

Pregnancy-Induced Hypertension

The exact etiology of this disorder is unknown, with several theories being advanced. Data have suggested that PIH may be the result of increased peripheral vascular resistance secondary to generalized vasospasm when the vessels are no longer refractory to the effects of pressor agents. New research proposes that elevated cardiac output and associated hyperdynamic vasodilation during the first trimester result in damage to the endothelium with compensatory vascular vasodilation. Regardless of the mechanisms, PIH is a chronic disease process, with a decrease in placental perfusion occurring before the late sign of hypertension is detected.

(This plan of care is to be used in conjunction with the Trimesters and the High-Risk Pregnancy CPs.)

CLIENT ASSESSMENT DATA BASE

Circulation

Persistent increase in BP over pregravid or first-trimester baseline readings after 20 wk of pregnancy (systolic elevation > 30 mm Hg, diastolic elevation > 15 mm Hg or a BP > 140/90 mm Hg on two consecutive readings assessed at least 6 hr apart).

History of chronic hypertension.

Jugular venous distension.

Gallop rhythm may be present.

Pulse may be decreased.

May have spontaneous bruising, prolonged bleeding, or epistaxis (thrombocytopenia).

Elimination

Oliguria/anuria may be present.

Hematuria may be noted.

Food/Fluid

Nausea/vomiting.

Weight gain of 2+ lb in 1 wk, 4 lb or more per month (depending on length of gestation).

Malnourished (overweight or underweight by 20% or greater); poor protein/caloric intake.

Edema may be present, ranging from mild to severe/generalized; and may involve facies, extremities, and organ systems (i.e., liver, brain).

Glycosuria (diabetes mellitus).

Neurosensory

Dizziness, frontal headaches.

Decreased responsiveness/somnolence.

Diplopia, blurred vision, or even loss of visual fields; scotomata (spots before eyes).

Hyperreflexia; clonus.

Convulsions—tonic, then tonic-clonic phases, followed by a period of loss of consciousness.

Funduscopy examination may reveal edema or vascular spasm.

Pain/Discomfort

Epigastric pain (right upper quadrant [RUQ] region)

Respiration

Respirations may be less than 14/min.

Bibasilar crackles may be present.

Dyspnea.

Safety

Rh incompatibility may be present.

Sexuality

Primigravida, multiple gestation, hydramnios, gestational trophoblastic disease (e.g.,: hydatidiform mole), hydrops fetalis (Rh antigen-antibody)

Fetal movement may be diminished

Signs of abruptio placentae may be present (e.g., uterine tetany, tenderness)

Teaching/Learning

Adolescent (under age 15 yr) and older primigravida (age 35 yr or older) are at greatest risk

Family history of pregnancy-induced hypertension (PIH)

DIAGNOSTIC STUDIES

Supine Pressor Test (“rollover test”): May be used to screen for clients at risk for PIH, 28–32 wk gestation, although accuracy is questionable; an increase of 20–30 mm Hg in systolic pressure or 15–20 mm Hg in diastolic pressure indicates a positive test.

Mean Arterial Pressure (MAP): 90 mm Hg at second trimester indicates PIH.

Hematocrit (Hct): Elevated with fluid shifts, or decreased in HELLP syndrome (*hemolysis, elevated liver enzymes, low platelet count*).

Hemoglobin (Hb): Low when hemolysis occurs (HELLP syndrome).

Peripheral Smear: Distended blood cells or schistocytes in HELLP syndrome or intravascular hemolysis.

Serum Platelet Counts: Less than 100,000/mm³ in disseminated intravascular coagulation (DIC) or in HELLP syndrome, because platelets adhere to collagen released from damaged blood vessels.

Serum Creatinine Level: Elevated above 1.0 mg/dL and blood urea nitrogen (BUN): Greater than 10 mg/dL reflects severe renal involvement.

AST, LDH, and Serum Bilirubin Levels (indirect particularly): Elevated in HELLP syndrome with liver involvement.

Uric Acid Level: Greater than 5 mg/100 mL. Helpful in distinguishing preeclampsia from uncomplicated chronic hypertension.

Prothrombin time (PT), Partial Thromboplastin Time (PTT), Clotting Time: Prolonged; fibrinogen decreased, FSP and FDP positive when coagulopathy occurs (DIC).

Urine-Specific Gravity: Elevated, reflecting fluid shifts/vascular dehydration.

Proteinuria: By dipstick, may be 1+ to 2+ (moderate), 3+ to 4+ (severe), or greater than 500 mg/dL in 24 h.

Creatinine Clearance: Decreased in preeclampsia (before serum BUN/Creatinine elevated).

Urinary/Plasma Estriol Levels: Decline indicates reduced placental functioning. (Estriols are not as useful a predictor as biophysical profile [BPP] because of the lag time between fetal problem and test results.)

Human Placental Lactogen Levels: Less than 4 mEq/ml suggests abnormal placental functioning (not frequently done in PIH screening).

Ultrasonography: At 20–26 weeks’ gestation and repeated 6–10 wk later, establishes gestational age and detects IUGR.

Tests of Amniotic Fluid (lecithin sphingomyelin [L/S] ratio, phosphatidylglycerol (PG), saturated phosphatidylcholine levels): Determine fetal lung maturity.

Biophysical Profile (BPP) (amniotic fluid volume, fetal tone, fetal breathing movements [FMB], fetal

movements, and fetal heart rate [FHR] reactivity)/Nonstress Test (NST): Determines fetal risk/well-being.

CST: Assesses the response of the fetus to the stress of uterine contractions.

NURSING PRIORITIES

1. Monitor maternal, fetal, and placental status.
2. Prevent or reduce progressive fluid accumulation and other complications.
3. Promote positive maternal/fetal outcome.
4. Provide information to enhance self-care and therapeutic management.

DISCHARGE GOALS

(Inpatient care generally not required, unless fetal compromise or eclampsia develops, or when labor process begins.)

If hospitalized:

1. Hemodynamically stable, free-of-seizure activity
2. Fetus active and in no distress
3. Condition, prognosis, therapeutic regimen understood
4. Participating in care with plan in place for home monitoring/management

NURSING DIAGNOSIS:

May Be Related To:

Possibly Evidenced By:

**DESIRED OUTCOMES/EVALUATION
CRITERIA—CLIENT WILL:**

Fluid Volume deficit [isotonic]

Plasma protein loss, decreasing plasma colloid osmotic pressure, allowing fluid shifts out of the vascular compartment

Edema formation, sudden weight gain, hemoconcentration, nausea/vomiting, epigastric pain, headaches, visual changes, decreased urine output

Verbalize understanding of need for close monitoring of weight, BP, urine protein, and edema.

Participate in therapeutic regimen and monitoring, as indicated.

Display Hct WNL and physiological edema with no signs of pitting.

Be free of signs of generalized edema (i.e., epigastric pain, cerebral symptoms, dyspnea, nausea/vomiting).

ACTIONS/INTERVENTIONS

RATIONALE

Independent

Weigh client routinely. Encourage client to monitor weight at home between visits.

Sudden, significant weight gain (e.g., more than 3.3 lb (1.5 kg)/month in the second trimester or more than 1 lb (0.5 kg)/wk in the third trimester) reflects fluid retention. Fluid moves from the vascular to interstitial space, resulting in edema.

Distinguish between physiological and pathological edema of pregnancy. Monitor location and degree of pitting.

The presence of pitting edema (mild, 1+ to 2+; severe, 3+ to 4+) of face, hands, legs, sacral area, or abdominal wall, or edema that does not disappear after 12 hr of bedrest is significant. Note: Significant edema may actually be present in nonpre-eclamptic clients and absent in clients with mild or moderated PIH.

Note signs of progressive or excessive edema (i.e., epigastric/RUQ pain, cerebral symptoms, nausea, vomiting). Assess for possible eclampsia. (Refer to ND: Injury, risk for maternal.)

Edema and intravascular fibrin deposition (in HELLP syndrome) within the encapsulated liver are manifested by RUQ pain; dyspnea, indicating pulmonary involvement; cerebral edema, possibly leading to seizures; and nausea and vomiting, indicating GI edema.

Note changes in Hct/Hb levels.

Identifies degree of hemoconcentration caused by fluid shift. If Hct is less than 3 times Hb level, hemoconcentration exists.

Reassess dietary intake of proteins and calories.
Provide information as needed.

Monitor intake and output. Note urine color, and measure specific gravity as indicated.

Test clean, voided urine for protein each visit, or daily/hourly as appropriate if hospitalized.
Report readings of 2+, or greater.

Assess lung sounds and respiratory rate/effort.

Monitor BP and pulse. (Refer to ND: Cardiac Output, decreased.)

Answer questions and review rationale for avoiding use of diuretics to treat edema.

Collaborative

Schedule prenatal visit every 1–2 wk if PIH is mild; weekly if severe.

Review moderate sodium intake of up to 6 g/day. Instruct client to read food labels and avoid foods high in sodium (e.g., bacon, luncheon meats, hot dogs, canned soups, and potato chips).

Refer to dietitian as indicated.

Place client on strict regimen of bedrest; encourage lateral position.

Refer to home monitoring/day-care program, as appropriate.

Adequate nutrition reduces incidence of prenatal hypovolemia and hypoperfusion; inadequate protein/calories increases the risk of edema formation and PIH. Intake of 80–100 g of protein may be required daily to replace losses.

Urine output is a sensitive indicator of circulatory blood volume. Oliguria and specific gravity of 1.040 indicate severe hypovolemia and kidney involvement. Note: Administration of magnesium sulfate (MgSO_4) may cause transient increase in output.

Aids in determining degree of severity/progression of condition. A 2+ reading suggests glomerular edema or spasm. Proteinuria affects fluid shifts from the vascular tree. Note: Urine contaminated by vaginal secretions may test positive for protein, or dilution may result in a false-negative result. In addition, PIH may be present without significant proteinuria.

Dyspnea and crackles may indicate pulmonary edema, which requires immediate treatment.

Elevation in BP may occur in response to catecholamines, vasopressin, prostaglandins, and, as recent findings suggest, decreased levels of prostacyclin.

Diuretics further increase state of dehydration by decreasing intravascular volume and placental perfusion, and they may cause thrombocytopenia, hyperbilirubinemia, or alteration in carbohydrate metabolism in fetus/newborn. Note: May be useful in treating pulmonary edema.

Necessary to monitor changes more closely for the well-being of the client and fetus.

Some sodium intake is necessary because levels below 2–4 g/day result in greater dehydration in some clients. However, excess sodium may increase edema formation.

Nutritional consult may be beneficial in determining individual needs/dietary plan.

Lateral recumbent position decreases pressure on the vena cava, increasing venous return and circulatory volume. This enhances placental and renal perfusion, reduces adrenal activity, and may lower BP as well as account for weight loss through diuresis of up to 4 lb in 24-hr period.

Some mildly hypertensive clients without proteinuria may be managed on an outpatient basis if adequate surveillance and support is provided and the client/family actively participates in the treatment regimen.

Replace fluids either orally or parenterally via infusion pump, as indicated.

Fluid replacement corrects hypovolemia, yet must be administered cautiously to prevent overload, especially if interstitial fluid is drawn back into circulation when activity is reduced. With renal involvement, fluid intake is restricted; i.e., if output is reduced (less than 700 ml/24 hr), total fluid intake is restricted to approximate output plus insensible loss. Use of infusion pump allows more accurate control delivery of IV fluids.

When fluid deficit is severe and client is hospitalized:

Insert indwelling catheter if kidney output is reduced or is less than 50 ml/hr.
Assist with insertion of lines and/or monitoring of invasive hemodynamic parameters, such as CVP and pulmonary artery wedge pressure (PAWP).
Monitor serum uric acid and creatinine levels, and BUN.

Administer platelets as indicated.

below 20,000.

Allows more accurate monitoring of output/renal perfusion.
Provides a more accurate measurement of fluid volume. In normal pregnancy, plasma volume increases by 30%–50%, yet this increase does not occur in the client with PIH.
Elevated levels, especially of uric acid, indicate impaired kidney function, worsening of maternal condition, and poor fetal outcome.
Clients with HELLP syndrome awaiting delivery of the fetus may benefit from transfusion of platelets when count is

NURSING DIAGNOSIS:

Cardiac Output, decreased

May Be Related To:

Hypovolemia/decreased venous return, increased systemic vascular resistance

Possibly Evidenced By:

Variations in blood pressure/hemodynamic readings, edema, shortness of breath, change in mental status

DESIRED OUTCOMES/EVALUATION CRITERIA—CLIENT WILL:

Remain normotensive throughout remainder of pregnancy.

Report absence and/or decreased episodes of dyspnea.

Alter activity level as condition warrants.

ACTIONS/INTERVENTIONS

RATIONALE

Independent

Monitor and graph BP and pulse.

The client with PIH does not manifest the normal cardiovascular response to pregnancy (left ventricular hypertrophy, increase in plasma volume, vascular relaxation with decreased peripheral resistance). Hypertension (the second manifestation of PIH after edema) occurs owing to increased sensitization to angiotensin II, which increases BP, promotes aldosterone release to increase sodium/water reabsorption from the renal tubules, and constricts blood vessels.

Assess MAP at 22 weeks' gestation. A pressure of 90 mm Hg is considered predictive of PIH. Assess for crackles, wheezes, and dyspnea; note respiratory rate/effort.

Institute bedrest with client in lateral position.

Collaborative

Monitor invasive hemodynamic parameters.

Administer antihypertensive drug such as hydralazine (Apresoline) PO/IV, so that diastolic readings are between 90 and 105 mm Hg. Begin maintenance therapy as needed, e.g., methyldopa (Aldomet) or nifedipine (Procardia).

Monitor BP and side effects of antihypertensive drugs. Administer propranolol (Inderal), as appropriate.

Prepare for birth of fetus by cesarean delivery, when severe PIH/eclamptic condition is stabilized, but vaginal delivery is not feasible.

Pulmonary edema may occur, with changes in peripheral vascular resistance and decline in plasma colloid osmotic pressure.

Increases venous return, cardiac output, and renal/placental perfusion.

Provides accurate picture of vascular changes and fluid volume. Prolonged vascular constriction, increased hemoconcentration, and fluid shifts decrease cardiac output.

If BP does not respond to conservative measures, short-term medication may be necessary in conjunction with other therapies, e.g., fluid replacement and MgSO₄. Antihypertensive drugs act directly on arterioles to promote relaxation of cardiovascular smooth muscle and help increase blood supply to cerebrum, kidneys, uterus, and placenta. Hydralazine is the drug of choice because it does not produce effects on the fetus. Sodium nitroprusside is being used with some success to lower BP (especially in HELLP syndrome).

Side effects such as tachycardia, headache, nausea, and vomiting, and palpitations may be treated with propranolol.

If conservative treatment is ineffective and labor induction is ruled out, then surgical procedure is the only means of halting the hypertensive problems.

NURSING DIAGNOSIS:

May Be Related To:

Possibly Evidenced By:

DESIRED OUTCOMES/EVALUATION CRITERIA—FETUS WILL:

Tissue Perfusion, altered: uteroplacental

Maternal hypovolemia, interruption of blood flow (progressive vasospasm of spiral arteries)

Intrauterine growth retardation, changes in fetal activity/heart rate, premature delivery, fetal demise

Demonstrate normal CNS reactivity on nonstress test (NST); be free of late decelerations; have no decrease in FHR on contraction stress test/oxytocin challenge test (CST/OCT).

Be full-term, AGA.

ACTIONS/INTERVENTIONS

RATIONALE

Independent

Provide information to client/couple regarding home assessment/recording of daily fetal movements and when to seek immediate medical attention.

Reduced placental blood flow results in reduced gas exchange and impaired nutritional functioning of the placenta. Potential outcomes of poor placental perfusion include a malnourished, LBW infant, and prematurity associated with early delivery, abruptio placentae, and fetal death. Reduced fetal activity indicates fetal compromise (occurs before detectable alteration in FHR and indicates need for immediate evaluation/intervention).

Identify factors affecting fetal activity.

Cigarette smoking, medication/drug use, serum glucose levels, environmental sounds, time of day, and sleep-wake cycle of the fetus can increase or decrease fetal movement.

Review signs of abruptio placentae (i.e., vaginal bleeding, uterine tenderness, abdominal pain, and decreased fetal activity).

Prompt recognition and intervention increases the likelihood of a positive outcome.

Provide contact number for client to ask questions, report changes in daily fetal movements, and so forth.

Provides opportunity to address concerns/misconceptions and intervene in a timely manner, as indicated.

Evaluate fetal growth; measure progressive fundal growth at each office visit or periodically during home visits, as appropriate.

Decreased placental functioning may accompany PIH, resulting in IUGR. Chronic intrauterine stress and uteroplacental insufficiency decrease amount of fetal contribution to amniotic fluid pool.

Note fetal response to medications such as MgSO₄, phenobarbital, and diazepam.

Depressant effects of medication reduce fetal respiratory and cardiac function and fetal activity level, even though placental circulation may be adequate.

Monitor FHR manually or electronically, as indicated.

Helps evaluate fetal well-being. An elevated FHR may indicate a compensatory response to hypoxia, prematurity, or abruptio placentae.

Collaborative

Assess fetal response to BPP criteria or CST, as maternal status indicates. (Refer to ND: Injury, risk for maternal.)

BPP helps evaluate fetus and fetal environment on five specific parameters to assess CNS function and fetal contribution to amniotic fluid volume. CST assesses placental functioning and reserves.

Assist with assessment of fetal maturity and well-being using L/S ratio, presence of PG, estriol levels, FBM, and sequential sonography beginning at 20–26 weeks' gestation. (Refer to CP: The High-Risk Frequency; ND: Injury, risk for fetal.)

In the event of deteriorating maternal/fetal condition, risks of delivering a preterm infant are weighed against the risks of continuing the pregnancy, using results from evaluative studies of lung and kidney maturity, fetal growth, and placental functioning. IUGR is associated with reduced maternal volume and vascular changes.

Assist with assessment of maternal plasma volume at 24–26 weeks' gestation using Evans' blue dye when indicated.

Identifies fetus at risk for IUGR or intrauterine fetal demise associated with reduced plasma volume and reduced placental perfusion.

Using ultrasonography, assist with assessment of placental size.

Decreased placental function and size are associated with PIH.

Administer corticosteroid (dexamethasone, betamethasone) IM for at least 24–48 hr, but not more than 7 days before delivery, when severe PIH necessitates premature delivery between 28 and 34 weeks' gestation.

Corticosteroids are thought to induce fetal pulmonary maturity (surfactant production) and prevent respiratory distress syndrome, at least in a fetus delivered prematurely because of condition or inadequate placental functioning. Best results are obtained when fetus is less than 34 weeks' gestation and delivery occurs within a week of corticosteroid administration.

NURSING DIAGNOSIS:**May Be Related To:****Possibly Evidenced By:****DESIRED OUTCOMES/EVALUATION CRITERIA—CLIENT WILL:****DESIRED OUTCOMES/EVALUATION CRITERIA—CLIENT WILL (cont.):****Injury, risk for maternal**

Tissue edema/hypoxia, tonic-clonic convulsions, abnormal blood profile and/or clotting factors

[Not applicable: Presence of signs/symptoms establishes an *actual* diagnosis]

Participate in treatment and/or environmental modifications to protect self and enhance safety.

Be free of signs of cerebral ischemia (visual disturbances, headache, changes in mentation).

Display normal levels of clotting factors and liver enzymes.

ACTIONS/INTERVENTIONS

RATIONALE**Independent**

Assess for CNS involvement (i.e., headache, irritability, visual disturbances or changes on funduscopic examination).

Stress importance of client promptly reporting signs/symptoms of CNS involvement.

Note changes in level of consciousness.

Assess for signs of impending eclampsia: hyperactivity of deep tendon reflexes (3+ to 4+), ankle clonus, decreased pulse and respirations, epigastric pain, and oliguria (less than 50 ml/hr).

Institute measures to reduce likelihood of seizures; i.e., keep room quiet and dimly lit, limit visitors, plan and coordinate care, and promote rest.

Implement seizure precautions per protocol.

In the event of a seizure:

Turn client on side; insert airway/bite block only if mouth is relaxed; suction nasopharynx, as indicated; administer oxygen; remove restrictive clothing; do not restrict movement. Document motor involvement, duration of seizure, and postseizure behavior.

Palpate for uterine tenderness or rigidity; check for vaginal bleeding. Note history

Cerebral edema and vasoconstriction can be evaluated in terms of symptoms, behaviors, or retinal changes.

Delayed treatment or progressive onset of symptoms may result in tonic-clonic convulsions or eclampsia.

In progressive PIH, vasoconstriction and vasospasms of cerebral blood vessels reduce oxygen consumption by 20% and result in cerebral ischemia.

Generalized edema/vasoconstriction, manifested by severe CNS, kidney, liver, cardiovascular, and respiratory involvement, precede convulsive state.

Reduces environmental factors that may stimulate irritable cerebrum and cause a convulsive state.

If seizure does occur, reduces risk of injury.

Maintains airway by reducing risk of aspiration and preventing tongue from occluding airway. Maximizes oxygenation. Note: Be cautious with use of airway/bite block, because attempts to insert when jaws are set may result in injury.

These signs may indicate abruptio placentae, especially if there is a preexisting medical problem,

of other medical problems.

Monitor for signs and symptoms of labor or uterine contractions.

Assess fetal well-being, noting FHR.

Monitor for signs of DIC easy/spontaneous bruising, prolonged bleeding, epistaxis, GI bleeding. (Refer to CP: Prenatal Hemorrhage.)

Collaborative

Hospitalize if CNS involvement is present.

Administer MgSO₄ IM or IV using infusion pump.

Monitor BP before, during, and after MgSO₄ administration. Note serum magnesium levels in conjunction with respiratory rate, patellar/deep tendon reflex (DTRs), and urine output.

Have calcium gluconate available. Administer 10 ml (1 g/10 ml) over 3 min as indicated.

Administer amobarbital (Amytal) or diazepam (Valium), as indicated.

Perform funduscopic examination daily.

Monitor test results of clotting time, PT, PTT, fibrinogen levels, and FPS/FDP.

Monitor sequential platelet count. Avoid amniocentesis if platelet count is less than 50,000/mm³. If thrombocytopenia is present during operative procedure, use general anesthesia. Transfuse with platelets, packed red blood cells, fresh frozen plasma, or whole blood, as indicated. Rule out HELLP syndrome.

such as diabetes mellitus, or a renal or cardiac disorder causing vascular involvement.

Convulsions increase uterine irritability; labor may ensue.

During seizure activity, fetal bradycardia may occur.

Abruptio placentae with release of thromboplastin predisposes client to DIC.

Prompt initiation of therapy helps to ensure safety and limit complications.

MgSO₄ a CNS depressant, decreases acetylcholine release, blocks neuromuscular transmission, and prevents seizures. It has a transient effect of lowering BP and increasing urine output by altering vascular response to pressor substances. Although IV administration of MgSO₄ is easier to regulate and reduces the risk of a toxic reaction, some facilities may still use the IM route if continuous surveillance is not possible and/or if appropriate infusion apparatus is not available. Note: Adding 1 ml of 2% lidocaine to the IM injection may reduce associated discomfort. (Current research suggests the use of phenytoin infusion may be effective in the treatment of PIH without the adverse side effects, such as respiratory depression, and tocolytic effect on uterine smooth muscle, which can impede labor during intrapartum therapy.)

A therapeutic level of MgSO₄ is achieved with serum levels of 4.0–7.5 mEq/L or 6–8 mg/dL. Adverse/toxic reactions develop above 10–12 mg/dL, with loss of DTRs occurring first, respiratory paralysis between 15–17 mg/dL, or heart block occurring at 30–35 mg/dL.

Serves as antidote to counteract adverse/toxic effects of MgSO₄.

Depresses cerebral activity; has sedative effect when convulsions are not controlled by MgSO₄. Not recommended as first-line therapy because sedative effect also extends to the fetus.

Helps to evaluate changes or severity of retinal involvement.

Such tests can indicate depletion of coagulation factors and fibrinolysis, which suggests DIC.

Thrombocytopenia may occur because of platelet adherence to disrupted endothelium or reduced prostacyclin levels (a potent inhibitor of platelet aggregation). Invasive procedures or anesthesia requiring needle puncture (such as spinal/epidural) could result in excessive bleeding.

Monitor liver enzymes and bilirubin; note hemolysis and presence of Burr cells on peripheral smear.

Prepare for cesarean birth if PIH is severe, placental functioning is compromised, and cervix is not ripe or is not responsive to induction.

Elevated liver enzyme (AST, ALT) and bilirubin levels, microangiopathic hemolytic anemia, and thrombocytopenia may indicate presence of HELLP syndrome, signifying a need for immediate cesarean delivery if condition of cervix is unfavorable for induction of labor.

When fetal oxygenation is severely reduced owing to vasoconstriction within malfunctioning placenta, immediate delivery may be necessary to save the fetus.

NURSING DIAGNOSIS:

May Be Related To:

Possibly Evidenced By:

**DESIRED OUTCOMES/EVALUATION
CRITERIA—CLIENT WILL:**

**DESIRED OUTCOMES/EVALUATION
CRITERIA—CLIENT WILL (cont.):**

Nutrition: altered, risk for less than body requirements

Intake insufficient to meet metabolic demands and replace losses

[Not applicable; presence of signs/symptoms establishes an *actual* diagnosis]

Verbalize understanding of individual dietary needs.

Demonstrate knowledge of proper diet as evidenced by developing a dietary plan within own financial resources.

Display appropriate weight gain.

ACTIONS/INTERVENTIONS

RATIONALE**Independent**

Assess client's nutritional status, condition of hair and nails, and height and pregravid weight.

Establishes guidelines for determining dietary needs and educating client. Malnutrition may be a contributing factor to the onset of PIH, specifically when client follows a low-protein diet, has insufficient caloric intake, and is overweight or underweight by 20% or more before conception.

Provide information about normal weight gain in pregnancy, modifying it to meet client's needs.

The underweight client may need a diet higher in calories; the obese client should avoid dieting because it places the fetus at risk for ketosis.

Provide oral/written information about action and uses of protein and its role in development of PIH.

Daily intake of 80–100 g/day (1.5 g/kg) is sufficient to replace proteins lost in urine and allow for normal serum oncotic pressure.

Provide information about effect of bedrest and reduced activity on protein requirements.

Reducing metabolic rate through bedrest and limited activity decreases protein needs.

Collaborative

Refer to dietitian, as indicated.

Helpful in creating individual dietary plan incorporating specific needs/restrictions.

NURSING DIAGNOSIS:**Knowledge deficit [Learning Need] regarding condition, prognosis, self care and treatment needs****May Be Related To:**

Lack of exposure/unfamiliarity with information resources, misinterpretation

Possibly Evidenced By:

Request for information, statement of misconceptions, inaccurate follow-through of instructions, development of preventable complications

DESIRED OUTCOMES/EVALUATION CRITERIA—CLIENT/COUPLE WILL:

Verbalize understanding of disease process and appropriate treatment plan.

Identify signs/symptoms requiring medical evaluation.

Perform necessary procedures correctly.

Initiate lifestyle/behavior changes as indicated.

ACTIONS/INTERVENTIONS

RATIONALE**Independent**

Assess client's/couple's knowledge of the disease process. Provide information about pathophysiology of PIH, implications for mother and fetus; and the rationale for interventions, procedures, and tests, as needed.

Establishes data base and provides information about areas in which learning is needed. Receiving information can promote understanding and reduce fear, helping to facilitate the treatment plan for the client. Note: Current research in progress may provide additional treatment options, such as using low-dose (60 mg/day) aspirin to reduce thromboxane generation by platelets, limiting the severity/incidence of PIH.

Provide information about signs/symptoms indicating worsening of condition, and instruct client when to notify healthcare provider.

Helps ensure that client seeks timely treatment and may prevent worsening of preeclamptic state or additional complications.

Keep client informed of health status, results of tests, and fetal well-being.

Fears and anxieties can be compounded when client/couple does not have adequate information about the state of the disease process or its impact on client and fetus.

Instruct client in how to monitor her own weight at home, and to notify healthcare provider if gain is in excess of 2 lb/wk, or 0.5 lb/day.

Gain of 3.3 lb or greater per month in second trimester or 1 lb or greater per week in third trimester is suggestive of PIH.

Assist family members in learning the procedure for home monitoring of BP, as indicated.

Encourages participation in treatment regimen, allows prompt intervention as needed, and may provide reassurance that efforts are beneficial.

Review techniques for stress management and diet restriction.

Reinforces importance of client's responsibility in treatment.

Provide information about ensuring adequate protein in diet for client with possible or mild preeclampsia.

Protein is necessary for intravascular and extravascular fluid regulation.

Review self-testing of urine for protein. Reinforce rationale for and implications of testing.

A test result of 2+ or greater is significant and needs to be reported to healthcare provider. Urine specimen contaminated by vaginal discharge or RBCs may produce positive test result for protein.