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Fluid Dynamics in Obstetrics: The Basics and Clinical implications

Paul Wendel, MD

University of Tennessee Health and Science Center - Memphis

Director, Division of Maternal / Fetal Medicine

MFM Fellowship Director

Objectives

1. Review the forces that influence the movement of fluid across a membrane
2. Examine the effects of pregnancy on these forces
3. Review clinical scenarios that demonstrate disease states in the pregnant patient
4. Outline key obstetric conditions that need attention in planning care

Foundational Concepts (Guyton Physiology)

Fluid dynamics across a semipermeable membrane

Key Forces:

Capillary Pressure (P_C)

- Moves fluid outward through the capillary membrane

Interstitial Fluid Pressure (P_{iF})

- Moves fluid inward through the capillary membrane
- Essentially a vacuum since it has a negative value

Plasma Colloid Pressure(COP_C)

- Pulls fluid into the capillary (osmosis)

Interstitial Fluid Oncotic Pressure (COP_{iF})

- Holds fluid in the intestinal space

Measurements of the Four Primary Forces

1. Mean Capillary Pressure (P_C): 17mmHg
2. Interstitial Fluid Pressure (P_{iF}): -6.3mm Hg (essentially a vacuum)
3. Plasma Oncotic (Colloid Osmotic) Pressure (COP): 28mm Hg
4. Interstitial Fluid Oncotic Pressure (COP_{iF}): 5 mm

Starling Principle

Near equilibrium exists between forces moving fluid in and out of capillaries

- Applies at both arterial and venous ends of capillaries

Mean Forces Moving Fluid Outward

Capillary pressure:
17mm Hg

Interstitial fluid pressure:
6.3 mm Hg

Interstitial oncotic
pressure:
5.0 mm Hg

Total outward force:
28.3 mm Hg

Mean Forces Moving Fluid Inward

Plasma oncotic pressure:
28.0 mm Hg

Net Effect:

Slight net outward
movement: 0.3 mm Hg

28.3 mm Hg Outward

-28.0 mm Hg Inward

0.3 mm Hg –Outward

Capillary Exchange (Starling Equilibrium)

- System is in **near equilibrium**
- Slightly more **filtration across the capillary membrane than reabsorption**
- Excess fluid in the intestines is returned to circulation via **lymphatic drainage**

Plasma Proteins and Oncotic Pressure

Osmotic pressure depends on the **number of dissolved molecules**

- **Normal Plasma Protein Distribution:**

- Albumin: ~4.5 g/dl
- Globulins (alpha, beta): ~2.5 g/dl
- Fibrinogen: ~0.3 g/dL

Key Points

- Albumin contributes about 70% of oncotic pressure
- Globulins and fibrinogen contribute **about 30%**
- Albumin exerts **approximately twice the oncotic effect** of globulins/fibrinogen

Pulmonary Fluid Balance & Edema

Clinical Examples of Derangement

- (Guyton Physiology Forces)
- Congestive Heart Failure
- Capillary hydrostatic pressure (P_c)
 - Fluid moves from capillaries interstitial space
- Mild increase in tissue hydrostatic pressure (P_i)
- Colloid osmotic pressure (**COP**) falls

Results:

- Net movement of fluid into the lungs
- Lead to **pulmonary edema**

Severe Proteinuria/Renal Disease

- Loss of proteins from plasma (excretion in urine)
 - Results in loss of Plasma colloid osmotic pressure (**COP**)
- Interstitial hydrostatic pressure (P_{iF}) slightly increased

Net Results:

- Movement of fluid from capillary to interstitium
- Causes **pulmonary edema**

Clinical Scenarios (continued)

Sepsis

- Capillary permeability increases
- Colloid osmotic pressure in vascular space falls
- Colloid osmotic pressure in interstitium increases thus pulling fluid out of vessels
- Interstitial hydrostatic pressure increases marginally

Results

- Fluid moves into lung parenchyma
- Can lead to **ARDS (Acute Respiratory Distress Syndrome)**

Pulmonary Lymphatic Obstruction

(Usually occurs with another underlying condition)

Normally:

- Small amounts of fluid/protein leak from capillaries
- Lymphatics remove this fluid (balance maintained)

When obstruction occurs:

- Interstitial hydrostatic pressure increases
- Collapse of lymphatic drainage
- Protein in interstitium build up leading to increased COP_{iF}

Results:

- Interstitial colloid osmotic pressure rises
- Draws fluid into lung tissue
- Leads to **pulmonary edema**

Normal Starling Forces in Pregnancy

- Moderate **decrease in plasma colloid osmotic pressure (COP_c)**
- Caused by **plasma volume expansion exceeding red cell/protein expansion**
- This leads to **reduced pulling force** holding fluid inside the vascular system
- There is also a **slight increase in capillary hydrostatic pressure (P_c)**
 - Leading to slight increase in fluid movement out

Overall Effects

These changes **increase net filtration:**

- Fluid moves from **vascular compartment to interstitial space**
- Under normal conditions this is balanced by increased lymphatic flow and return

When Balance is Disrupted

If lymphatic drainage is overwhelmed
(e.g., increased hydrostatic pressure or excess fluid)

Results:

- Fluid accumulates in tissues
- Leads to **edema** (commonly in lower extremities)

General Concepts

These changes tend to increase **net filtration** from the vascular compartment to the interstitium

- This net effect is balanced by **increased lymphatic flow and return**
- When lymphatic flow is overwhelmed, **edema develops**

Four Different Alterations of Guyton Fluid Dynamics Leading to Maternal Morbidity

- Tocolysis in preterm labor
- Antepartum management of chronic hypertension (CHTN)
 - Rule out preeclampsia
- Preeclampsia
- Postpartum complications (ex: hypertension in twins)

Tocolysis

22-year-old white female, G2P0, at 29 weeks with **threatened preterm contractions (PTC)**

- Treated initially with hydration
- Then given **3 doses SQ terbutaline (night shift) and 3 doses (day shift)**
- **28 hours after presentation, develops:**
 - O2 saturation: 87%
 - Respiratory rate: 32

Issues

- **Hydration: plasma colloid osmotic pressure (COP) dilution**
- **Terbutaline: slight in plasma hydrostatic pressure**
- **Repeated doses: ADH like effect on kidneys- fluid retention**
- **Decreased plasma COP: increased hydrostatic pressure:**
- **Fluid shift into pulmonary interstitial**
- **Pulmonary edema**

Chronic Hypertension/Possible Preeclampsia

40 year old, G6P4, at 32 weeks, chronic hypertension (CHTN) admitted with elevated BP

- Started on labetalol 200 mg BID, increased over 2 days to 400 mg TID
- 24-hour urine protein: ~1.1g
- Labs:
 - Hematocrit ~40%
 - Platelets: ~122k
 - Creatinine: 1.1
 - RR: 28
 - O₂ Saturation 92%

Pathophysiology

- **Rising BP:** capillary hydrostatic pressure
- **Proteinuria:** leads to loss of plasma proteins and fall in COP_C
- **Falling platelets:** endothelial damage (suggestive preeclampsia)
- **Interstitial COP increases:**
 - 2+ fluid shifts out of vasculature
- **Leads to:**
 - Hemoconcentration (hematocrit, elevated creatinine)
 - Fluid accumulation in interstitium

Outcome

Net fluid movement into interstitium

- Pulmonary edema
- Oliguria
- Fetal compromise

Summary

- Fluid balance depends on **hydrostatic pressure, oncotic pressure, and lymphatic drainage**
- Disruption (e.g., meds, hypertension, protein loss) **fluid shifts into tissues**
- When compensatory lymphatic drainage fails - **edema, especially pulmonary edema**

Pyelonephritis

Initial findings:

- Temp: 38.8°C
- Patient had rigors
- BP: 86/46
- Pulse: 128/min
- RR: 24/min
- WBC: 24,000
- Hct: 23%

Given:

- 2 Liters LR (Lactated Ringer's)
 - To fluid resuscitate and correct hypotension

Pyelonephritis (continued)

After fluids:

- BP 96/58
- Pulse: 108/min
- RR: 22/min
- Urine output: 35-40 cc/hr.
- **Transferred to ward**

After first antibiotic:

- Called to room for SOB
- RR: 32/min
- O₂ saturation 83%

After 24 hrs of IV antibiotics

Guyton Derangement

- Anemia (Hct 23%): blood transfusion may raise plasma colloid osmotic pressure (COP)
 - Mild decrease in plasma COP due to ↓ globulins
 - COP_p ↓ falls
- Fluid hydration for hypotension causes dilution of proteins (COP_p)
- Double effect of loss of plasma oncotic pressure
- With bacteria cell lysis secondary to IV antibiotics:
 - Endotoxin release
 - Endothelial damage
 - Protein plasma leakage into interstitium

Platelets: 244k

Guyton Derangement (continued)

Net Effects

- Loss of plasma COP
- Net movement of fluid to interstitium
- Endothelial damage results in easier and greater movement of fluid into interstitium
- Especially affects lungs
- Leads to pulmonary edema

Treatment

- Moved to ICU
- Intubated with PEEP
- Increase interstitial hydrostatic pressure
- Blood transfusion to increase plasma colloid osmotic pressure and pull fluid out of lungs
- Lasix to offload excess intravascular fluid and increase COP, helping pull fluid out of lungs

Postpartum Hypertension in Patient with Twins

41-year-old African American female, G3P delivered twins via C-section after 18 hours of failed induction

- **Twin intrauterine pregnancy:**

- Twin A vertex at 36 weeks
- Mild chronic hypertension superimposed on severe preeclampsia

Findings

- Increased blood pressure
- Increased proteinuria
- Estimated blood loss:
 - 1500cc

Postpartum Hypertension Findings (continued)

Postpartum:

- Hct 23%
- Urine output > 30-40 cc/hr
- Platelets: 151k
- Creatinine: 0.9
- AST/ALT: WNL

Blood pressure range:

- 150-160 / 95-104

Discharge home on POD #4

- BP: approximately 150/96-100

Noted:

- Postpartum report normal
- Significant edema of
 - Hands
 - Face
 - Lower extremities

Postpartum Hypertension Findings (continued)

Post-op Day 5:

Called office with headache

- Worsening edema
- Rapid pulse
- Increased respiratory rate

Triage:

- BP: 168/105
- Pulse: 110/min
- RR: 32/min
- Pulse O₂: 91%

Laboratory findings:

- Hct: 22%
- Platelets: 120k
- Urinalysis: 3+ protein

Chest X-ray:

- Pulmonary edema

Ejection fraction:

- 55-60%

AST/ALT:

- Normal

Colloid/Starling Derangements

- Plasma hydrostatic pressure 
 - Due to uncontrolled hypertension
- Plasma oncotic pressure 
 - Loss of proteins due to endothelial damage from preeclampsia
 - Loss of protein due to proteinuria
 - Low Hct further decreases plasma COP
- Fluid overload
 - From 18-hour induction
 - C-section for failed induction
- Initial colloid oncotic pressure in plasma decreased
 - Effect of twins

Colloid/Starling Derangements

Treatment:

- Admit
- Rule out cardiac pathology
- Lasix:
 - Decreased plasma hydrostatic pressure
 - Increase effective COP
- Blood pressure control
- Blood transfusion to increase plasma colloid osmotic pressure

