralazine, but more data were needed for oral nifedipine and IV hydralazine.[60] A 2019 NMA restricted to first-line agents (17 trials, 1591 women), found oral nifedipine lowered BP more successfully than IV hydralazine.[61] As there is little reason to believe that agents effective antepartum would not lower BP postpartum, future work may best focus on antihypertensives that cannot (ie, renin-angiotensin-aldosterone system [RAAS] inhibitors) or are not often (eg, amlodipine) administered antepartum. There is a pressing need to study RAAS inhibitors, as enalapril is now first-line therapy for postpartum hypertension in the UK,[62] and amlodipine is an increasingly popular antihypertensive that women have often taken before pregnancy. Each agent has a half-life (ie, ≈11 hours for

enalaprilat, the active metabolite of enalapril, and ≈30−50 hours for amlodipine) longer than for nifedipine (≈6−11 hours for extended-release), and each has been associated with more use of additional antihypertensive therapy (vs oral nifedipine).

For uterine curettage, our conclusion is consistent with Cairns *et al*,[41] although the full MSc dissertation was obtained for 1 abstract they included, and another trial they included has now been retracted.[63] The risk of uterine perforation may be higher than associated with dilatation and curettage for postpartum hemorrhage (5%),[64] and cannot be recommended to accelerate postpartum recovery from preeclampsia. We were unable to identify relevant active research.

To our knowledge, ours is the first comprehensive review of RCTs to include literature on models of postnatal care. Whilst BP self-monitoring/management may increase BP recording and its frequency, particularly among those suffering from inequalities,[57] findings from the handful of small trials should be considered provisional, as should the impact of postnatal lifestyle change on BP. Larger trials are underway, undertaking longer follow-up and detailed assessment of cardiac structure and function.[65]

Strengths and limitations

Strengths of our review include the comprehensive nature of management strategies included, and peer-review of the protocol and its publication by the Cochrane Pregnancy and Childbirth Group. We minimized potential biases by having clearly defined inclusion criteria and prespecified outcomes, and excluded members of the review team from decisions about studies in which they were involved.

Limitations relate to the published literature included. Few studies mentioned breastfeeding or reported outcomes beyond the first postpartum week, and one reported satisfaction. Certainty of evidence was primarily VERY LOW. Data quantity was compromised by 3 trials that enrolled antepartum and postpartum women, but did not report their outcomes separately.

Conclusions and implications

Care of women with postpartum hypertension is guided by largely VERY LOW certainty evidence and remains incomplete. Research gaps of greatest relevance to current practice are whether RAAS inhibitors or amlodipine are as effective for postnatal BP control as nifedipine, and the role of BP self-measurement and treatment on the incidence of chronic hypertension and cardiac remodeling. ■

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