

Trichinellosis

Transmission, larval migration, skeletal-muscle pathology and host response

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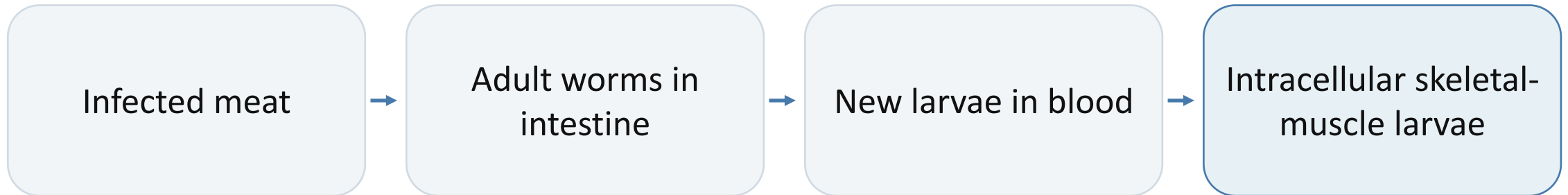
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Learning objectives

- Trace infection from ingestion of larvae to intramuscular encystment.
- Explain clinical manifestations from organ distribution and inflammation.
- Recognize the muscle, cardiac, pulmonary and CNS pathologic patterns.
- Interpret the Th2 response and the role—and limits—of serodiagnosis.

One infection, two principal human compartments



Intestinal adult stage and intramuscular larval stage create distinct clinicopathologic phases.

Trichinella is a tissue-invasive nematode

- Trichinella spp. Are roundworms acquired as larvae in meat.
- Unlike an infection confined to the gut, new larvae disseminate into host tissues.
- Skeletal muscle encystment is the defining persistent tissue relationship.

Species cited

T. Spiralis

The species used for the detailed pathogenetic description.

T. Nativa

One of the Trichinella species named in infected animals.

T. Britovi

Another species cited in the foodborne transmission cycle.

Species examples come from the assigned text; This is not an exhaustive taxonomy.

The infective exposure is larval tissue

Larvae already in animal muscle



Meat is inadequately cooked



Human ingests viable larvae

The entry point is oral ingestion of infected meat—not direct skin penetration or insect transmission.

Animal foods implicated by the text

Pigs

Common domestic-meat source described in the chapter.

Wild boars and other game

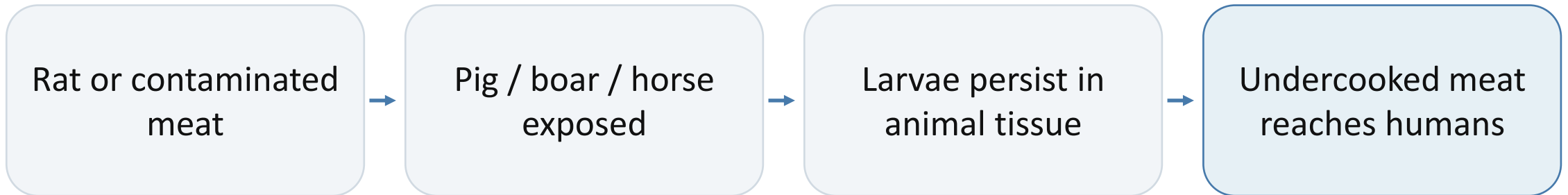
Exposure may occur outside regulated livestock systems.

Horses and bears

Additional cited sources of contaminated meat.

Risk depends on infected tissue being eaten without adequate cooking.

Transmission is embedded in a carnivorous food chain



This sequence explains why both animal feeding practices and food preparation matter.

Human risk depends on how meat is handled

Lower-risk pathway	Higher-risk pathway
Animal feed subject to cooking controls	Animals consume infectious animal tissue
Meat undergoes adequate cooking	Meat consumed undercooked
Fewer viable larvae reach humans	Viable tissue larvae may be ingested

Livestock control modifies the reservoir

- The text links lower infection in U.S. Pigs to rules for cooking food fed to hogs.
- Reducing infected material in the animal diet interrupts an upstream transmission step.
- This is distinct from adequate cooking of meat before human consumption.

Textbook epidemiology requires temporal context

Worldwide (textbook estimate)	United States (textbook report)
About 10,000 cases per year	Usually about 20 reported human infections yearly
Described as still widespread in many regions	Marked decline in infected pigs attributed to feeding regulation
These are Robbins estimates, not verified current surveillance counts	These are Robbins estimates, not verified current surveillance counts

Noncommercial livestock and game remain relevant

- The chapter emphasizes settings in which undercooked game meat is consumed.
- Commercial-livestock prevention measures do not remove all wildlife exposures.
- Exposure history must specify the animal source and meat preparation.

Intestinal maturation initiates the human phase

Ingested larvae



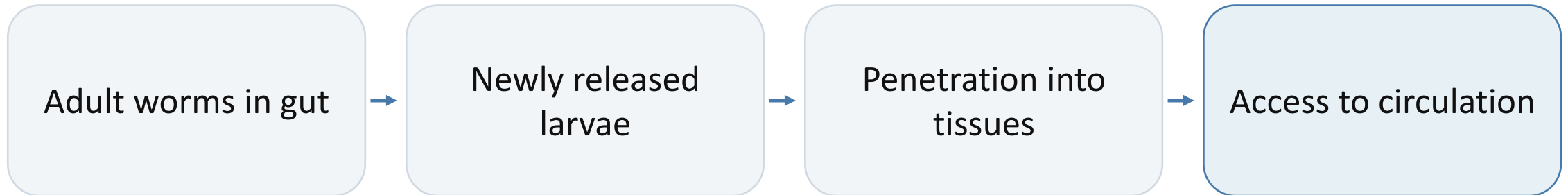
Development into intestinal
adults



Mating of adult worms

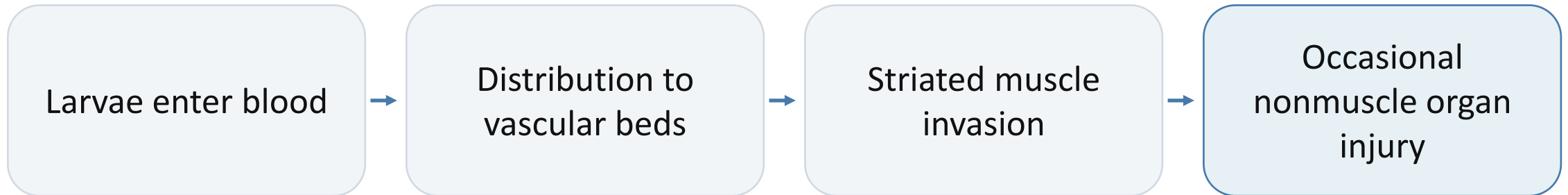
The intestinal stage precedes generation of larvae that invade host tissues.

New larvae change the anatomical target



Larval tissue invasion connects the intestinal stage to the systemic phase.

Hematogenous spread explains multisystem disease



Circulating larvae account for skeletal-muscle predominance with potential heart, lung and brain involvement.

Humans are dead-end hosts in this cycle

Within the infected human	Transmission onward
Adult worms develop in the intestine	Human-to-human continuation is not the cycle described
Larvae enter human tissues	Persistent skeletal-muscle infection does not by itself propagate the food chain
Muscle contains intracellular larvae	The source identifies humans as dead-end hosts

Larval fate is highly tissue dependent

Skeletal muscle

Intracellular encystment and long survival

Heart

Inflammation, but no encystment

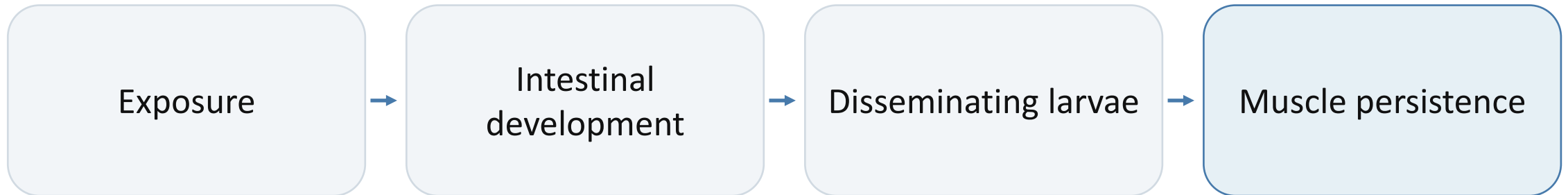
Lung

Trapping, edema and hemorrhage

Brain

Inflammation and focal gliosis

The sequence determines clinical interpretation



The prominent pathology changes as worms transition between compartments.

The invasive phase produces systemic illness

Finding

Fever

Pathologic basis

Larval invasion with host inflammatory reaction

Interpretation

Consider the fever together with exposure and tissue manifestations.

Myalgias localize the clinical burden to muscle

Finding

Myalgias

Pathologic basis

Larvae penetrate and transform striated skeletal-muscle cells

Interpretation

Pain fits the muscle-centered phase of the lifecycle.

Eosinophilia is a prominent host-response clue

Finding

Marked eosinophilia

Pathologic basis

Eosinophil-associated type 2 inflammatory response

Interpretation

The laboratory pattern is linked to an invasive helminth, not unique to *Trichinella*.

Periorbital edema belongs to the invasive pattern

Finding

Periorbital edema

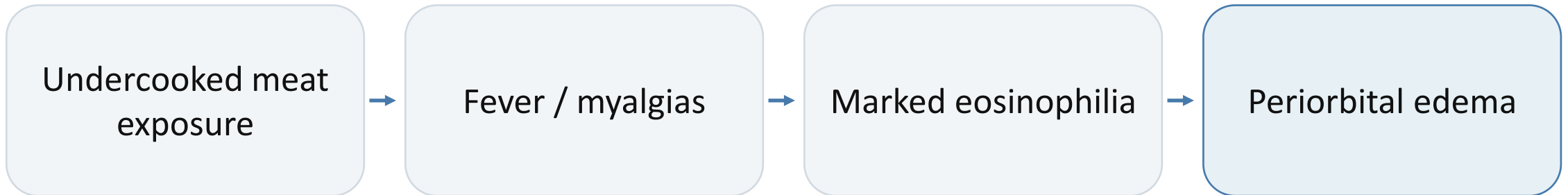
Pathologic basis

Manifestation accompanying disseminating *Trichinella* larvae

Interpretation

Its diagnostic significance increases when paired with myalgias and eosinophilia.

The characteristic symptom cluster is relational



The source presents these as associated clinical findings—not as a validated diagnostic scoring rule.

Heavy infection can become destructive

- The invasive phase may cause widespread cellular destruction.
- The assigned text explicitly describes potentially lethal disease when infection is heavy.
- Serious complications reflect inflammatory injury beyond skeletal muscle.

Organ-specific symptoms signal different lesions

Skeletal muscle

Myalgias accompany muscle invasion

Heart

Myocarditis can progress to cardiac failure

Lung

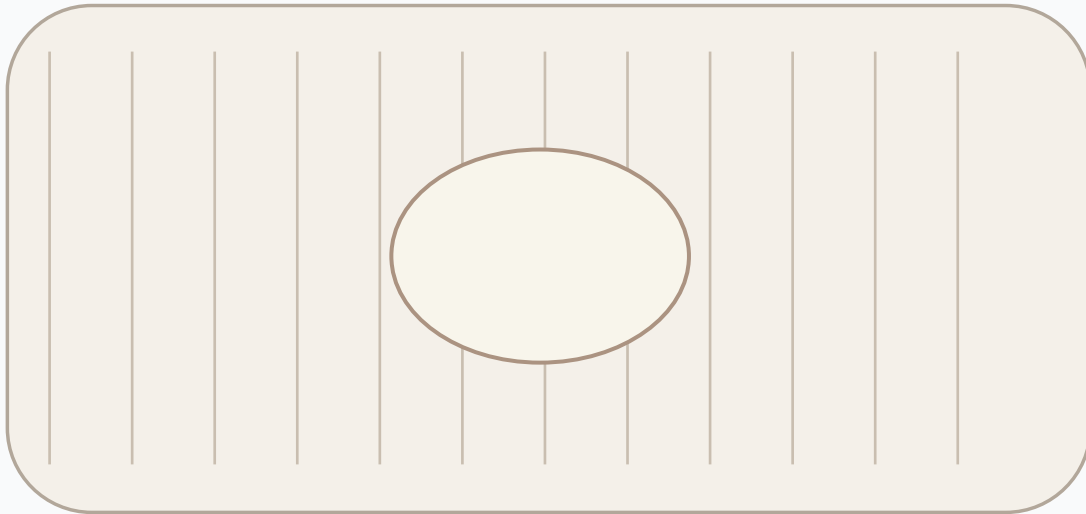
Pulmonary injury can produce dyspnea

CNS

Inflammation can produce encephalitis

Striated skeletal muscle is the main encystment site

Skeletal-muscle lesion — schematic



- Larvae enter skeletal-muscle cells.
- The parasite persists intracellularly, rather than simply remaining in interstitial space.
- The host cell is progressively reorganized around the parasite.

Highly perfused muscles are preferential targets

Respiratory

Diaphragm; Intercostal muscles

Ocular

Extraocular muscles

Upper limb

Deltoid muscle

Other

Laryngeal and gastrocnemius muscles

Perfusion and location are connected

Larvae disseminate in blood



Muscle-rich vascular beds are reached

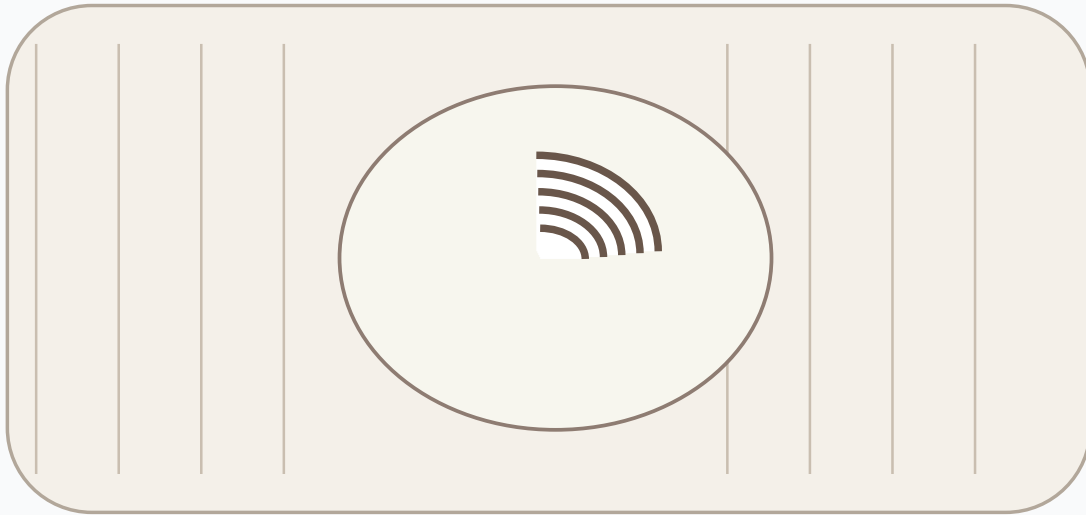


Intracellular skeletal-muscle lesions form

The source identifies preferential involvement of muscles with the richest blood supply.

The larva changes its host muscle cell

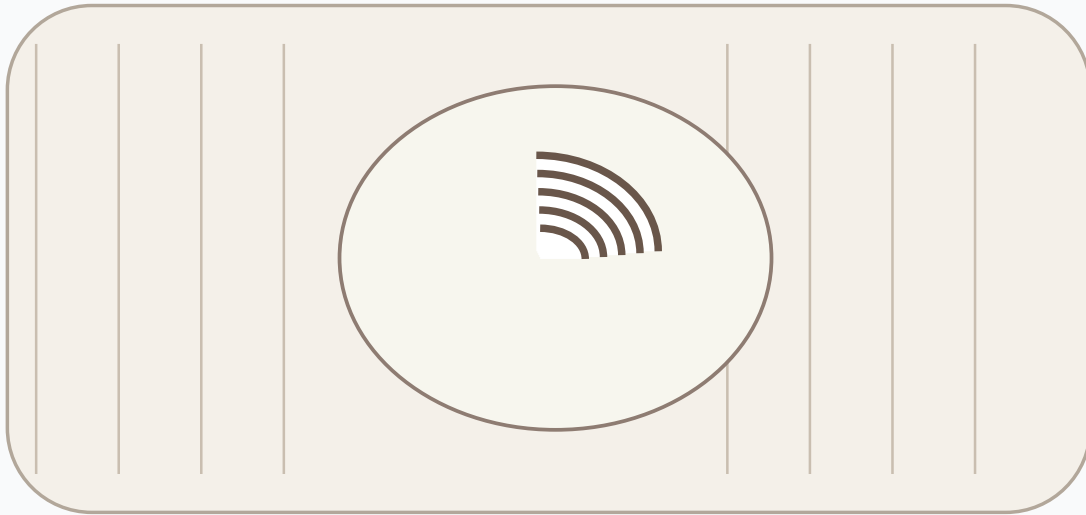
Skeletal-muscle lesion — schematic



- Loss of the normal striated appearance is characteristic.
- The parasite-associated cell is not an unchanged functioning myofiber.
- Host-cell remodeling helps explain distinctive biopsy morphology.

Intracellular larval growth is substantial

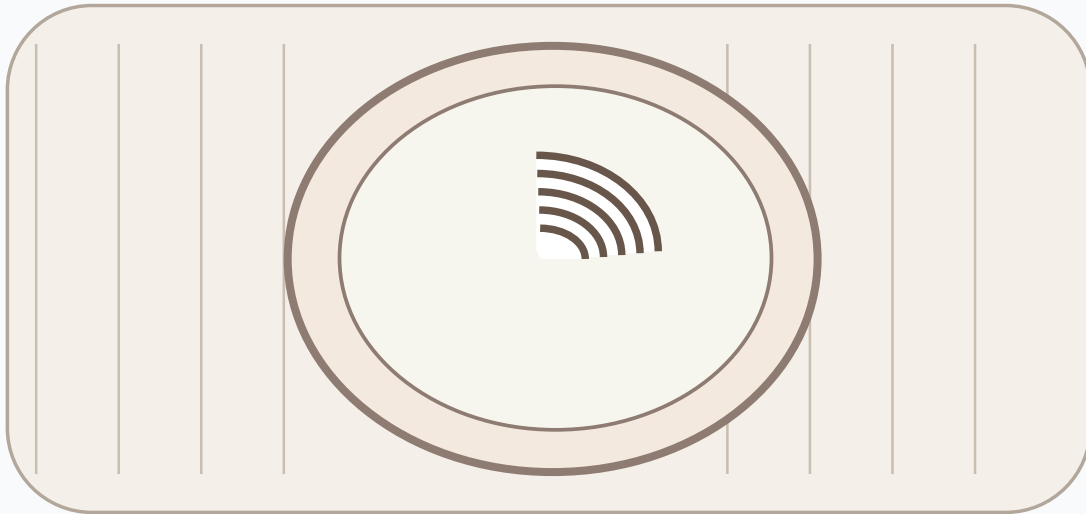
Skeletal-muscle lesion — schematic



- The source describes a dramatic increase in larval size within muscle.
- Coiled larvae are approximately 1 mm long.
- A mature lesion should be understood as a cell–parasite complex.

A collagenous capsule develops around the complex

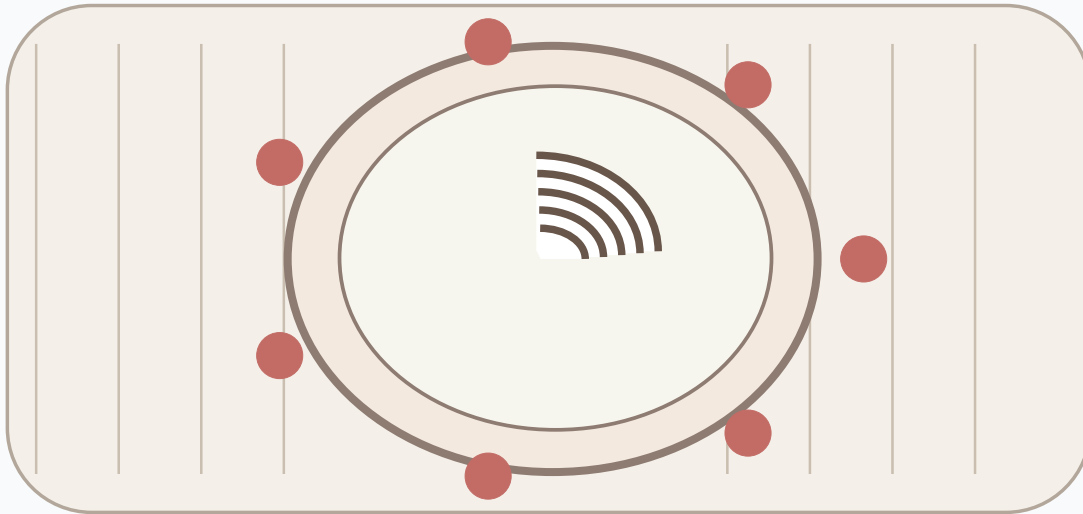
Skeletal-muscle lesion — schematic



- The altered skeletal-muscle cell gains a collagenous capsule.
- The capsule is part of the tissue response to persistent encystment.
- A capsule alone must not be confused with a hydatid cyst wall.

New vessels surround the infected muscle cell

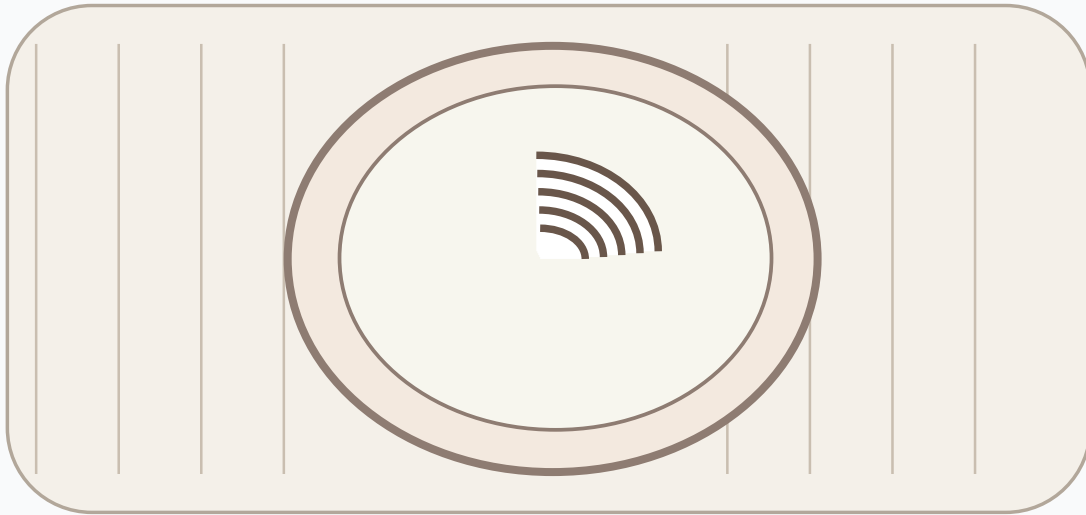
Skeletal-muscle lesion — schematic



- The parasite modifies its host cell and a vascular plexus develops outside.
- Neovascularization accompanies the stable muscle cell–parasite complex.
- The vessels lie around the infected cell, not inside the larval body.

A membrane-bound vacuole surrounds the coiled larva

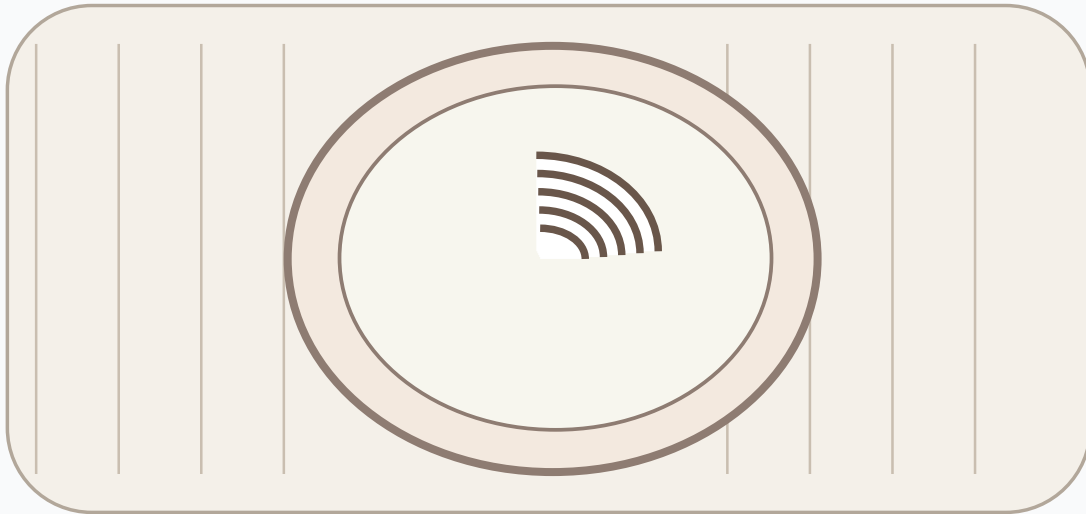
Skeletal-muscle lesion — schematic



- The larva is enclosed by an intracellular membrane-bound vacuole.
- This cellular boundary lies within the altered skeletal-muscle cell.
- Outside it is the vascular and inflammatory tissue response.

Coiling is a recognizable morphologic clue

Skeletal-muscle lesion — schematic



- The source describes coiled larvae in skeletal-muscle cells.
- Coiling, surrounding cell change and vascular response should be read together.
- The drawing is a schematic, not a substituted biopsy image.

Muscle inflammation has two major cell categories

Eosinophils

A conspicuous component of the infiltrate.

Mononuclear cells

Cells around affected fibers accompany the eosinophils.

Parasite status

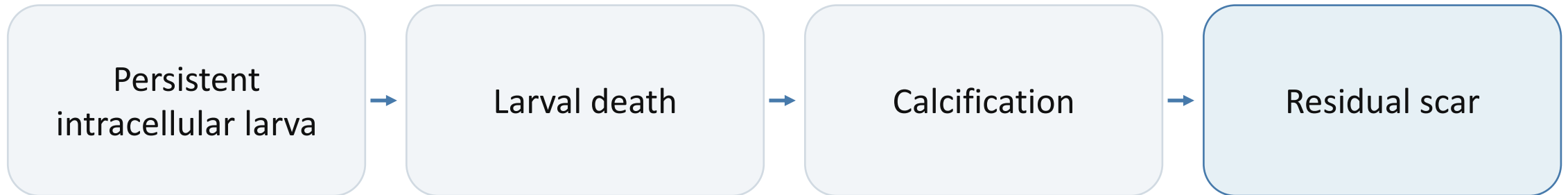
Inflammation intensifies around dying larvae.

The infiltrate is not itself proof that living intramuscular larvae are eliminated.

Parasite viability alters inflammatory intensity

Persistent parasite complex	Dying parasite complex
May remain largely asymptomatic	Greater surrounding inflammatory infiltrate
Long-term tissue persistence	Progression toward calcification and scar
Coiled larva may remain in muscle	Residual lesion may outlast the parasite

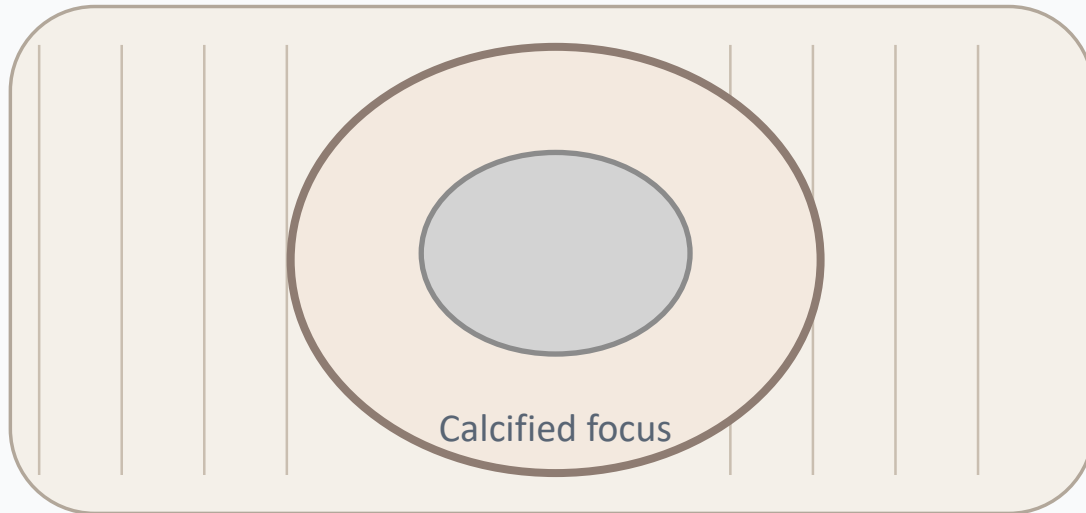
Calcification is an end-stage tissue event



The scar may be sufficiently characteristic to suggest the diagnosis even after the worm dies.

Muscle scarring provides retrospective evidence

Skeletal-muscle lesion — schematic



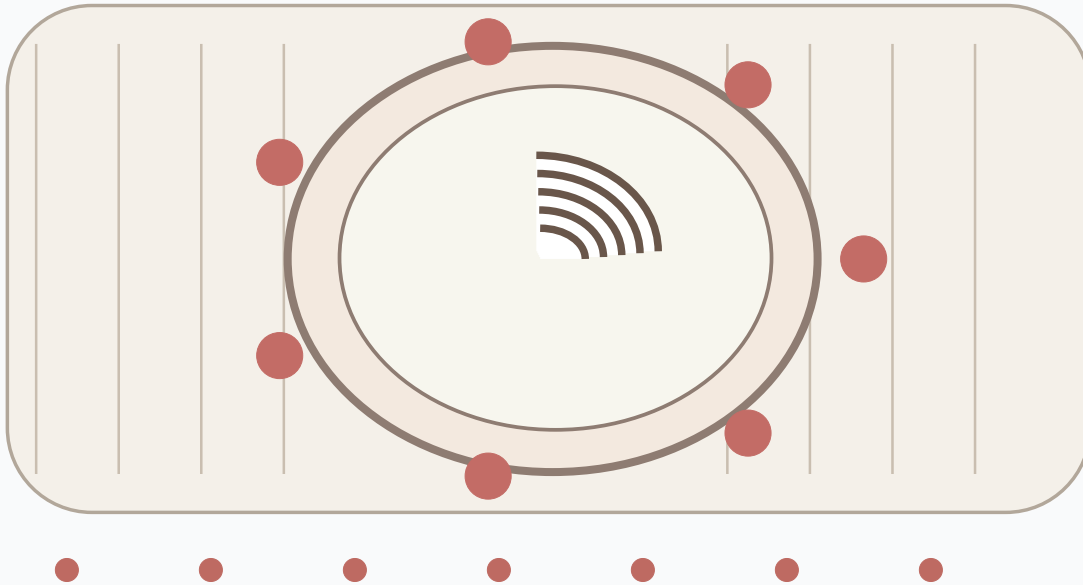
- The late lesion can contain calcified parasite remnants.
- A localized scar remains after larval degeneration.
- Such morphology suggests prior infection; Clinical context remains important.

Persistence need not imply persistent symptoms

Parasite in muscle	Clinical expression
The cell–parasite complex may survive for years	The complex is described as largely asymptomatic
Larval degeneration ultimately occurs	Inflammation and residual calcified scarring may follow
Morphology may persist	Current symptoms cannot be inferred from tissue persistence alone

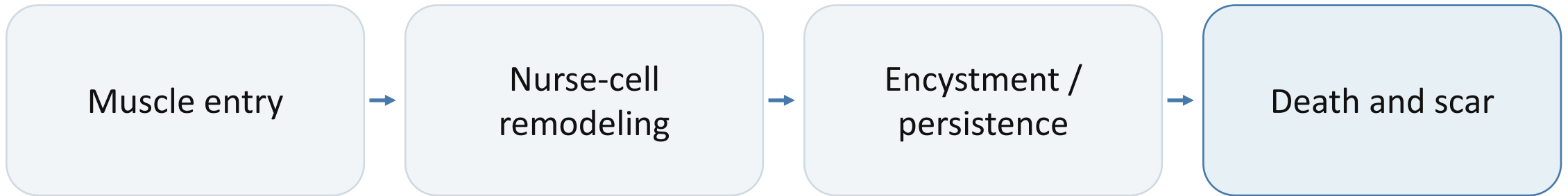
Read the complete muscle lesion as layered anatomy

Skeletal-muscle lesion — schematic



- Central coiled intracellular larva and vacuole.
- Altered striated muscle cell with collagenous capsule.
- Outside: New vessels plus eosinophil-rich mononuclear inflammation.

Histologic findings change with lesion age



The same infection can yield morphologically different tissue snapshots depending on timing.

Cardiac injury is interstitial myocarditis

Finding

Patchy interstitial myocarditis

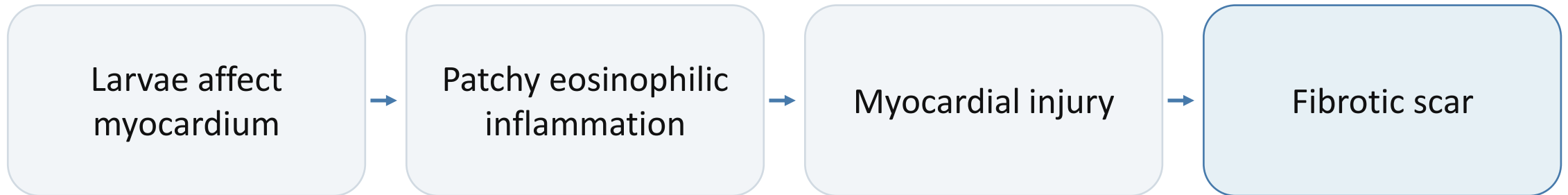
Pathologic basis

Eosinophils and scattered giant cells are described in the heart.

Interpretation

Inflammatory myocardial injury can compromise function.

Myocarditis can leave cardiac scarring



Cardiac failure is a potential severe clinical consequence of cardiac involvement.

Cardiac larvae may be hard to demonstrate

Heart	Striated skeletal muscle
Larvae do not encyst in the heart	Larvae can persist as intracellular encysted forms
They die and disappear	Lesions can remain detectable for years
The inflammatory pattern may dominate biopsy	The coiled larval complex may dominate morphology

Pulmonary larvae cause vascular-centered injury

Finding

Focal pulmonary edema

Pathologic basis

Larvae trapped in lung tissue produce local injury.

Interpretation

The source also reports focal hemorrhage.

Eosinophils may accompany lung injury

Structural pulmonary findings	Inflammatory accompaniment
Focal edema	Sometimes an allergic-type infiltrate
Focal hemorrhage	Eosinophils can be prominent
Trapped larvae are implicated	The inflammatory pattern is variable

The CNS shows inflammation and gliosis

Finding

Diffuse CNS inflammatory infiltrate

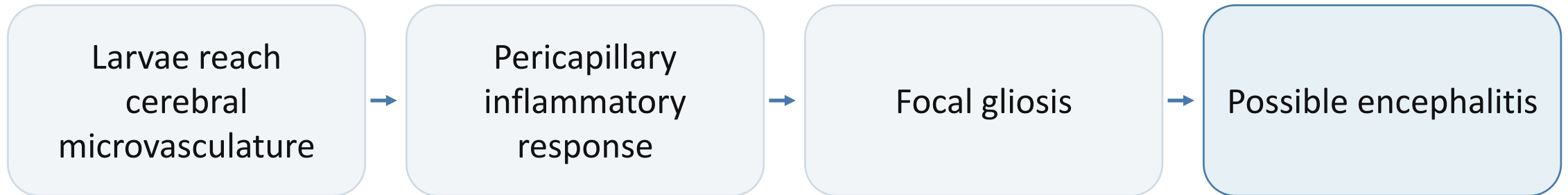
Pathologic basis

Lymphocytes and eosinophils accompany disseminated larvae.

Interpretation

Focal gliosis develops around small cerebral capillaries.

Pericapillary gliosis locates CNS tissue response



The source places focal gliosis in and around small brain capillaries.

Do not confuse organ tropism with encystment

Tissue	Robbins lesion	Encystment?
Striated skeletal muscle	Coiled intracellular larvae; Vascularized complex	Yes
Heart	Patchy myocarditis; Eosinophils / giant cells	No
Lung	Focal edema / hemorrhage; Possible eosinophils	Not described
Brain	Lymphocytes / eosinophils; Focal gliosis	Not described

Severe manifestations are organ specific

Heart

Potential cardiac failure

Lungs

Potential dyspnea

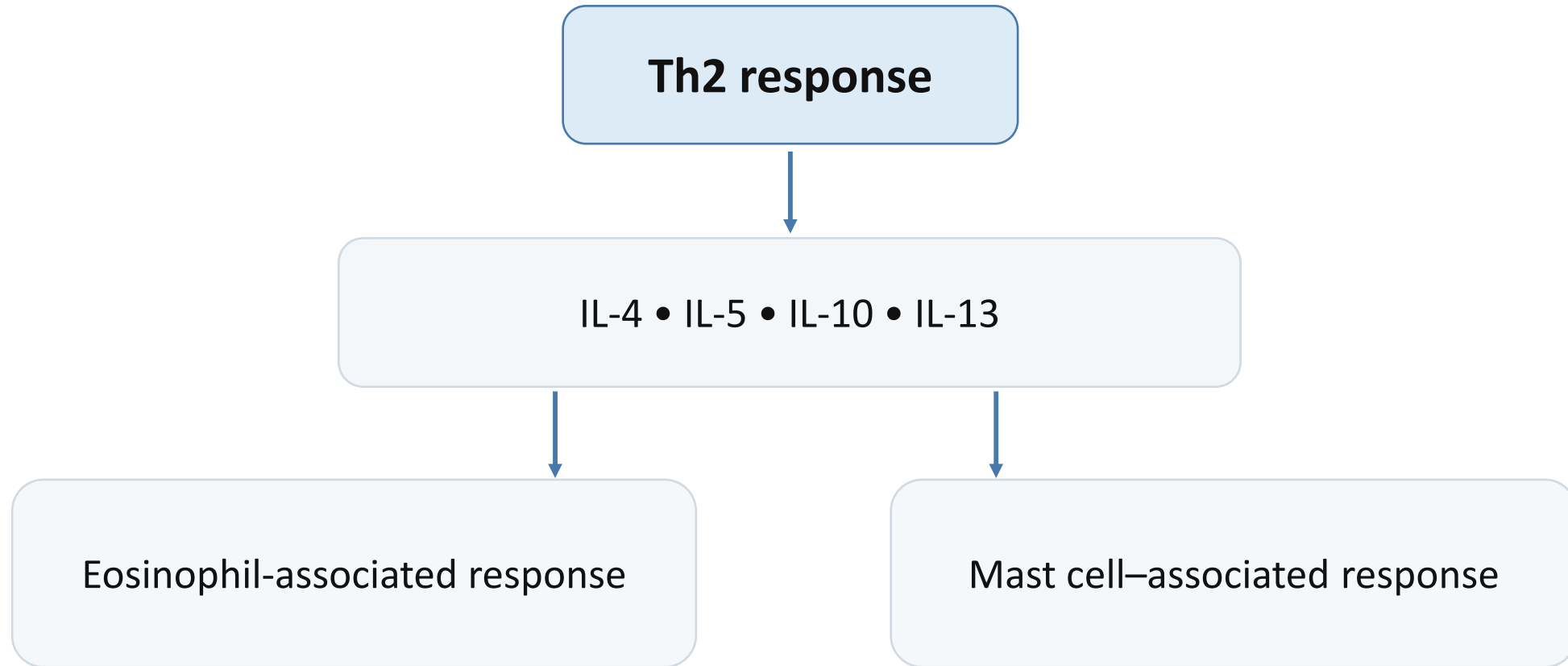
Brain

Potential encephalitis

Skeletal muscle

Myalgias and chronic lesion persistence

Trichinella elicits a Th2-type response



An immune response is not proof of clearance of intramuscular larvae.

The cited Th2 cytokines are a defined set

IL-4 and IL-13

Listed in the *Trichinella* Th2 profile.

IL-5

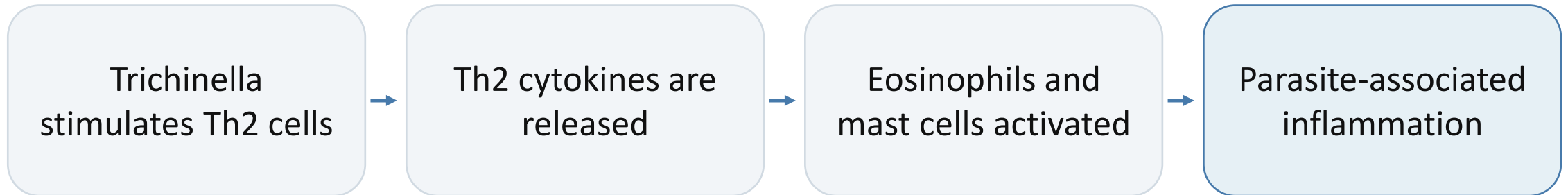
Listed in the profile associated with eosinophils.

IL-10

Also explicitly included' Th2-associated response.

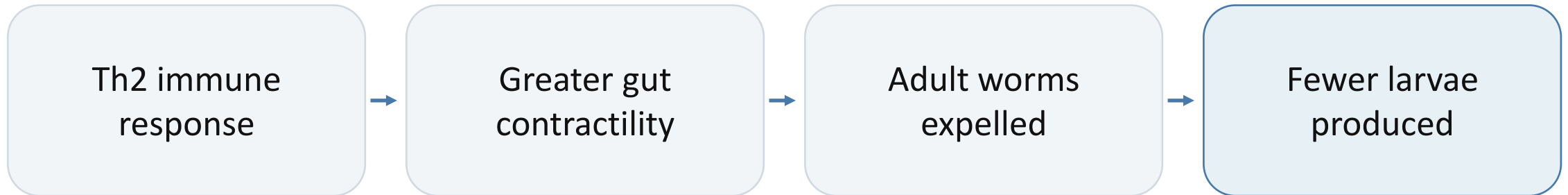
This slide reproduces the cytokine set in the assigned excerpt without inventing pathway details.

Eosinophils and mast cells link immunity to lesions



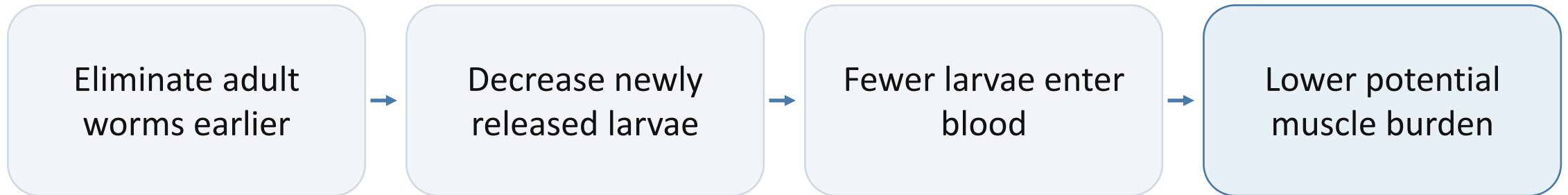
Immune activation helps explain the marked eosinophilia and eosinophil-rich tissue infiltrates.

Intestinal contractility is protective in animal models



Robbins specifically attributes this observation to animal models of *T. Spiralis* infection.

Reducing intestinal adults can reduce muscle seeding



The protection is indirect; It follows reduced ongoing larval production in the intestine.

Intramuscular immune efficacy remains uncertain

Supported in animal models	Unresolved in source
Th2-associated intestinal expulsion	Whether muscle eosinophils/mononuclear cells eliminate larvae
Indirectly lower muscle larval numbers	Inflammatory intensity is not a measure of efficacy
Association with host protection	Avoid inferring complete larval clearance

Antibodies recognize larval antigens

Persistent / invading larval
antigens



Host antibody response



Antibody detectable in
serologic assay

Serodiagnosis is supported in the source; No assay sensitivity, cutoff or testing window is specified.

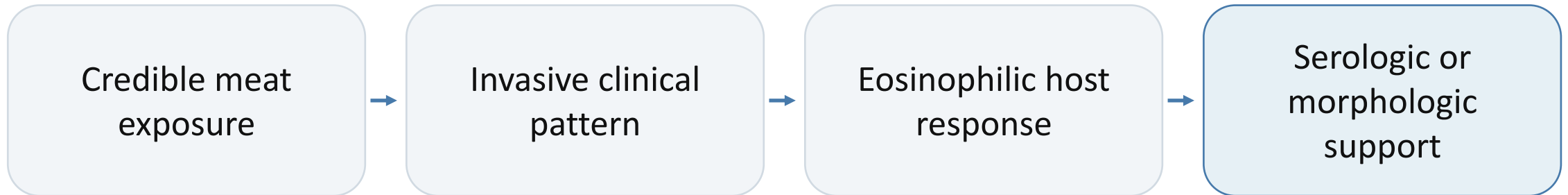
Tyvelose is the named immunodominant epitope

- Tyvelose is identified as a carbohydrate epitope in larval antigens.
- Antibodies to larval antigens are useful for serodiagnosis.
- The chapter also notes that such antibodies may reduce reinfection.

Serology and morphology answer different questions

Antibody evidence	Tissue morphology
Detects host response to larval antigens	Can display the coiled muscle parasite–cell complex
Useful for serodiagnosis	Can show inflammatory response, calcification or scar
Does not itself locate a lesion	Morphologic interpretation depends on stage and sampling

A diagnosis is a clinicopathologic synthesis



Conceptual integration only: Robbins does not prescribe a formal four-item diagnostic algorithm.

Case: Exposure before febrile myalgia

A patient develops fever and diffuse muscle pain after eating inadequately cooked wild game. Marked eosinophilia and periorbital edema are present.

What parasite life-cycle transition best explains the syndrome?

- A. Mature worms confined to the intestine
- B. Larval dissemination from the intestine into tissues
- C. A parasite restricted to the biliary tree

Case: Interpret the muscle lesion

A skeletal-muscle section contains a coiled intracellular larva, an altered muscle fiber, and nearby vascular and eosinophil-rich inflammatory tissue.

Which structural change is especially characteristic?

- A. Formation of a hepatic daughter hydatid cyst
- B. Adult worm development inside a cardiomyocyte
- C. Muscle cell remodeling with loss of striations

Case: Why is myocarditis not a negative biopsy?

A patient with compatible exposure and systemic manifestations develops myocarditis. The cardiac sample has eosinophils and scattered giant cells but no identifiable intact larvae.

Which source-based explanation is most defensible?

A. Skeletal muscle and heart show identical permanent cysts

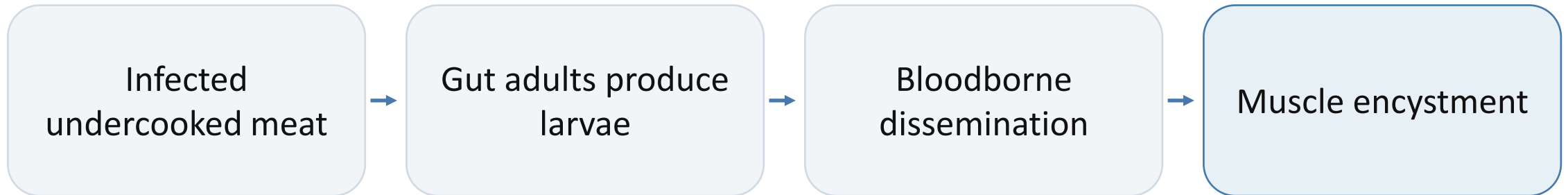
B. Cardiac larvae die and disappear without encysting

C. The disease cannot involve the heart

Differentiate Trichinella from cestode tissue forms

Trichinellosis	Cysticercosis	Hydatid disease
Nematode larva in skeletal muscle	Larval Taenia solium (cysticercus)	Larval Echinococcus cyst
Coiled intracellular larva	Ovoid cyst with an invaginated scolex	Cyst with a laminated outer wall
Remodeled muscle cell; New vessels	Cyst degeneration can lead to scar or calcification	Germinative layer may form daughter cysts

Integrated pathogenesis: The essential chain



Extra-muscular inflammation can threaten the heart, lungs and CNS; Th2 immunity has compartment-specific effects.

Five high-yield clinicopathologic anchors

- Exposure: viable larvae in inadequately cooked infected meat.
- Pathway: intestinal adults → disseminating larvae → encysted skeletal muscle.
- Muscle: altered cell, intracellular coil, capsule, new vessels and eosinophils.
- Complications: cardiac, pulmonary and cerebral inflammation.
- Response: Th2 immunity, uncertain muscle clearance, antibody-based serodiagnosis.