

Maternal Vascular Malperfusion

Placental circulation, mechanisms, morphology and clinical correlation

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Learning objectives

By the end of the lecture, you should be able to:

- Distinguish maternal intervillous perfusion from fetal villous circulation.
- Explain how abnormal spiral-artery remodeling can compromise placental perfusion.
- Recognize the defining constellation of MVM-associated placental lesions.
- Relate placental findings to fetal growth restriction and preeclampsia.
- Differentiate MVM from fetal vascular malperfusion and selected mimics.

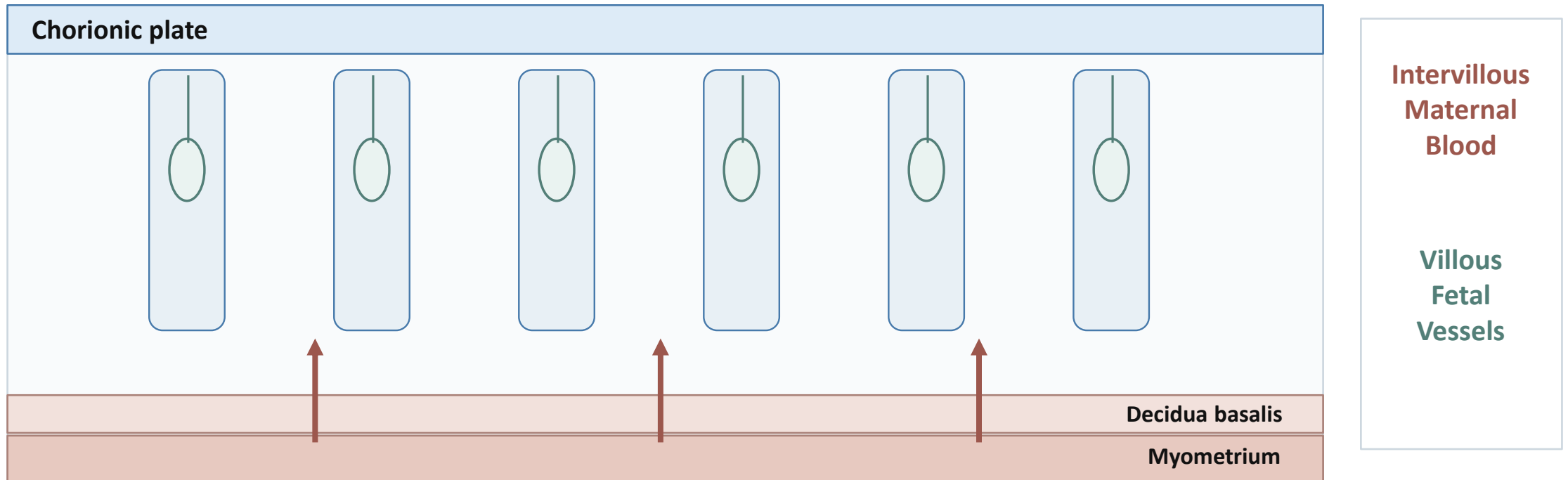
Maternal vascular malperfusion: Definition

MVM is a disorder of maternal blood delivery to the placenta.

- Abnormal maternal placental circulation reduces supply to intervillous spaces.
- The precipitating problem may be abnormal implantation, development, or maternal vascular disease.
- MVM is identified by a constellation of placental findings—not a single obligatory lesion.

The anatomic compartment affected is the maternal side of exchange.

The maternal circulation supplies intervillous blood



Red arrows: Maternal inflow from spiral arteries

Schematic—not a specimen

Two placental circulations must be distinguished

Maternal compartment

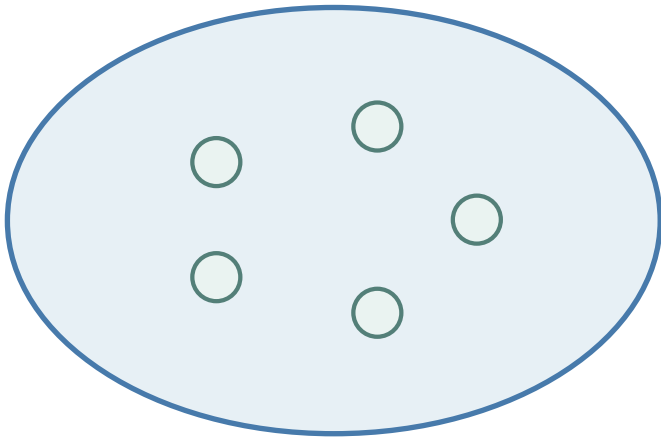
- Blood reaches intervillous space from maternal spiral arteries.
- Maternal perfusion supports transfer across villous trophoblast.
- MVM concerns this incoming maternal blood supply.

Fetal compartment

- Fetal vessels travel via umbilical vessels and villous capillaries.
- Fetal-side obstruction may cause vascular thrombi or avascular villi.
- Fetal vascular malperfusion is a different morphologic process.

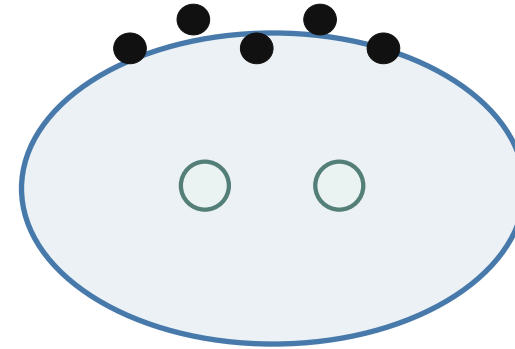
The chorionic villus is the exchange structure

Ordinary terminal villus



Fetal capillaries

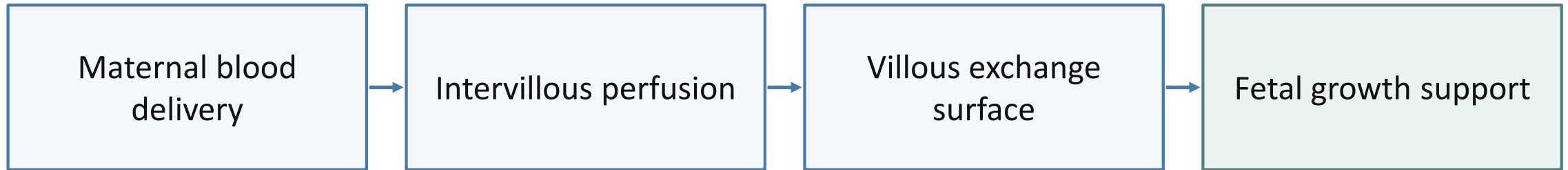
Ischemic distal villus



Smaller villus + syncytial knots

The drawing explains features—not diagnostic thresholds or specimen appearance.

Why adequate maternal inflow matters



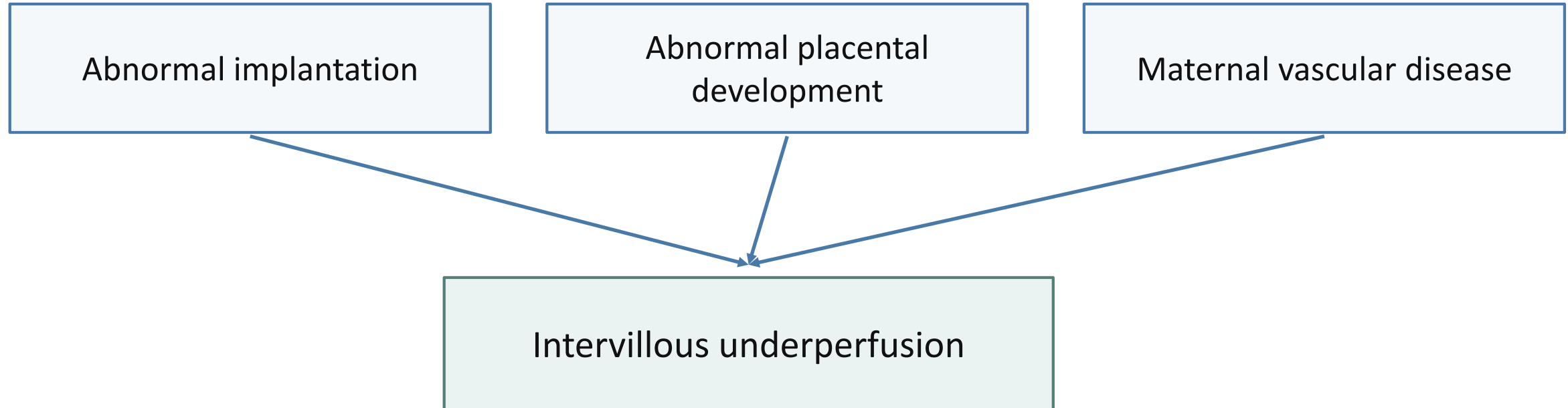
Reduced inflow can compromise the placenta's capacity to sustain fetal growth.

Placental demand increases with gestation

The context for uteroplacental underperfusion:

- Fetal nutritional demand increases rapidly in the second trimester.
- A developmentally immature placenta has less reserve for exchange.
- Severe perfusion abnormalities may therefore manifest before the third trimester.

Causes converge on inadequate intervillous supply



Each category can precipitate MVM; They are not necessarily successive steps.

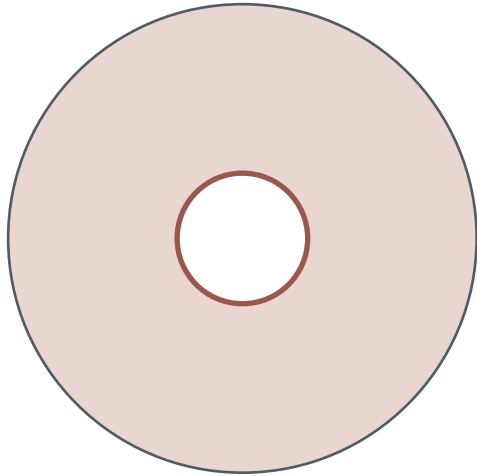
Maternal vascular disease as a precipitant

Maternal conditions can compromise uteroplacental blood delivery.

- Maternal vascular disease can precipitate reduced placental blood supply.
- Hypertension and coagulopathies appear in related discussions of pregnancy complications.
- The placenta's morphology records effects of impaired supply; It does not by itself identify a unique maternal cause.

Physiologic spiral-artery remodeling changes resistance

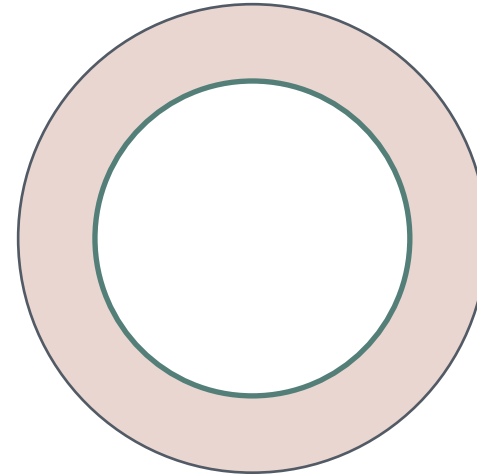
Unremodeled artery



Narrow lumen

Retained muscular wall and higher resistance

Remodeled artery



Wide lumen

Trophoblast-mediated smooth-muscle loss

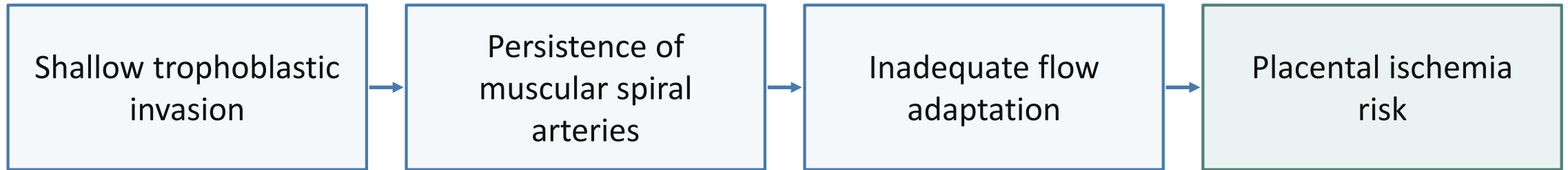
Cross-sectional schematic — not histology

Normal remodeling is trophoblast mediated

Placental-bed vascular transformation is active, not passive.

- Extravillous trophoblast invades maternal decidua and decidual vessels.
- Vascular smooth muscle is destroyed and maternal endothelial lining is partly replaced.
- The result is a large-capacity, low-resistance uteroplacental vascular bed.

Failed remodeling leaves high-resistance supply



Failed remodeling is a central placental abnormality in preeclampsia.

Remodeled and unremodeled placental-bed arteries

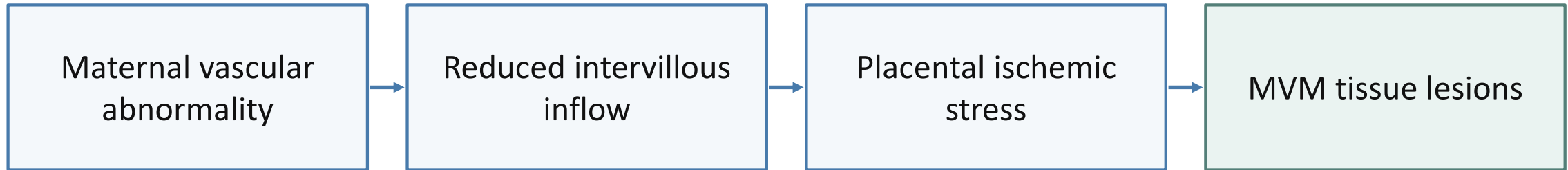
Physiologic remodeling

- Large-capacity arterial channels
- Smooth-muscle coat largely lost
- Low-resistance uteroplacental pathway
- Adapts to later gestational demand

Failure of remodeling

- Smaller, muscular resistance vessels persist
- Reduced perfusion adaptation
- Greater susceptibility to placental ischemia
- Can accompany preeclampsia-associated MVM

Pathogenesis connects maternal vessels to villi



The morphologic pattern follows impaired supply to the exchange compartment.

Severity is a spectrum

Reduced perfusion has variable clinicopathologic expression.

- At the milder end: Restricted fetal growth.
- At the severe end: Uteroplacental ischemia and association with maternal preeclampsia.
- Severity cannot be inferred from one lesion without considering the complete placenta and clinical context.

A five-part morphologic constellation

Placental
hypoplasia

Placental infarcts

Decidual arterial
lesions

Ischemic villous
changes

Abruption /
retroplacental
bleed

These findings can coexist in different combinations; None is required in every specimen.

Placental hypoplasia has a size definition

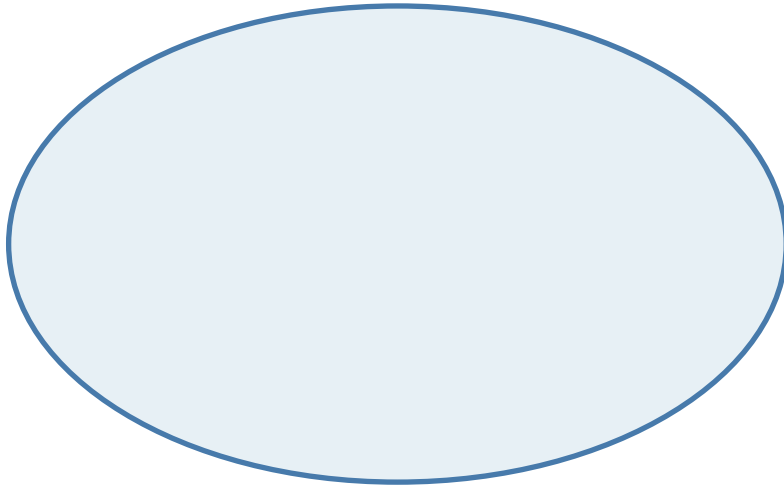
Hypoplasia against gestational age.

- Placental weight is below the 10th percentile for gestational age.
- Interpret weight relative to age, not as an isolated absolute number.
- Reduced placental mass is one component of the MVM pattern.

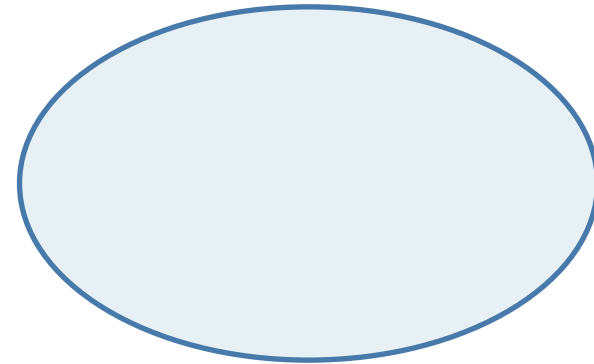
The book provides the percentile; This deck does not invent a gram cutoff.

Placental hypoplasia changes the whole-organ assessment

Gestational-age comparison—not an absolute gram cutoff



Reference placenta



Placental hypoplasia

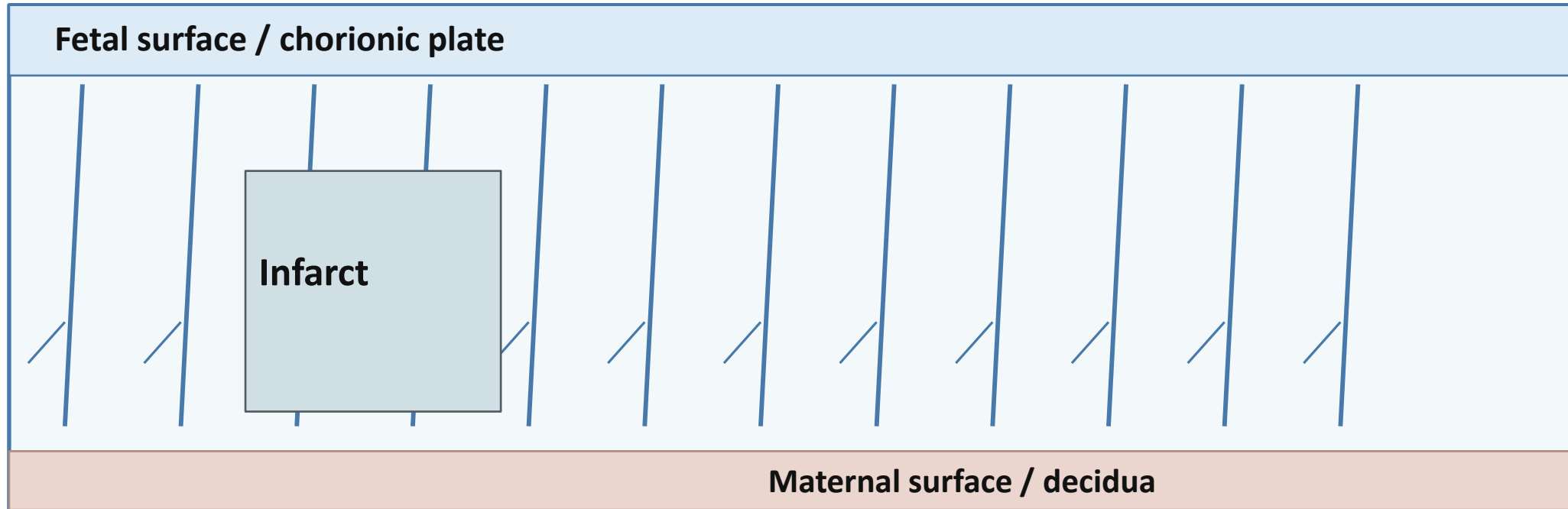
Weight below 10th percentile for
gestational age

A small placenta is not a cause-specific diagnosis

The weight criterion must be interpreted in context.

- Weight alone is not sufficient to specify abnormal spiral-artery remodeling.
- Examine infarcts, villous ischemia, decidual vessels, and retroplacental lesions together.
- Correlate with fetal growth and maternal obstetric findings.

Placental infarction localizes ischemic injury



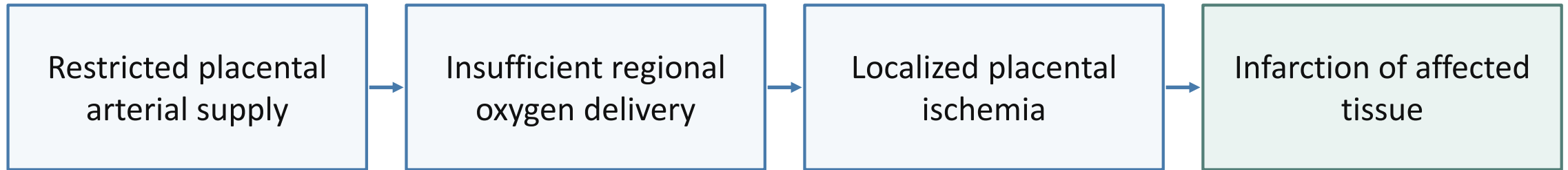
Compartment schematic — not gross pathology

Placental infarcts are an MVM-associated lesion

Infarction reflects tissue injury in the setting of impaired perfusion.

- Placental infarction is a principal component of the MVM pattern.
- In preeclampsia-associated MVM, infarcts may be large and numerous.
- Interpret extent alongside the other placental lesions and pregnancy history.

Why infarction follows maternal underperfusion



A causal schema derived' placement of infarcts among ischemic MVM lesions; The diagram is conceptual.

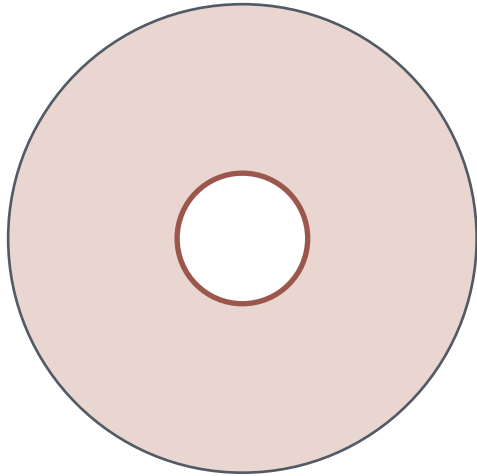
Decidual vessels provide mechanistic evidence

Placental-bed vessel pathology complements villous findings.

- Failure of normal spiral-artery remodeling can be visible in decidual vasculature.
- Decidual arterial thrombi may accompany MVM.
- Fibrinoid necrosis and intimal lipid deposition are additional described changes.

Decidual arterial lesions must be separated from villous changes

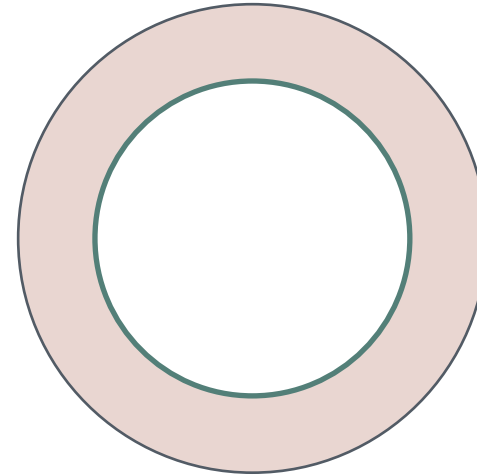
Unremodeled artery



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Trophoblast-mediated smooth-muscle loss

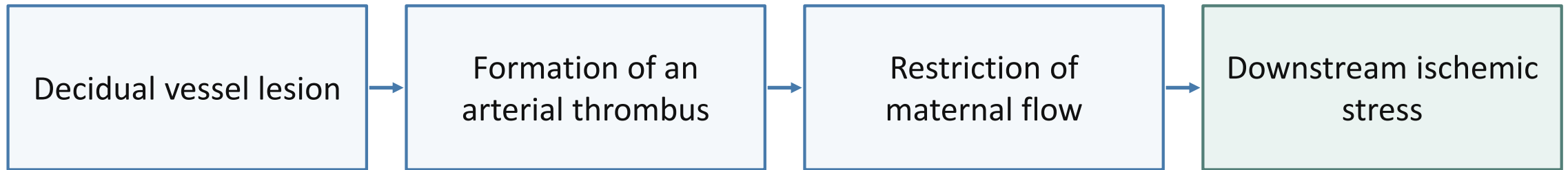
Cross-sectional schematic — not histology

Unremodeled spiral arteries retain a muscular phenotype

Mechanism and morphology are linked.

- Normal adaptation removes smooth muscle and lowers resistance.
- Failed remodeling leaves vessels less suited to high gestational perfusion demands.
- An abnormal decidual vessel is not itself the same thing as a villous infarct.

Decidual thrombi: Obstruction of a maternal pathway



Decidual-vessel thrombi may occur within the MVM morphologic pattern.

Fibrinoid necrosis affects decidual vessels

The abnormality is in a maternal vessel wall.

- Fibrinoid necrosis is explicitly listed among decidual-vessel changes.
- It belongs to the vascular pathology component of the MVM constellation.
- Do not confuse this lesion with villous vascular thrombosis on the fetal side.

Acute atherosclerosis is intimal lipid deposition

A specific form of decidual arterial pathology.

- Intimal lipid deposition in decidual vessels is termed “acute atherosclerosis.”
- It is listed among abnormal decidual vessels in preeclampsia-related MVM.
- Its diagnostic location is the decidual arterial compartment—not fetal villous capillaries.

Decidual pathology versus villous pathology

Maternal vessel compartment

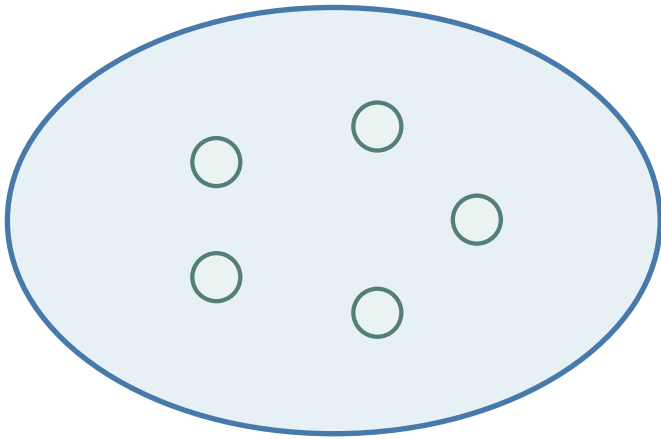
- Abnormal spiral-artery remodeling
- Decidual arterial thrombi
- Fibrinoid necrosis
- Acute atherosclerosis

Villous exchange compartment

- Increased syncytial knots
- Small, shrunken distal villi
- Expanded intervening maternal vascular space
- Placental tissue infarcts

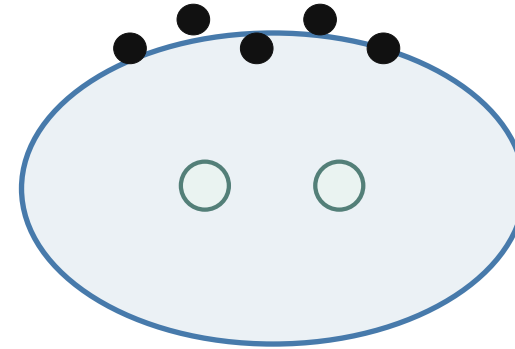
Villous ischemic changes: A schematic contrast

Ordinary terminal villus



Fetal capillaries

Ischemic distal villus



Smaller villus + syncytial knots

The drawing explains features—not diagnostic thresholds or specimen appearance.

Original schematic — not a histologic specimen

Increased syncytial knots signal villous stress

Exaggerated knotting forms part of the ischemic villous phenotype.

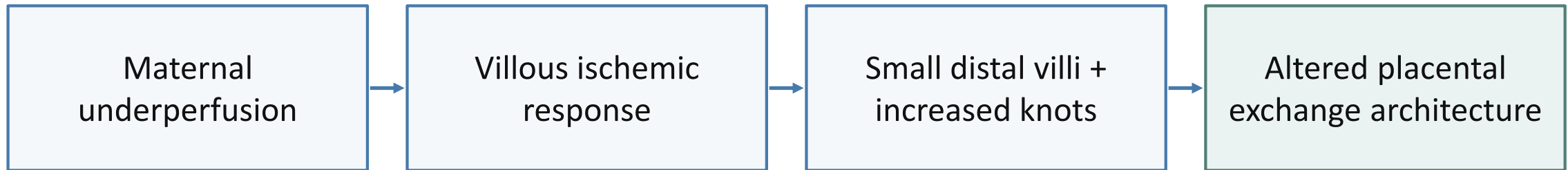
- An exaggerated number of syncytial knots is described in affected chorionic villi.
- The finding belongs to a pattern of villous and trophoblastic changes.
- Interpret the change together with villous size and maternal placental findings.

Distal villous hypoplasia changes villous architecture

The microscopic pattern is defined morphologically.

- Chorionic villi are smaller and shrunken.
- Increased maternal vascular space separates the altered villi.
- This combination constitutes distal villous hypoplasia.

Villous underdevelopment reduces exchange architecture



The final relationship is conceptual: A structural consequence, not a measured exchange-rate claim.

Placental hypoplasia is not distal villous hypoplasia

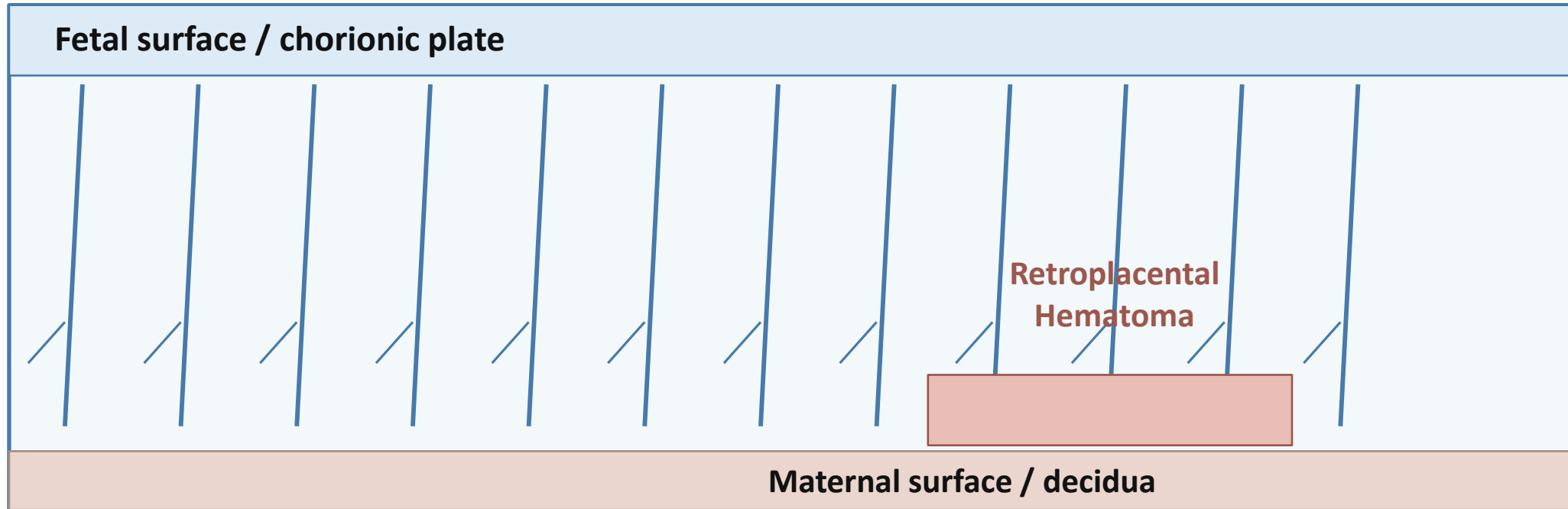
Placental hypoplasia

- Whole-organ assessment
- Placental weight below 10th percentile for gestational age
- Gross organ mass reduced
- A component of MVM

Distal villous hypoplasia

- Microscopic exchange architecture
- Small, shrunken terminal villi
- Increased intervillous maternal space
- An ischemic villous pattern

Retroplacental hematoma implicates the maternal interface



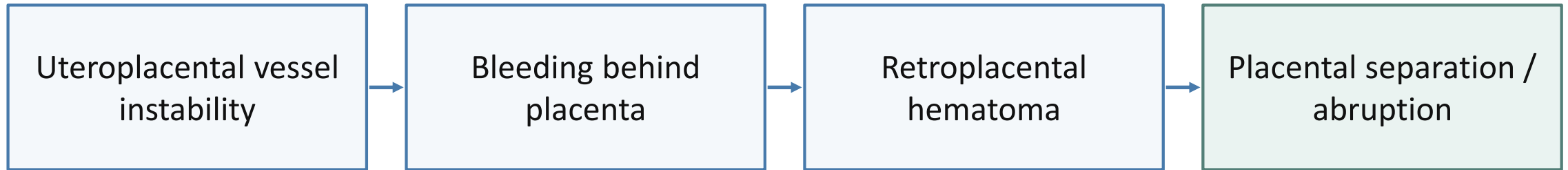
Compartment schematic — not gross pathology

Retroplacental hematoma reflects bleeding at the interface

Bleeding and unstable uteroplacental vessels may coexist with MVM.

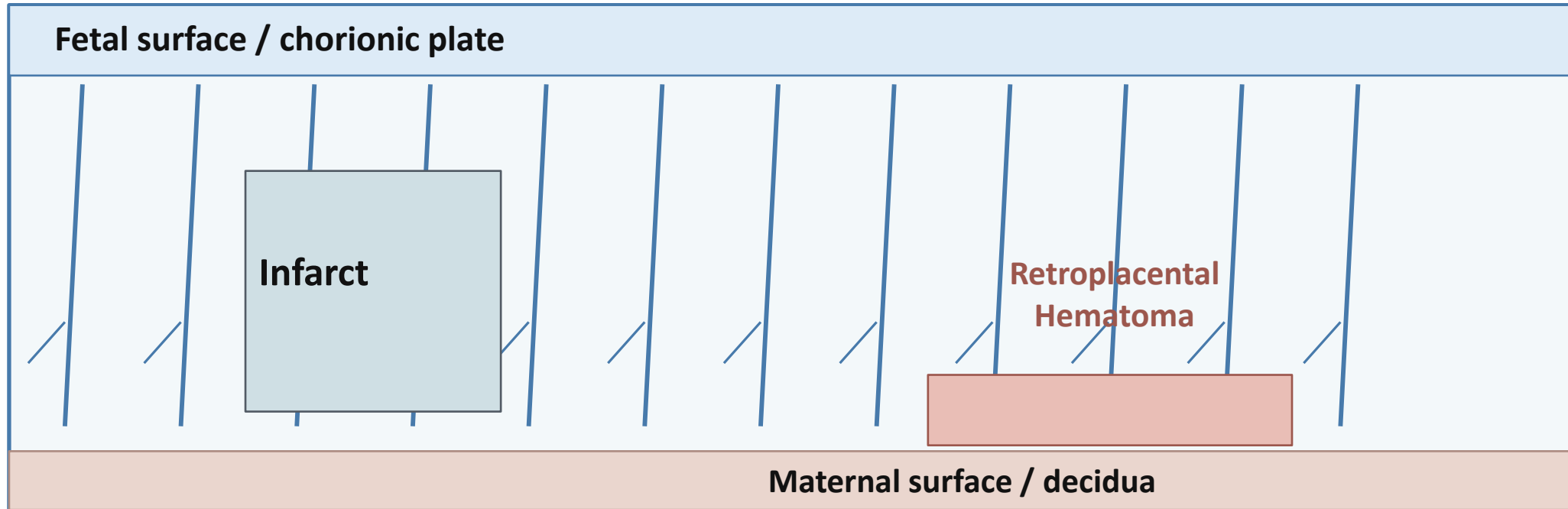
- Retroplacental hematomas may accompany preeclampsia-associated malperfusion.
- A retroplacental blood collection is anatomically distinct from villous capillary thrombosis.
- Placental abruption is also included among the MVM-associated findings.

Abruption links maternal bleeding to placental compromise



This simplified relationship explains why a maternal-interface lesion may accompany poor perfusion.

Recognize lesions by placental compartment



Compartment schematic — not gross pathology

Morphologic pattern of maternal vascular malperfusion

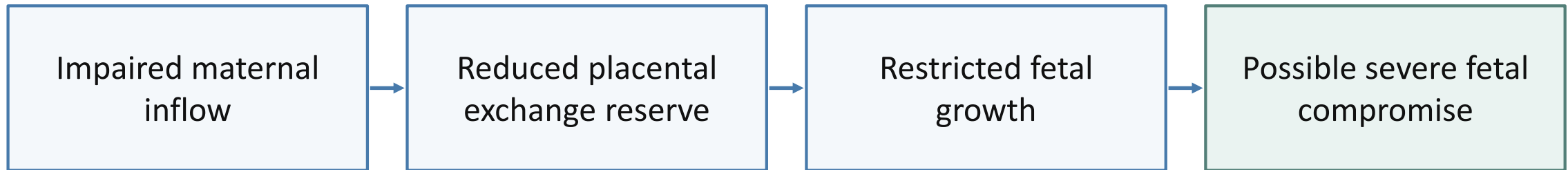
Finding	Morphologic feature	Compartment
Placental hypoplasia	Organ weight <10th percentile	Whole placenta
Placental infarcts	Ischemic tissue damage	Placental parenchyma
Decidual vasculopathy	Remodeling failure / thrombi / atherosclerosis	Maternal vessels
Distal villous hypoplasia	Small villi + increased maternal space	Chorionic villi
Retroplacental hematoma	Bleeding at maternal interface	Basal placental surface

Fetal growth restriction is a major clinical correlate

The fetus depends on sustained placental delivery.

- The primary MVM section identifies intrauterine growth restriction at the milder end of its effect spectrum.
- Reduced maternal intervillous supply provides the pathophysiologic link.
- Placental pathology and fetal growth information should be interpreted together.

Clinical consequence: A fetoplacental sequence



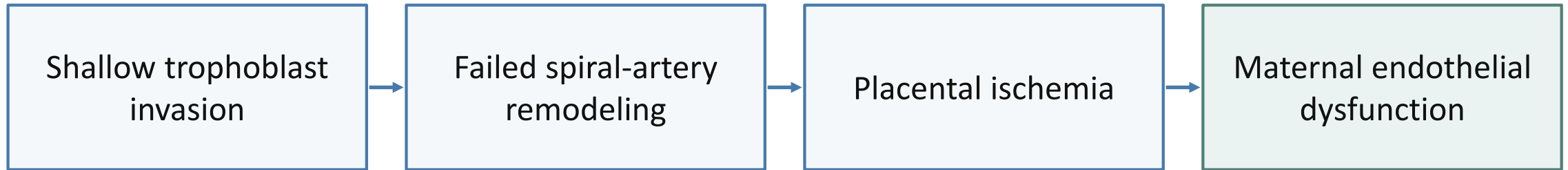
Source supports fetal growth restriction and severe uteroplacental ischemia; Severity is variable.

Preeclampsia is an associated maternal syndrome

Placental perfusion and systemic maternal effects intersect.

- MVM may be associated with severe uteroplacental ischemia and maternal preeclampsia.
- Preeclampsia involves diffuse maternal endothelial dysfunction.
- Clinical preeclampsia and morphologic MVM are related—but not synonymous—descriptions.

Preeclampsia begins with abnormal placentation



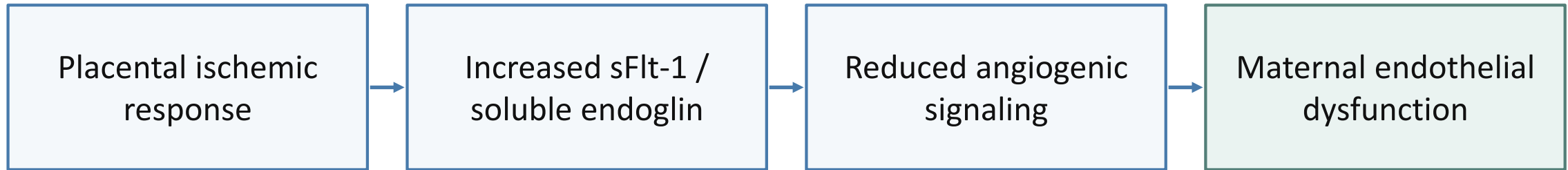
The placenta-centered sequence is presented by Robbins as the principal pathophysiologic model.

Placental antiangiogenic factors link placenta to mother

The source describes a proposed mediator mechanism.

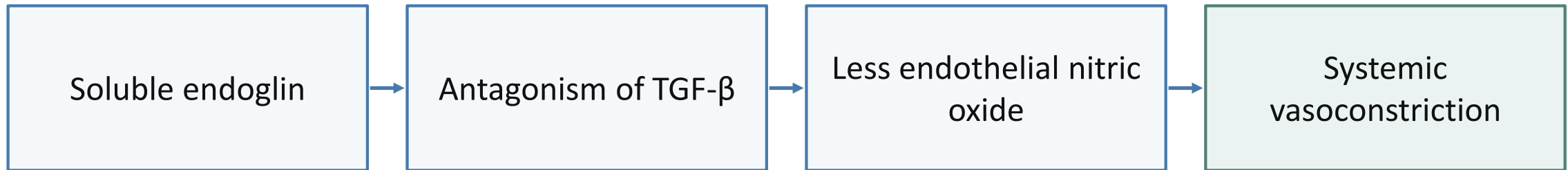
- Ischemic placental tissue is proposed to release factors into maternal circulation.
- SFlt-1 antagonizes vascular endothelial growth factor (VEGF) signaling.
- Soluble endoglin antagonizes transforming growth factor- β (TGF- β) signaling.
- Together these pathways are implicated in systemic endothelial dysfunction.

Antagonism of VEGF and TGF- β is mechanistically important



This is a proposed pathogenic pathway in preeclampsia, not a stand-alone placental histologic diagnostic test.

Endoglin can contribute to vasoconstriction



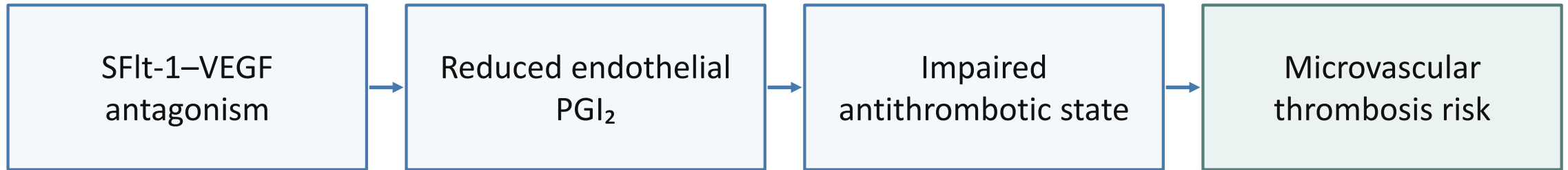
A mechanistic formulation (“may contribute”), so the arrow is an explanatory model.

Endothelial dysfunction explains maternal manifestations

Preeclampsia is systemic, despite a placental origin.

- Vasoconstriction contributes to maternal hypertension.
- Increased endothelial permeability contributes to proteinuria and edema.
- Coagulation abnormalities may produce microvascular thrombi and organ injury.

A thrombotic pathway accompanies endothelial injury



Decreased prostacyclin (PGI₂) is associated with hypercoagulability in preeclampsia.

Placental lesion and maternal syndrome are distinct

MVM: Pathology descriptor

- Pattern of placental and vascular lesions
- Hypoplasia, infarcts and villous ischemia
- Decidual artery pathology ± hematoma
- Interpretation on placental examination

Preeclampsia: Clinical syndrome

- Maternal endothelial dysfunction
- Hypertension and systemic manifestations
- Can be linked to abnormal placentation
- May be accompanied by MVM morphology

Maternal versus fetal vascular malperfusion

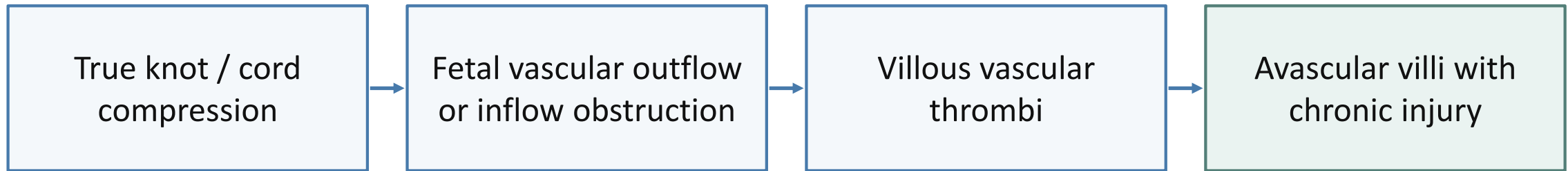
Maternal vascular malperfusion

- Problem originates in maternal inflow.
- Intervillous perfusion is impaired.
- Findings include infarcts, decidual vasculopathy and ischemic villi.
- Fetal growth restriction may result.

Fetal vascular malperfusion

- Problem involves the fetal-side placental circulation.
- Cord obstruction is a major source example.
- Villous vascular thrombi and avascular villi are described.
- Do not relabel FVM morphology as MVM.

Umbilical cord obstruction produces a different pathway



Chronic/intermittent cord obstruction is associated with fetal vascular malperfusion.

Avascular villi favor a fetal-side vascular process

Location of injured vessels matters.

- Avascular villi occur in fetal vascular malperfusion.
- Vascular thrombi within fetal villous circulation support the fetal-side pathway.
- This differs from shrunken distal villi and maternal arterial lesions emphasized in MVM.

Nonvascular inflammatory patterns are a different process

MVM-associated pattern

- Abnormal maternal vessels
- Small ischemic villi and syncytial knots
- Infarcts or placental hypoplasia
- Possible retroplacental hematoma

Placental infection pattern

- Ascending infection may inflame membranes.
- Neutrophilic chorioamnionitis is described.
- Hematogenous pathogens may produce villitis.
- Diagnose inflammatory lesions separately from MVM.

Placenta previa is an implantation-site disorder

This adjacent topic is assigned to this teaching unit by the source archive.

- Placenta implants in the lower uterine segment and may cover the cervix.
- Its defining mechanism is abnormal location, not simply maternal arterial underperfusion.
- Severe bleeding in late pregnancy is a key clinical consequence.

Placenta accreta spectrum is an attachment disorder

Defective decidua changes the villus–myometrium interface.

- Absence of decidua allows villi to contact or invade myometrium.
- Failure of placental separation at delivery can cause major postpartum hemorrhage.
- It should not be equated with MVM simply because both involve abnormal implantation.

MVM, previa and accreta answer different questions

Primary abnormality

- MVM: Maternal circulation cannot supply intervillous space adequately.
- Previa: Low uterine implantation in relation to cervix.
- Accreta spectrum: Focal/complete decidual absence with adherent or invasive villi.

Dominant pathologic/clinical signal

- MVM: Ischemic placental and decidual vascular lesions.
- Previa: Potentially serious antepartum hemorrhage.
- Accreta: Failure to separate and postpartum hemorrhage.

Applied case: A small placenta

A placenta from a growth-restricted pregnancy weighs below the gestational-age 10th percentile. Examination also identifies infarcts and small distal villi.

Which maternal-perfusion pattern best integrates these findings?

Case interpretation: Multiple MVM features

Interpretation

- Placental hypoplasia is defined relative to gestational age.
- Infarcts and small distal villi are additional MVM-associated lesions.
- A pattern-based MVM interpretation is stronger than a conclusion from weight alone.

Applied case: Hypertension and decidual vessels

A pregnancy is complicated by maternal hypertension. The placenta has numerous infarcts; Decidual arteries demonstrate persistent muscular walls and intimal lipid deposition.

Which pathophysiologic link explains this lesion combination?

Case interpretation: Failed arterial remodeling

Interpretation

- Persistence of muscular spiral arteries indicates impaired physiologic remodeling.
- Intimal lipid deposition is termed acute atherosclerosis.
- Reduced uteroplacental perfusion connects vascular pathology to ischemic placental lesions.

Applied case: Obstruction of a cord vessel

A tightly constricting umbilical-cord knot accompanies placental villous vascular thrombi and avascular villi.

Is this a maternal or fetal vascular malperfusion pattern?

Case interpretation: Fetal vascular malperfusion

Interpretation

- The umbilical-cord lesion obstructs the fetal circulation.
- Villous vascular thrombi and avascular villi are described FVM findings.
- Do not classify these lesions as MVM solely because both can impair fetal growth.

Applied case: Severe bleeding at delivery

Following prior uterine surgery, the placenta does not separate; Chorionic villi directly contact the myometrium where decidua is absent.

Which placental disorder is illustrated, and why is it not MVM?

Case interpretation: Placenta accreta spectrum

Interpretation

- The principal defect is absence of decidua with villous attachment to myometrium.
- Placental separation fails, producing a risk of major postpartum hemorrhage.
- This is a decidual attachment disorder, not the definition of maternal intervillous underperfusion.

Approach to an MVM-pattern placenta

Use morphology, compartment and context.

- First: Assess placental size relative to gestational age.
- Second: Identify infarcts and ischemic villous alterations.
- Third: Inspect decidual vessels and the retroplacental interface.
- Finally: Correlate with fetal growth, maternal disease and obstetric events.

Diagnostic pitfalls and safeguards

Avoid collapsing unlike pathologic patterns into one label.

- No single listed MVM lesion is obligatory in the Robbins definition.
- Placental weight requires a gestational-age reference.
- Avascular villi from cord obstruction indicate the fetal circulation.
- MVM morphology and maternal preeclampsia are related, not interchangeable diagnoses.
- Previa or accreta should be classified by their own defining abnormalities.

Core principles to retain

Five linked statements summarize the lecture.

- MVM = inadequate maternal supply to intervillous placental blood spaces.
- Abnormal spiral-artery remodeling is a major pathophysiologic link.
- Hypoplasia, infarcts, vascular lesions and villous ischemia constitute a pattern.
- Fetal growth restriction and maternal preeclampsia are important associations.
- Fetal-side thrombi/avascular villi and implantation disorders require separate classification.

Source and reading

Primary textbook

Robbins, Cotran & Kumar: Pathologic Basis of Disease. 11th ed. Chapter 22, The Female Genital Tract; pp. 934–939.

Focus

Maternal Vascular Malperfusion; placental anatomy and gestational context; associated preeclampsia pathology. Abnormalities of Placental Implantation are included as distinct related entities.

Diagrams in this presentation are editable teaching schematics, not diagnostic images.