

Placental Abnormalities

Structural, vascular, inflammatory and implantation-related mechanisms of fetal compromise

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Learning outcomes

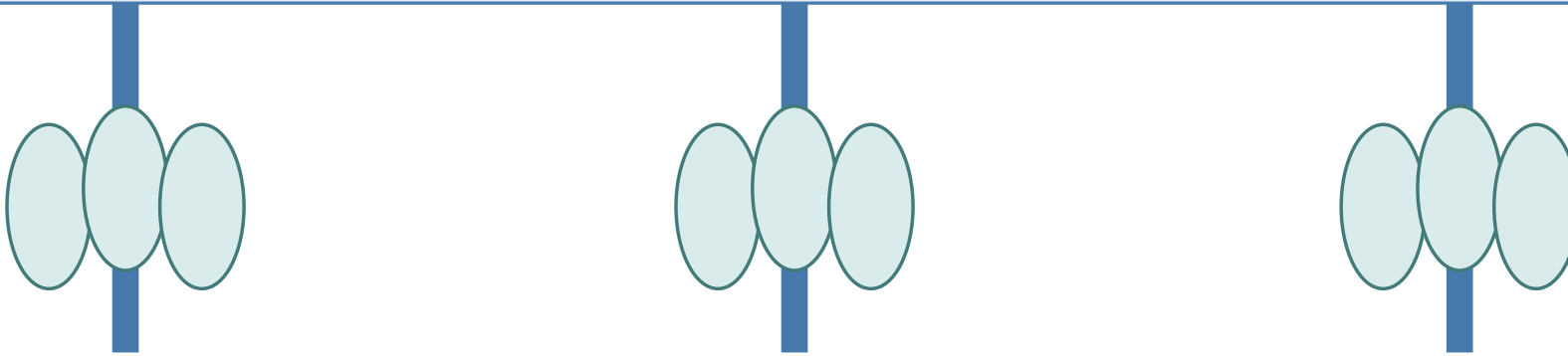
- Explain how placental function determines fetal growth in late gestation.
- Distinguish maternal from fetal vascular malperfusion and their morphology.
- Relate implantation, cord and infectious lesions to pregnancy outcomes.
- Interpret selected placental patterns in fetal growth restriction.

The placenta is a critical growth organ

- Third-trimester fetal growth places major demands on uteroplacental flow.
- Placental growth during the mid-trimester determines subsequent exchange capacity.
- Reduced delivery of nutrients or oxygen may constrain fetal growth.

Functional placental architecture

Chorionic plate — fetal vascular branches



Chorionic villi with fetal vessels

Maternal blood surrounds villi in intervillous spaces

Decidua basalis — maternal vascular interface

Maternal and fetal circulations are distinct

Maternal compartment	Fetal compartment
Maternal arteries supply blood to intervillous spaces	Umbilical vessels connect fetus to vessels in chorionic villi
Intervillous blood surrounds villous surfaces	Fetal blood remains within fetal vascular channels
Defective spiral arteries compromise maternal inflow	Cord obstruction compromises fetal-side perfusion

Exchange requires two functioning pathways



Sequential mechanism (schematic)

Fetal growth restriction is not synonymous with prematurity

- Premature infants may be small yet appropriate for gestational age.
- Fetal growth restriction (FGR) denotes failure to achieve expected fetal growth.
- Small-for-gestational-age (SGA) is a size description; Etiologic assessment remains necessary.

Three broad origins of growth restriction

Maternal

Impaired maternal placental flow; Vascular disease and other maternal exposures.

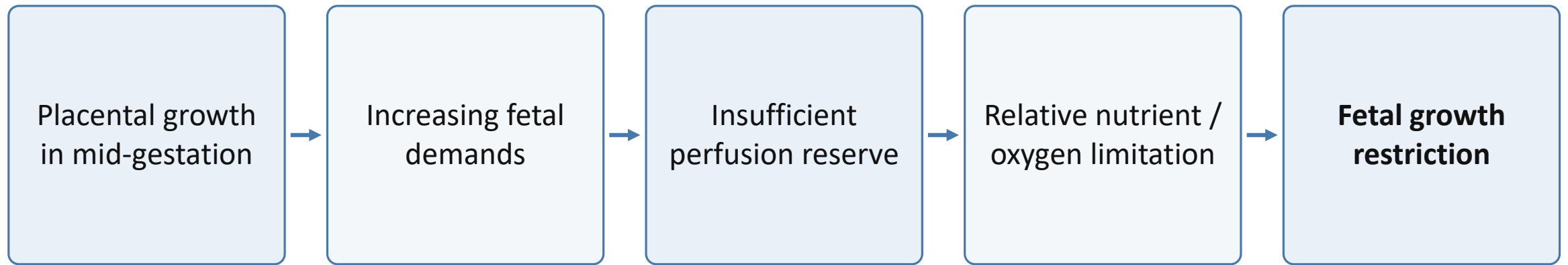
Fetal

Intrinsic restriction of growth potential; Chromosome anomalies and infection.

Placental

Inadequate placental development, perfusion or exchange.

Why timing matters



Sequential mechanism (schematic)

Placental insufficiency is a functional concept

- An inadequate placenta cannot consistently meet fetal metabolic demands.
- Perfusion abnormalities and primary villous disease can both limit exchange.
- Placental size alone does not identify every underlying mechanism.

Placental etiologies named

- Umbilical–placental vascular anomalies, thrombosis and infarction.
- Placental abruption or placenta previa.
- Chronic villitis, placental infection and multiple gestations.
- Some small placentas have no identifiable cause.

Placental FGR: Central physiologic sequence



Sequential mechanism (schematic)

Patterns of growth restriction

Predominantly placental	Predominantly fetal
Usually asymmetric (disproportionate)	Usually symmetric (proportionate)
Relative preservation of brain growth	Many organ systems similarly restricted
Reflects supply limitation in later pregnancy	Reflects intrinsically reduced fetal growth potential

Brain sparing: Interpretation of the pattern

- Asymmetric FGR implies unequal effects on organ growth.
- Relative sparing of the brain in placental FGR.
- The morphologic pattern supports—but does not prove—a placental cause.

From a growth pattern to a working differential

Observed pattern	Etiologic direction
Asymmetric small fetus	Consider limited placental nutrient / oxygen supply
Proportionately small fetus	Consider intrinsic fetal causes or congenital infection
Mixed or indeterminate pattern	Assess maternal, fetal and placental factors together

Maternal vascular disease can affect the placenta

- Chronic hypertension and preeclampsia may reduce uteroplacental flow.
- The fetus may be intrinsically normal yet receive inadequate placental supply.
- Maternal vascular insufficiency and placental lesions often coexist.

Intrinsic fetal disorders can mimic placental FGR

- Chromosomal disorders and structural anomalies can limit fetal growth.
- Congenital infections may restrict growth through fetal mechanisms.
- Symmetric growth restriction prompts evaluation beyond perfusion lesions.

Anatomic sites of placental dysfunction

Maternal inflow

Spiral artery remodeling;
Decidual vasculopathy;
Abruptio.

Exchange tissue

Placental hypoplasia;
Infarction; Inflammatory
villous injury.

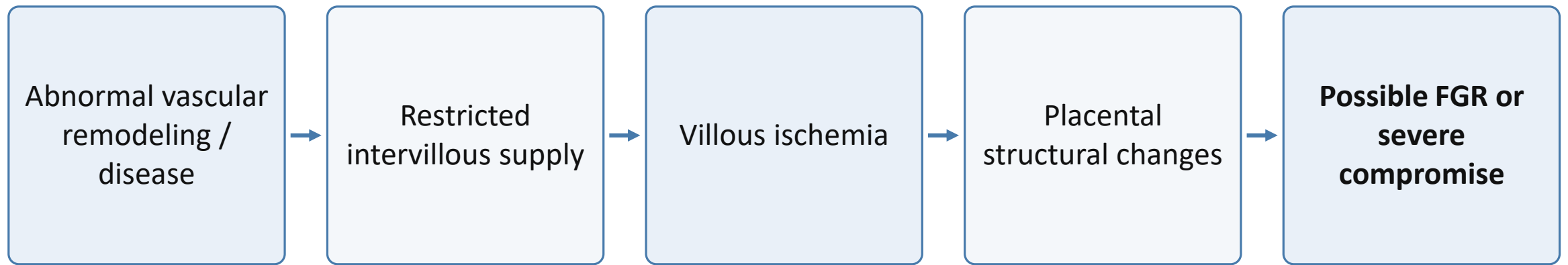
Fetal outflow

Cord compromise; Fetal
vascular thrombi;
Avascular villi.

Maternal vascular malperfusion: Definition

- Maternal vascular malperfusion (MVM) signifies inadequate maternal blood supply to intervillous spaces.
- Abnormal implantation, placental development or maternal vascular disease may initiate it.
- Effects extend from growth restriction to severe placental ischemia.

Maternal malperfusion causal map



Sequential mechanism (schematic)

Placental hypoplasia: One MVM feature

- Robbins specifies placental weight below the tenth percentile for gestational age.
- Reduced placental mass may reduce the available exchange surface.
- Hypoplasia is one element of the MVM pattern, not its sole feature.

Placental infarcts

- Infarcts are areas of ischemic placental injury.
- Large or numerous infarcts are described in maternal malperfusion.
- Functional significance depends on the cumulative loss of viable exchange tissue.

Villous consequences of ischemia

Small villi

Distal villi may appear shrunken.

Syncytial knots

Ischemic villous changes include increased syncytial knotting.

Expanded intervillous space

Distal villous hypoplasia includes relatively increased maternal vascular space.

Distal villous hypoplasia

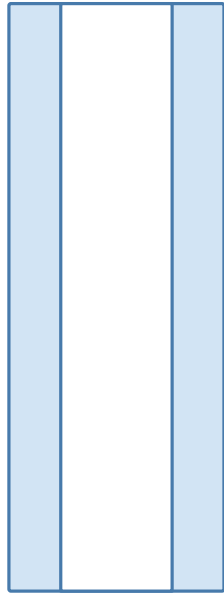
- The distal villi are abnormally small in affected regions.
- The proportion of surrounding maternal vascular space increases.
- The finding belongs to a constellation of ischemic villous alterations.

Syncytial knots as an ischemic sign

- Increased syncytial knotting is described in placental MVM.
- Interpret knotting alongside placental age and other structural findings.
- It is not a standalone proof of a specific maternal condition.

Normal versus poorly remodeled spiral arteries

Physiologic remodeling



Large-caliber
Low-resistance vessel

Incomplete remodeling



Persistently narrow
Higher-resistance vessel

Conceptual vessels; Not a histologic image

Failure of spiral artery remodeling

- Normal extravillous trophoblast transforms spiral arteries into low-resistance vessels.
- Incomplete remodeling retains features of high-resistance maternal vessels.
- The resulting placenta is less able to accommodate late-gestational demand.

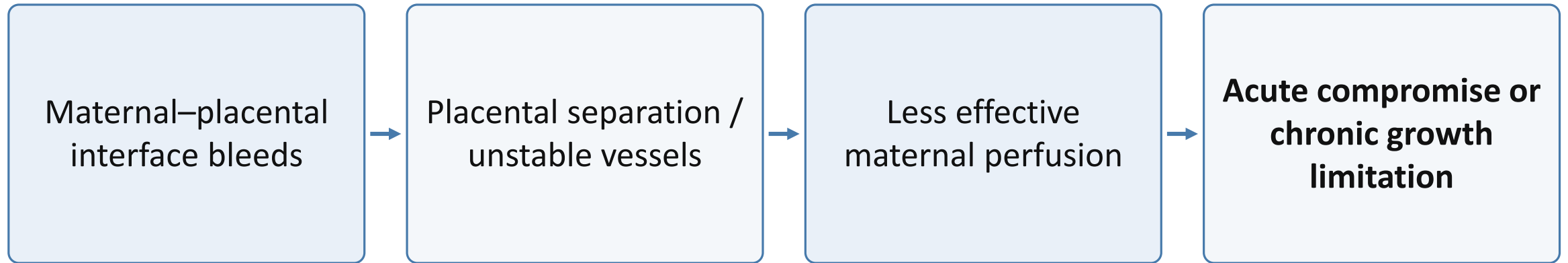
Decidual arteriopathy in maternal malperfusion

- Affected vessels may lack expected physiologic remodeling.
- Other possible findings include thrombi, fibrinoid necrosis and acute atherosclerosis.
- These changes support maternal vascular disease in the appropriate context.

Placental abruption

- Abruption represents premature separation of the placenta from the uterine wall.
- Retroplacental bleeding can destabilize uteroplacental perfusion.
- Abruption among placental causes of growth restriction and prematurity.

Abruptio: Why fetal injury can follow



Sequential mechanism (schematic)

Maternal vascular malperfusion: Integrated lesion set

Gross or macroscopic clues	Microscopic clues
Small placenta for gestation	Distal villous hypoplasia
Infarcts; possibly large or numerous	Increased syncytial knots
Retroplacental hematoma	Abnormal decidual arteries; thrombi / atherosclerosis

Case: Small placenta and infarcts

CLINICAL SCENARIO

A fetus shows late-gestation growth restriction. The placenta is small and contains multiple infarcts.

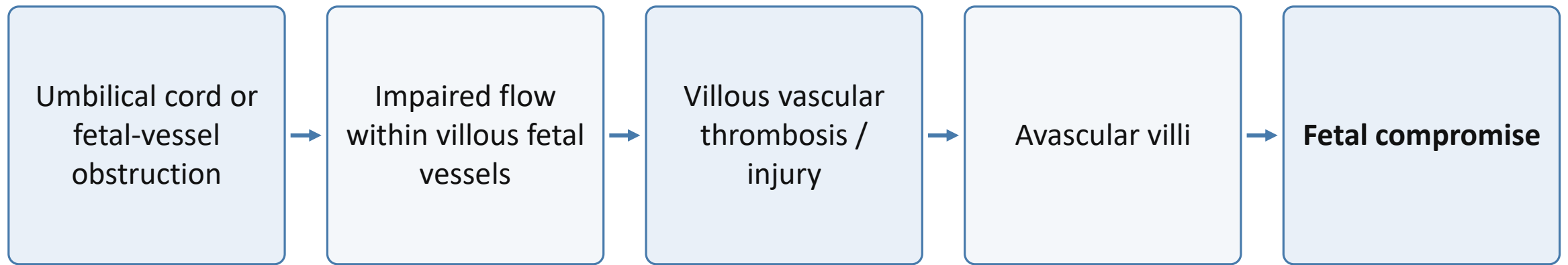
Which primary functional compartment is likely compromised?

Interpretation: Maternal uteroplacental perfusion; Interpret as a pattern of maternal vascular malperfusion.

Fetal vascular malperfusion: Concept

- Fetal vascular malperfusion (FVM) involves diminished perfusion of placental villi from the fetal side.
- Cord obstruction and fetal-side vascular lesions are important upstream abnormalities.
- Thrombi and avascular villi provide morphologic clues.

Fetal malperfusion causal map



Sequential mechanism (schematic)

Umbilical cord obstruction

- Tight knots, kinks, nuchal or body cords and prolapse may interrupt flow.
- Marked acute obstruction can be fatal yet leave few placental morphologic clues.
- Chronic or intermittent obstruction can produce fetal vascular malperfusion.

Why a true knot matters

- Constriction of umbilical vessels reduces fetal-side blood delivery.
- The downstream placenta may develop avascular villi and vascular thrombi.
- A gross cord lesion and a downstream villous lesion support a causal interpretation.

Avascular villi

- Chorionic villi normally contain fetal capillaries.
- Loss or absence of vessels within involved villi characterizes avascular villi.
- The finding is illustrated in the setting of fetal vascular malperfusion.

Villous vascular thrombi

- Thrombosis in fetal placental vessels may interrupt flow to dependent villi.
- Thrombi and endothelial damage are described in second-trimester losses.
- Evaluate thrombi together with cord abnormalities and villous changes.

Maternal versus fetal malperfusion

Maternal vascular malperfusion	Fetal vascular malperfusion
Maternal blood reaching intervillous space is limited	Fetal blood flowing into villous vessels is limited
Placental infarcts; decidual arteriopathy	Avascular villi; fetal vascular thrombi
Small villi and syncytial knots may occur	Cord lesions or compression may be found

Single umbilical artery as a placental vascular anomaly

- A single umbilical artery among potential placental causes of FGR.
- The cord must be assessed together with placental perfusion and fetal findings.
- A vascular anomaly does not alone establish the cause of restricted growth.

Abnormal cord insertion

- Abnormal umbilical insertion is included among placental contributors to FGR.
- It identifies a structural abnormality of the placental vascular connection.
- Interpret its clinical significance in the context of the complete placenta.

Case: Knot and avascular villi

CLINICAL SCENARIO

A tight, complex true umbilical-cord knot is accompanied by extensive avascular villi.

What is the mechanistic diagnosis suggested by these findings?

Interpretation: Umbilical vascular obstruction with downstream fetal vascular malperfusion.

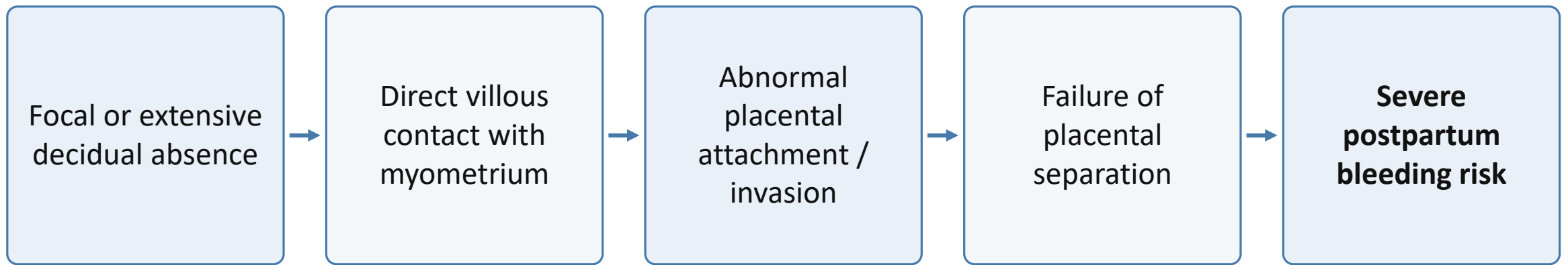
Placenta previa: Low implantation

- The placenta implants in the lower uterine segment and covers the cervix partially or completely.
- Clinically significant bleeding may occur in the third trimester.
- Previa among placental abnormalities contributing to poor pregnancy outcomes.

Placenta accreta spectrum

- Loss of decidua allows chorionic villous tissue to contact or invade myometrium.
- Depth and extent of abnormal attachment vary within the spectrum.
- Failure of normal placental separation creates a risk of major postpartum hemorrhage.

How placenta accreta spectrum develops



Sequential mechanism (schematic)

Previa, accreta and abruption are not interchangeable

Placenta previa	Placenta accreta spectrum	Placental abruption
Low implantation over cervix	Abnormally adherent / invasive placenta	Premature placental separation
Third-trimester bleeding risk	Retained placenta / postpartum bleeding	Retroplacental bleeding; impaired perfusion
Location-related abnormality	Implantation-interface abnormality	Separation-related abnormality

Multiple gestations increase placental vulnerability

- Twins may differ in growth, malformations and placental pathology.
- Monochorionic twins share a placenta and vascular connections.
- Multiple gestations appear among placental settings associated with FGR.

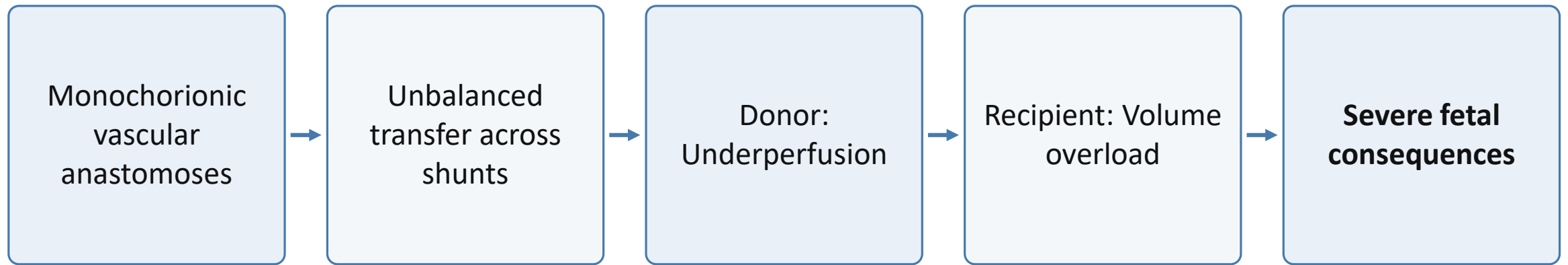
Twin placentation: Chorion and amnion

Placentation	Anatomic feature
Dichorionic diamniotic	Separate chorions and amniotic sacs; placentas may be fused
Monochorionic diamniotic	Single chorion; two amniotic sacs
Monochorionic monoamniotic	Single chorion and one shared amniotic cavity

Chorionicity determines vascular sharing

- Monochorionic placentas imply monozygotic twins.
- Shared placental vasculature allows anastomoses between fetal circulations.
- Dichorionic placentation can occur in monozygotic or dizygotic pregnancy.

Twin-to-twin transfusion: Vascular imbalance



Sequential mechanism (schematic)

Twin-to-twin transfusion syndrome

Donor twin	Recipient twin
Relatively underperfused	Receives excess transferred blood flow
Disadvantaged by unbalanced shunt	Subject to fluid overload
May experience severe compromise	May experience severe compromise

Case: Discordant monochorionic twins

CLINICAL SCENARIO

Monochorionic twins are substantially discordant; One appears underperfused and the other fluid overloaded.

What placental connection explains this pattern?

Interpretation: Intertwin vascular anastomoses causing unbalanced twin-to-twin transfusion.

Two principal routes of placental infection

- Ascending microorganisms enter via the birth canal and involve the membranes.
- Hematogenous maternal spread can seed villous tissue through the circulation.
- Route of spread predicts the dominant tissue compartment affected.

Ascending infection sequence



Sequential mechanism (schematic)

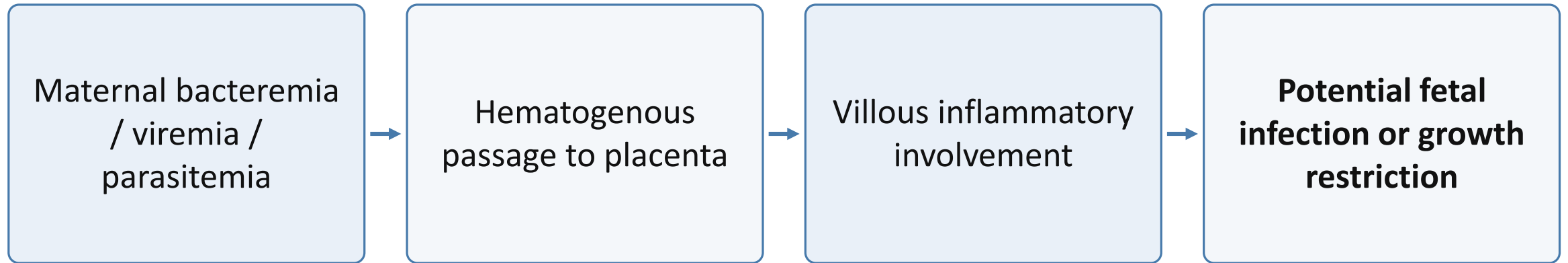
Acute chorioamnionitis: Membrane pathology

- Ascending infection is most often bacterial.
- Maternal neutrophils infiltrate chorion–amnion membranes.
- Inflammation can accompany premature membrane rupture and preterm birth.

Fetal inflammatory response

- Inflammation may extend to umbilical vessels or fetal chorionic plate vessels.
- This vascular involvement as a fetal inflammatory response.
- Intense responses correlate with perinatal morbidity and increased cerebral palsy risk.

Hematogenous placental infection



Sequential mechanism (schematic)

Acute versus chronic villous inflammation

Acute villitis	Chronic villitis
May arise in bacterial hematogenous infection	Chronic inflammatory infiltrate of the villi
Robbins documents acute infectious villitis	TORCH-associated placental involvement may be chronic
Assess accompanying necrosis / organisms	Relate to infection and FGR where supported

TORCH infections and the villous compartment

- Hematogenous spread permits several TORCH pathogens to infect the placenta.
- These infections with chronic villitis and fetal growth restriction.
- Specific pathogens require independent microbiologic / clinical correlation.

Chronic villitis of unknown etiology

- Robbins separately names chronic villitis of unknown etiology as a placental contributor to FGR.
- Unknown etiology should not be labeled as a confirmed infectious process.
- Morphology and clinical context are necessary before assigning a cause.

Recognize infection by involved compartment

Predominant site	Most pertinent pathway
Chorion–amnion membranes with neutrophils	Usually ascending infection
Umbilical / chorionic-plate fetal vessels	Fetal inflammatory response to an ascending process
Chronically inflamed chorionic villi	Consider hematogenous infection or other villitis

Case: Membrane neutrophils and funisitis

CLINICAL SCENARIO

Membranes contain a neutrophil-rich infiltrate; Fetal umbilical vessels are also inflamed.

What two inflammatory components are present?

Interpretation: Maternal acute chorioamnionitis plus a fetal inflammatory vascular response.

Placental lesions and possible fetal outcome

Placental abnormality	Functional link
Placental hypoplasia, infarction	Reduced exchange capacity / maternal supply
Cord obstruction or vascular thrombosis	Reduced fetal-side placental perfusion
Chronic villitis or infection	Inflammatory injury with possible growth restriction
Monochorionic vascular shunts	Unequal distribution of circulation between twins

Placental lesions can act at different gestational stages

- Earlier severe placental disease may produce fetal loss when reserve is limited.
- The third trimester amplifies the consequences of inadequate placental perfusion.
- An acute cord event may leave fewer histologic clues than prolonged compromise.

Why a small infant needs etiologic interpretation

- Size alone cannot distinguish maternal, fetal or placental growth restriction.
- Symmetry of growth and associated malformations help direct the differential.
- Placental gross and microscopic abnormalities add independent mechanistic information.

Placental FGR: Beyond the perinatal period

- Morbidity extending into childhood and adult life.
- Possible consequences include cerebral dysfunction and learning disability.
- Hearing and visual impairment are also among the documented risks.

Clinical implications depend on the mechanism

- Placental growth impairment primarily concerns chronic nutrient / oxygen limitation.
- Cord interruption may produce severe acute fetal compromise.
- Ascending infection relates to preterm delivery and inflammatory complications.
- Abnormal placental implantation can threaten maternal hemostasis.

Approach to a suspected placental cause of FGR

Question	Look for / interpret
Is maternal inflow compromised?	Hypoplasia, infarcts, villous ischemia, decidual arteriopathy
Is fetal-side flow obstructed?	Cord lesion, fetal thrombi, avascular villi
Is there inflammatory injury?	Membrane neutrophils, fetal vascular response, villitis
Is placentation abnormal?	Previa, accreta, chorionicity and vascular shunts

Case: Disproportionate fetal growth restriction

CLINICAL SCENARIO

A fetus has disproportionate late-gestation growth restriction with relative preservation of head growth.

Which etiologic category is most characteristic of this pattern?

Interpretation: Placental insufficiency is favored, although the growth pattern is not pathognomonic.

Key clinicopathologic distinctions

- Maternal malperfusion: Impaired inflow, villous ischemia and placental infarction.
- Fetal malperfusion: Fetal vascular obstruction, thrombi and avascular villi.
- Infection: Identify membrane, fetal vascular or villous involvement.
- Implantation and monochorionic disorders have distinct hemorrhagic or shunting risks.

Final synthesis: Anatomy explains the pathology

- Adequate maternal inflow, exchange villi and fetal vascular flow are all essential.
- Structural and inflammatory lesions can reduce effective placental performance.
- Placental FGR is often asymmetric, with relative brain sparing.
- Interpret placental findings with fetal growth, maternal disease and gestational timing.

Primary textbook reference

- Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th edition. 2026.
- Relevant sections: Prematurity and Fetal Growth Restriction; Placental anatomy and disorders.
- All diagrams are original conceptual PowerPoint schematics. No histopathology images are represented as diagnostic specimens.