

Association Between Hypertensive Disorders of Pregnancy and Postpartum Cardiovascular Risk: Nursing Interventions

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Abstract

Hypertensive disorders of pregnancy (HDP)—encompassing chronic hypertension, gestational hypertension, preeclampsia, and eclampsia—complicate roughly 8% to 10% of pregnancies globally and represent leading contributors to acute maternal morbidity and mortality. Beyond peripartum dangers, epidemiologic and translational data show that HDP reflects early microvascular, endothelial, and metabolic dysfunction that unmasks or accelerates lifetime cardiovascular disease (CVD). Women with a history of preeclampsia experience a two- to four-fold increased relative risk of future chronic hypertension, ischemic heart disease, heart failure, and cerebrovascular accidents within five to fifteen years following delivery.

Despite official recognition by the American Heart Association (AHA) and the American College of Obstetricians and Gynecologists (ACOG) identifying HDP as independent sex-specific cardiovascular risk-enhancing factors, a critical gap persists across the "fourth trimester" and longitudinal primary care transitions. Nursing professionals—including inpatient obstetric nurses, community and public health home-visiting nurses, and advanced practice registered nurses (APRNs)—occupy an essential vantage point across the healthcare continuum.

This paper examines the pathophysiologic links bridging HDP with remote cardiovascular degeneration, synthesizes clinical evidence on long-term risk trajectories, and presents an evidence-based clinical nursing framework. Emphasizing early inpatient hemodynamic stabilization, remote patient blood pressure monitoring, structured transition clinics, motivational lifestyle coaching, and health-equity-focused community interventions, this review outlines operational strategies for the nursing discipline to mitigate cardiovascular risk following hypertensive pregnancies.

Keywords:

Hypertensive Disorders of Pregnancy; Preeclampsia; Gestational Hypertension; Postpartum Cardiovascular Risk; Cardiovascular Disease; Nursing Interventions; Postpartum Care; Remote Patient Monitoring; Maternal Health; Cardiovascular Risk Prevention.

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Introduction and Background

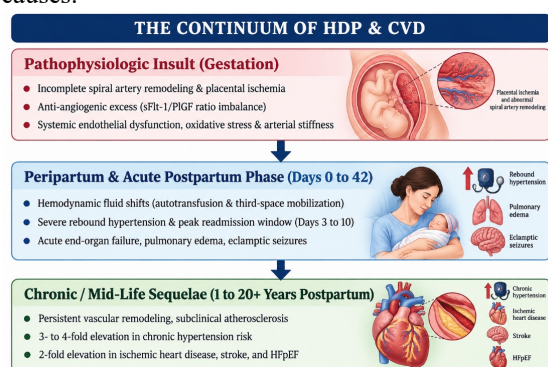
Cardiovascular disease (CVD) remains the principal cause of death among women globally. For decades, traditional Framingham and atherosclerotic cardiovascular disease (ASCVD) risk calculators categorized young, reproductive-aged women into low-risk strata, obscuring early disease trajectories. Emerging evidence demonstrates that pregnancy acts as a physiological "stress test" for the maternal metabolic and vascular beds. Under normal conditions, pregnancy induces extensive physiological adaptations: maternal blood volume expands by 40% to 50%, cardiac output increases by 30% to 50%, systemic vascular resistance drops through prostacyclin- and nitric oxide-mediated

vasodilation, and resting heart rate accelerates. In pregnancies compromised by hypertensive disorders, this stress test triggers decompensation, exposing underlying subclinical vascular fragility or inducing vascular injury that persists long after delivery (Valdés et al., 2017).

Hypertensive disorders of pregnancy (HDP) complicate approximately one in every ten pregnancies worldwide. Rather than transient pathologies isolated to gestation and resolving spontaneously following placental delivery, HDP produces lasting biological perturbations. Cohort studies reveal that individuals experiencing preeclampsia or gestational hypertension face

elevated trajectories of incident chronic hypertension, coronary artery calcification, myocardial infarction, heart failure with preserved ejection fraction (HFpEF), and ischemic stroke within five to twenty years postpartum (Wu et al., 2017; Mongraw-Chaffin et al., 2010).

Historically, postpartum management focused on acute maternal stabilization, neonatal transition, and contraception. Discharge marked the end of intensive surveillance, returning the patient to fragmented primary care. This fragmented paradigm contributes directly to postpartum maternal morbidity: over 50% of pregnancy-related maternal deaths occur after hospital discharge, with cardiovascular conditions, cardiomyopathy, and cerebrovascular accidents representing primary causes.



Professional nursing operates as an integrative discipline across hospital, outpatient, and community ecosystems. Nurses are strategically positioned to bridge acute obstetric care and long-term preventive cardiology. Delivering targeted nursing interventions requires an understanding of HDP pathophysiology, long-term cardiovascular epidemiology, and patient-centered clinical strategies.

Classification, Diagnostic Criteria, and Pathophysiology

Classification and Diagnostic Criteria

According to standard criteria established by the American College of Obstetricians and Gynecologists (ACOG) and the International Society for the Study of Hypertension in Pregnancy (ISSHP), HDP encompasses four diagnostic categories:

Diagnostic Classification	Clinical Criteria & Blood Pressure Cutoffs	Primary Diagnostic Distinctions
Chronic	SBP $\geq$	Pre-

Diagnostic Classification	Clinical Criteria & Blood Pressure Cutoffs	Primary Diagnostic Distinctions
Hypertension	140 mmHg or DBP $\geq$ 90 mmHg predating pregnancy or diagnosed before 20 weeks of gestation.	existing vascular pathology ; persists indefinitely beyond 12 weeks postpartum.
Gestational Hypertension	New-onset SBP $\geq$ 140 mmHg or DBP $\geq$ 90 mmHg after 20 weeks of gestation in the absence of proteinuria or systemic signs.	Blood pressure normalizes by 12 weeks postpartum; up to 50% may progress to preeclampsia.
Preeclampsia	New-onset hypertension after 20 weeks accompanied by proteinuria ($\geq$ 300 mg/24 h or protein-to-	Multisystem endothelial syndrome ; further classified as

Diagnostic Classification	Clinical Criteria & Blood Pressure Cutoffs	Primary Diagnostic Distinctions
	creatinine ratio ≥ 0.3), OR signs of end-organ dysfunction (thrombocytopenia, transaminitis, renal insufficiency, pulmonary edema, persistent cerebral/visual disturbances).	preeclampsia with or without severe features.
Chronic Hypertension with Superimposed Preeclampsia	Pre-existing chronic hypertension that develops sudden escalation of BP, acute onset of proteinuria, or systemic end-organ dysfunction.	Carries high risk of maternal morbidity, placental abruption, and preterm birth.

Preeclampsia with severe features is recognized when systolic blood pressure reaches ≥ 160 mmHg or diastolic reaches ≥ 110 mmHg on two separate occasions at least 4 hours apart while resting (unless

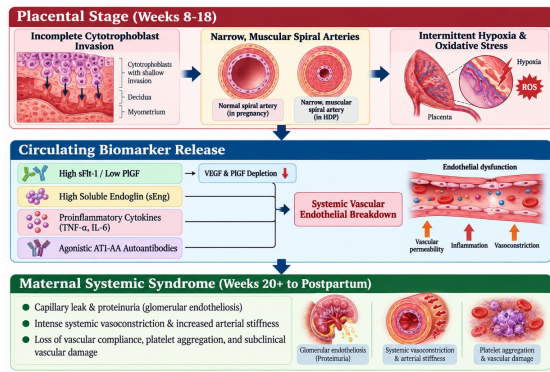
antihypertensives are started immediately), platelet counts drop below $100 \times 10^9/L$, serum transaminases rise to twice baseline normal, serum creatinine exceeds 1.1 mg/dL, or pulmonary edema manifests.

Cellular and Molecular Pathophysiology

The pathophysiology of preeclampsia centers on abnormal placentation followed by systemic maternal endothelial dysfunction. In early physiological human gestation, invasive extravillous cytotrophoblasts infiltrate the maternal decidua and myometrium, remodeling narrow, muscular maternal spiral arteries into high-capacitance, low-resistance conduits. This remodeling ensures continuous intervillous blood flow independent of maternal vasomotor tone.

In preeclampsia, this trophoblastic invasion is incomplete. Maternal spiral arteries remain narrow and muscular, causing chronic placental hypoperfusion, intermittent hypoxia, and reperfusion injury. Ischemia within the intervillous space induces local oxidative stress and cellular necrosis, causing the syncytiotrophoblast to release circulating anti-angiogenic and proinflammatory factors:

1. **Soluble Fms-Like Tyrosine Kinase-1 (sFlt-1):** A circulating splice variant of the vascular endothelial growth factor receptor 1 (VEGFR-1). sFlt-1 circulates at elevated levels, binding and neutralizing free vascular endothelial growth factor (VEGF) and placental growth factor (PlGF). Because basal endothelial maintenance and glomerular capillary fenestration require tonic VEGF signaling, excess sFlt-1 causes systemic endothelial breakdown, loss of endothelial nitric oxide synthase (eNOS) activation, and endotheliosis.
2. **Soluble Endoglin (sEng):** A circulating coreceptor for transforming growth factor-beta (TGF- β) that acts synergistically with sFlt-1. sEng blocks TGF- β signaling within vascular endothelial beds, inhibiting downstream capillary tube formation and impairing nitric-oxide-dependent vasodilation.
3. **Proinflammatory Cytokines and Angiotensin II Type-1 Receptor Autoantibodies (AT1-AA):** Ischemic placental debris triggers immune activation, marked by elevated tumor necrosis factor-alpha (TNF- α), interleukin-6 (IL-6), and AT1-AA. AT1-AA chronically stimulates angiotensin receptors on vascular smooth muscle, promoting ongoing vasoconstriction and aldosterone production.



This biological insult does not dissipate completely with the delivery of the placenta. While removal of the placenta arrests immediate gestational driver production, the systemic vascular and endothelial damage accrued during pregnancy often persists, leaving sustained subclinical vascular changes that accelerate cardiovascular disease progression (Valdés et al., 2017).

Postpartum Cardiovascular Risk Trajectories

Longitudinal epidemiological and observational studies confirm that experiencing an HDP serves as an early harbinger of cardiovascular disease across mid-life and advanced age.

Incident Chronic Hypertension and Arterial Stiffness

The most immediate cardiovascular sequel of HDP is premature chronic hypertension. In a landmark meta-analysis of over 3 million women, Bellamy et al. (2007) documented that women with a history of preeclampsia demonstrated an approximate 3.7-fold relative risk (95% CI: 2.70–5.05) of developing clinical chronic hypertension compared to women with normotensive pregnancies over a mean follow-up of 14 years.

Recent large multicenter prospective studies show that this transition occurs rapidly. Hauspurg et al. (2023) evaluated nulliparous women followed 2 to 7 years postpartum, establishing that individuals with gestational hypertension or preeclampsia demonstrated a 1.5- to 2-fold increased risk of developing incident Stage 1 or Stage 2 chronic hypertension within 3 to 5 years after delivery compared to their normotensive peers. Furthermore, pulse wave velocity (PWV) measurements demonstrate that women who experienced preeclampsia exhibit elevated central aortic stiffness and impaired flow-mediated dilation (FMD) within 6 to 12 months postpartum, confirming that structural arterial stiffening is already underway long before clinical coronary disease appears.

Ischemic Heart Disease and Coronary Calcification

The association between HDP and future ischemic heart disease (IHD) is robust and demonstrates a dose-dependent relationship based on the severity and timing of the hypertensive disorder. Systematic reviews and large population-based cohort studies indicate that women who experience preeclampsia

carry a 2.0- to 2.5-fold increased relative risk of fatal and non-fatal ischemic heart disease (Wu et al., 2017; Bellamy et al., 2007).

This risk scales according to clinical features:

- **Mild or late-term preeclampsia:** ~1.8- to 2.0-fold increased risk.
- **Severe, early-onset preeclampsia (<34 weeks of gestation):** Up to a 5- to 8-fold increase in myocardial infarction and cardiovascular mortality (Mongraw-Chaffin et al., 2010).
- **Recurrent preeclampsia across multiple pregnancies:** Marked acceleration of coronary disease, resulting in cardiac ischemic events occurring up to a decade earlier than in peers without complicated pregnancies (Lailier et al., 2024).

Imaging studies using coronary computed tomography angiography (CCTA) identify higher rates of coronary artery calcification (CAC) and coronary atherosclerotic plaque burden among asymptomatic women in their 40s who experienced preeclampsia, even after controlling for conventional cardiovascular risk factors.

Heart Failure and Left Ventricular Remodeling

Preeclampsia produces profound acute hemodynamic shifts, forcing the maternal left ventricle to pump against heightened afterload. Echocardiographic studies performed during active preeclampsia document elevated left ventricular mass index (LVMI), abnormal geometry (concentric hypertrophy), and impaired myocardial relaxation indicative of diastolic dysfunction.

While systolic ejection fraction typically remains preserved, subclinical diastolic impairment often persists postpartum. Long-term registries show that women with a history of preeclampsia face an adjusted 3- to 4-fold elevated risk of developing clinical heart failure later in life, with a pronounced tendency toward Heart Failure with Preserved Ejection Fraction (HFpEF).

Cerebrovascular Disease and Microvascular Injury

Hypertensive disorders during pregnancy heighten the lifetime risk of both ischemic and hemorrhagic stroke. Women with preeclampsia face an approximate 1.8- to 2.2-fold increased risk of remote cerebrovascular accidents (Wu et al., 2017; Bellamy et al., 2007). In the acute and subacute postpartum windows (days 1 to 42), severe unchecked hypertension accounts for severe cerebrovascular complications, such as Posterior Reversible Encephalopathy Syndrome (PRES) and intracranial hemorrhage. Long term, early cerebral microvascular injury and ongoing systemic endothelial impairment predispose these patients to silent lacunar infarcts, cerebral white matter hyperintensities, and premature cognitive decline.

Sociodemographic Factors, Disparities, and Health Equity

The intersection of hypertensive disorders of pregnancy and cardiovascular outcomes is marked by substantial racial, ethnic, and socioeconomic disparities. In high-income nations, structural racism, implicit clinical bias, socioeconomic disenfranchisement, and differential access to high-quality healthcare amplify both acute maternal complications and long-term cardiovascular risks:

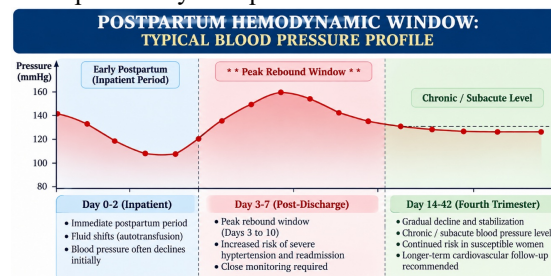
1. **Racial Disparities in Prevalence and Outcomes:** Black and Indigenous individuals experience higher rates of preeclampsia and are three to four times more likely to die from pregnancy-related cardiovascular causes than non-Hispanic white women. Black patients also face higher rates of early-onset, severe preeclampsia and earlier progression to refractory chronic hypertension.
2. **Social Determinants of Health (SDOH):** Food insecurity, housing instability, environmental chronic stress, lack of accessible transportation, and geographic distribution of maternity care deserts compound postpartum cardiovascular vulnerability. Chronic activation of the hypothalamic-pituitary-adrenal (HPA) axis from systemic discrimination and economic stress drives chronic inflammation, elevated sympathetic tone, and accelerated vascular aging—a process conceptualized as "weathering."
3. **Insurance Disruption and the "Postpartum Cliff":** In many healthcare jurisdictions, public medical insurance (such as pregnancy-related Medicaid in parts of the United States) historically lapsed 60 days postpartum. This abrupt coverage loss leaves vulnerable patients without ongoing medical supervision right when postpartum hemodynamics require careful titration of antihypertensive medications and metabolic surveillance. Expanding continuous postpartum insurance coverage to 12 months is an essential policy measure to preserve the fourth-trimester care continuum.

Acute and Subacute Nursing Interventions (Hospital Discharge to 6 Weeks Postpartum)

The initial six weeks following delivery represent an unstable physiological phase. Following placental separation, loss of the low-resistance uteroplacental circuit, maternal autotransfusion of 500 to 1,000 mL of blood from the contracting uterus, and rapid mobilization of extravascular third-spaced fluid into the central circulation abruptly increase left ventricular preload and cardiac output.

Blood pressure typically drops transiently over the first 48 hours, only to rebound and peak between

postpartum days 3 and 7—coinciding directly with hospital discharge. Consequently, comprehensive nursing interventions during this phase are vital for averting readmission, intracranial hemorrhage, and cardiopulmonary collapse.



Inpatient Assessment, Stabilization, and Discharge Preparation

Inpatient postpartum nursing care focuses on structured hemodynamic surveillance and proactive protocolized management:

- **Standardized Blood Pressure Assessment:** Blood pressure must be measured utilizing properly calibrated equipment with a cuff bladder encircling at least 80% of the upper arm, taken while the patient is seated quietly with back supported and feet uncrossed for a minimum of 5 minutes.
- **Rapid Treatment Protocols:** Nurses must execute standardized hospital safety bundles (e.g., California Maternal Quality Care Collaborative [CMQCC] or Alliance for Innovation on Maternal Health [AIM] bundles). Persistent severe hypertension (SBP ≥ 160 mmHg or DBP ≥ 110 mmHg verified for 15 minutes) requires immediate first-line IV antihypertensive treatment within 30 to 60 minutes:
 - **Intravenous Labetalol:** 20 mg IV push over 2 minutes, followed by 40 mg, then 80 mg every 10 to 20 minutes (maximum cumulative dose 300 mg).
 - **Intravenous Hydralazine:** 5 to 10 mg IV push over 2 minutes; repeat 10 mg in 20 minutes if needed.
 - **Oral Immediate-Release Nifedipine:** 10 to 20 mg capsules orally (useful when IV access is delayed or compromised).
- **Seizure Prophylaxis and Monitoring:** In cases of preeclampsia with severe features, intravenous Magnesium Sulfate (4 to 6 g loading dose over 20 minutes, followed by 1 to 2 g/h maintenance) is maintained intrapartum and for 24 hours postpartum. Nurses perform hourly assessments of patellar reflexes, respiratory rate (≥ 12 breaths/min), continuous pulse oximetry, and strict fluid balance through

an indwelling urinary catheter (alerting providers if urine output drops below 30 mL/h).

- **Pulmonary Edema Surveillance:** Extravasation of fluid into alveolar spaces represents an acute postpartum emergency. Nurses routinely auscultate pulmonary lung bases, evaluate oxygen saturation, and monitor for orthopnea, dry persistent cough, or unexpected tachycardia.

Patient Education on Critical Postpartum Warning Signs

Before discharge, nurses deliver structured health education using adult-learning methodologies such as the "teach-back" technique. Education must be tailored to ensure patients understand that preeclampsia can develop *de novo* up to 6 weeks postpartum:

- Severe, unremitting, throbbing frontotemporal headaches unresponsive to acetaminophen.
- Visual disturbances (scotomata, blurred vision, photophobia, diplopia).
- Persistent epigastric or right upper quadrant abdominal pain (indicating hepatic capsular distension).
- Sudden rapid facial, periorbital, or digital edema, or weight gain exceeding 3 to 5 pounds within 48 hours.
- Dyspnea, tachypnea, orthopnea, or chest tightness.

Remote Patient Monitoring (RPM) and Digital Health Implementations

Because maternal blood pressure peaks 3 to 7 days postpartum—when patients are recovering at home—home surveillance models are critical. Traditional recommendations requiring an in-person clinic check within 3 to 10 days often fail due to newborn care demands, maternal fatigue, lack of transport, and logistical barriers.

Nurse-led Remote Patient Monitoring (RPM) programs bridge this gap. Landmark implementations, such as the *Heart Safe Motherhood* initiative, provide patients at discharge with an automated, cellular-connected or Bluetooth-enabled blood pressure cuff and enrollment in a bidirectional text-messaging platform:



Patients transmit blood pressure readings twice daily

for 10 to 14 days postpartum. The clinical dashboard automatically alerts the supervising registered nurse to abnormal readings. Nurse-driven titration algorithms facilitate timely dose escalations of oral antihypertensives (such as oral extended-release nifedipine or labetalol), preventing acute re-hospitalizations and reducing racial disparities in postpartum blood pressure retention.

Long-Term and Chronic Nursing Interventions (The "Fourth Trimester" and Beyond)

Mitigating lifetime cardiovascular disease requires transitioning patients from acute obstetric services to primary care and preventive cardiology. When this handoff is unstructured, up to 70% of women who experience HDP are lost to follow-up, remaining unaware of their heightened cardiovascular risk (Countouris et al., 2025).

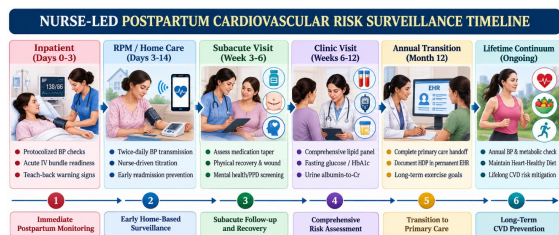
The Specialized Postpartum Heart Health Clinic Model

To overcome fragmented care, specialized multidisciplinary postpartum clinics—termed "Postpartum Heart Health Clinics" or "Fourth Trimester Cardio-Obstetrics Transitions"—have emerged. Often directed or coordinated by Advanced Practice Registered Nurses (APRNs), clinical nurse specialists, or nurse navigators, these clinics see patients at 6 to 12 weeks postpartum, continuing longitudinal care through 12 months.

Key components of the nurse-led clinic protocol include:

1. **Comprehensive Hemodynamic Evaluation:** Resting blood pressure assessment, assessment of orthostatic vitals, and evaluation for residual peripheral edema.
2. **Metabolic and Laboratory Profiling:** Screening for emerging dyslipidemia (total cholesterol, HDL, LDL, triglycerides), glucose intolerance (fasting glucose and hemoglobin A1c), and residual renal parenchymal or endothelial damage (serum creatinine, estimated glomerular filtration rate [eGFR], and urine albumin-to-creatinine ratio).
3. **Cardiovascular Risk Stratification:** Establishing a baseline cardiovascular trajectory. Because standard 10-year ASCVD risk estimators systematically underestimate risk in women under age 40, nurses integrate HDP as an independent "risk-enhancing factor," as endorsed by the AHA/ACC primary prevention guidelines.
4. **Formal Communication and Handoff:** Generating a formalized "Obstetric-Cardiovascular Transition Summary" transmitted directly to the patient's primary care clinician, detailing the specific gestational diagnosis, peak blood pressures, total medication requirements, and recommended annual surveillance

timelines.



Lifestyle Modification and Behavioral Coaching

Nurses excel in implementing behavior-change methodologies to promote sustainable lifestyle modifications. Grounded in Miller and Rollnick's motivational interviewing principles, nurses collaborate with patients to build realistic personal wellness goals that balance infant care demands:

- **Nutritional Guidance (The Mediterranean and DASH Patterns):** Emphasizing dietary sodium restriction (<2,300 mg daily, ideally moving toward 1,500 mg daily), rich consumption of leafy green vegetables, antioxidant-rich berries, whole grains, monounsaturated fats (extra virgin olive oil), and lean protein sources, which helps restore nitric oxide bioavailability and downregulate systemic inflammatory cytokines.
- **Prescription of Physical Activity:** Gradual resumption of aerobic activity, progressing toward guideline-directed targets of ≥ 150 minutes of moderate-intensity exercise (e.g., brisk walking, swimming, cycling) or 75 minutes of vigorous exercise weekly, paired with two weekly sessions of resistance training. Physical activity enhances skeletal muscle glucose disposal, mitigates insulin resistance, and directly reduces peripheral vascular resistance.
- **Weight Management and Metabolic Balancing:** Supporting gentle, sustained postpartum weight loss to return to a body mass index (BMI) between 18.5 and 24.9 kg/m², helping mitigate the progression toward metabolic syndrome.
- **Support and Preservation of Lactation:** Nurses educate patients that sustained lactation exerts measurable protective effects on maternal cardiovascular metabolic profiles. Breastfeeding for a cumulative duration exceeding six months is associated with decreased maternal risks of incident hypertension, type 2 diabetes, and hyperlipidemia through oxytocin-mediated vascular calming and maternal metabolic resetting.

Psychosocial Support and Trauma-Informed Nursing

Experiencing an acute hypertensive crisis during pregnancy—often culminating in emergency

cesarean section, preterm birth, or neonatal intensive care unit (NICU) admission—causes considerable psychological distress. Up to 30% of women who experience severe preeclampsia meet criteria for postpartum depression (PPD), generalized anxiety disorder, or post-traumatic stress disorder (PTSD).

Psychological stress and cortisol overproduction induce sustained adrenergic activity, contributing to chronic arterial hypertension. Nurses implement routine mental health screening using the Edinburgh Postnatal Depression Scale (EPDS) and the Generalized Anxiety Disorder-7 (GAD-7) scale at each postpartum clinical encounter, facilitating prompt linkages to behavioral health professionals and trauma-informed support groups.

Evidence-Based Nursing Care Plan

To provide a concrete clinical template, the following care plan outlines three priority nursing diagnoses for a postpartum patient following a hypertensive disorder of pregnancy, utilizing NANDA-I nursing diagnoses, Nursing Outcomes Classification (NOC), and Nursing Interventions Classification (NIC) frameworks.

Nursing Diagnosis 1

- **Diagnostic Statement:** Ineffective Peripheral Tissue Perfusion related to persistent systemic arteriolar vasoconstriction and vascular endothelial damage, as evidenced by elevated blood pressure (154/96 mmHg), systemic edema, and a history of preeclampsia with severe features.
- **Desired Outcomes (NOC):**
 - *Tissue Perfusion: Peripheral:* Systolic blood pressure maintained between 110–139 mmHg and diastolic between 70–89 mmHg; absence of end-organ symptoms (headaches, scotomata).
 - *Vital Signs:* Patient demonstrates stable blood pressure readings within individualized target parameters across home monitoring logs.
- **Nursing Interventions (NIC) & Rationale:**
 - *Hemodynamic Management:* Obtain resting blood pressure following standard positioning protocols every 4 hours while hospitalized and twice daily via home monitoring; timely identification prevents sudden malignant hypertensive spikes and cerebrovascular accidents.
 - *Medication Administration:* Administer antihypertensive agents (e.g., oral nifedipine extended-release 30 mg daily) as

prescribed; titrate based on standing medical parameters to reduce systemic vascular resistance while maintaining renal perfusion.

- *Fluid Monitoring:* Monitor intake and output; assess for signs of fluid overload or pulmonary crackles to prevent pulmonary edema during post-delivery autotransfusion phases.

Nursing Diagnosis 2

- **Diagnostic Statement:** Deficient Knowledge regarding the association between hypertensive disorders of pregnancy and long-term cardiovascular disease risk, related to insufficient information exposure and discharge communication gaps, as evidenced by the patient stating, "Now that my baby is delivered, my high blood pressure is completely cured."
- **Desired Outcomes (NOC):**
 - *Knowledge: Cardiovascular Disease Prevention:* Patient describes the pathophysiologic link between preeclampsia and future cardiovascular risks; identifies at least three modifiable risk factors.
 - *Health Literacy:* Patient demonstrates accurate use of an automated home blood pressure monitor and recites critical postpartum red-flag warning signs requiring emergency care.
- **Nursing Interventions (NIC) & Rationale:**
 - *Health Education: Postpartum Recovery:* Conduct a structured teaching session prior to discharge utilizing teach-back methodology; clarifying that placental delivery stops active anti-angiogenic production but leaves potential vascular risks empowers patients to engage in long-term surveillance.
 - *Discharge Planning / Transition Management:* Provide written and digital education detailing the need for lifetime annual blood pressure, lipid, and glycemic screening; establishing an obstetric-to-primary care handoff closes transitional care gaps.
 - *Demonstration / Practice:* Instruct patient on proper home blood pressure cuff placement, posture, and resting requirements

prior to obtaining measurements; accurate technique ensures reliable data for medication titration.

Nursing Diagnosis 3

- **Diagnostic Statement:** Readiness for Enhanced Health Management related to a expressed desire to adopt long-term heart-healthy behaviors following a high-risk pregnancy, as evidenced by inquiries regarding appropriate nutrition, safe postpartum physical exercise, and cardiovascular prevention.
- **Desired Outcomes (NOC):**
 - *Health Promoting Behavior:* Patient establishes a weekly target for physical activity; adheres to dietary modifications consistent with the DASH or Mediterranean eating plan.
 - *Self-Efficacy:* Patient expresses confidence in personal capacity to maintain lifelong cardiovascular self-care practices while caring for an infant.
- **Nursing Interventions (NIC) & Rationale:**
 - *Motivational Interviewing & Lifestyle Coaching:* Engage in collaborative, goal-directed dialogue to identify realistic, incremental lifestyle adjustments; client-centered goals enhance long-term compliance compared to didactic instructions.
 - *Nutritional Counseling:* Collaborate with a registered dietitian to provide concrete examples of low-sodium, nutrient-dense meal plans that accommodate cultural food preferences and lactation caloric demands; dietary intervention improves lipid profiles and long-term endothelial function.
 - *Referral to Community & Peer Support:* Connect patient with local and digital peer-led support groups (e.g., Preeclampsia Foundation); peer community validation mitigates social isolation and reinforces sustained health management.

Health Policy, Clinical Systems, and Nursing Leadership

Addressing maternal cardiovascular health demands broader institutional and health policy reforms. Nursing leaders play central roles in driving these systemic improvements:



- 1. Electronic Health Record (EHR) Architecture and Decision Support:** Nursing informatics leaders must advocate for structural modifications within hospital and ambulatory EHRs. A history of HDP must be permanently recorded as an enduring cardiovascular risk-enhancing factor, triggering automated clinical decision support prompts during annual wellness examinations to monitor blood pressure, lipid fractions, and fasting blood glucose.
- 2. Legislative Advocacy for Extended Postpartum Coverage:** Professional nursing associations (such as the Association of Women's Health, Obstetric and Neonatal Nurses [AWHONN] and the American Nurses Association [ANA]) continue to champion policy initiatives that expand continuous postpartum health insurance coverage from 60 days to a minimum of 12 full months. Uninterrupted coverage ensures patients with persistent postpartum hypertension or metabolic abnormalities receive continuous medical and nursing management.
- 3. Equitable Community-Clinic Linkages:** Healthcare institutions must invest in community public health nurse home-visiting models (such as the Nurse-Family Partnership) and deploy mobile health outreach units. Expanding care into underserved regions and culturally tailoring clinical interventions reduces systemic health disparities and ensures equitable care delivery.

Conclusion

Hypertensive disorders of pregnancy are critical indicators of future cardiovascular disease, rather than self-limiting obstetric events. The vascular, endothelial, and metabolic challenges seen during preeclampsia and gestational hypertension offer an early clinical window to identify individuals at elevated risk for future hypertension, myocardial infarction, heart failure, and stroke.

Nurses possess the clinical expertise, communicative proficiency, and holistic view of patient care needed to bridge the fourth trimester

transition. By leading acute inpatient protocolized care, managing digital remote patient monitoring networks, coordinating structured cardio-obstetrics bridge clinics, and delivering trauma-informed lifestyle coaching, the nursing profession helps transform a complicated pregnancy from an overlooked risk factor into an actionable opportunity for lifelong cardiovascular health.

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