

Electrical Injury

Pathogenesis, tissue effects and clinicopathologic interpretation

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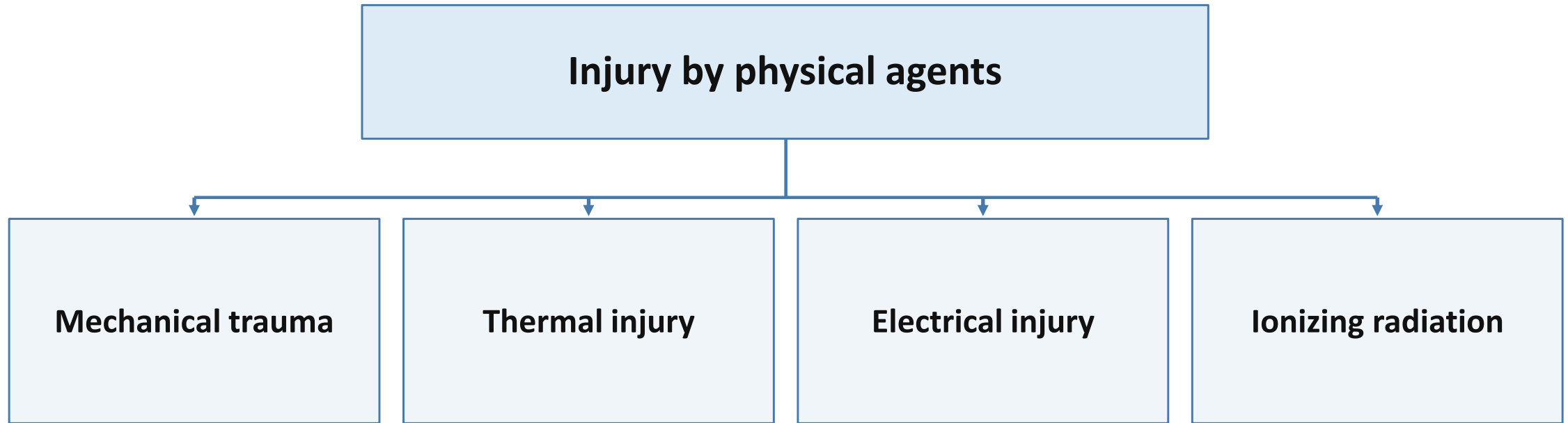
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Learning objectives

- Distinguish thermal burns from electrical disruption of excitable tissues.
- Explain why amperage, contact duration and current path govern injury.
- Relate alternating current, wet contact and high-voltage exposure to major outcomes.
- Apply burn morphology and systemic burn complications to extensive electrical injury.

Electrical injury among physical-agent disorders



Electrical injury has distinct electrical and heat-generating mechanisms.

Two fundamental classes of electrical damage

Heat-mediated tissue damage	Disruption of nerve impulse conduction
Burns at contact sites	Ventricular fibrillation
Burns within internal organs	Failure of cardiac or respiratory centers
Extent influenced by current and time	Immediate functional catastrophe may precede visible burns

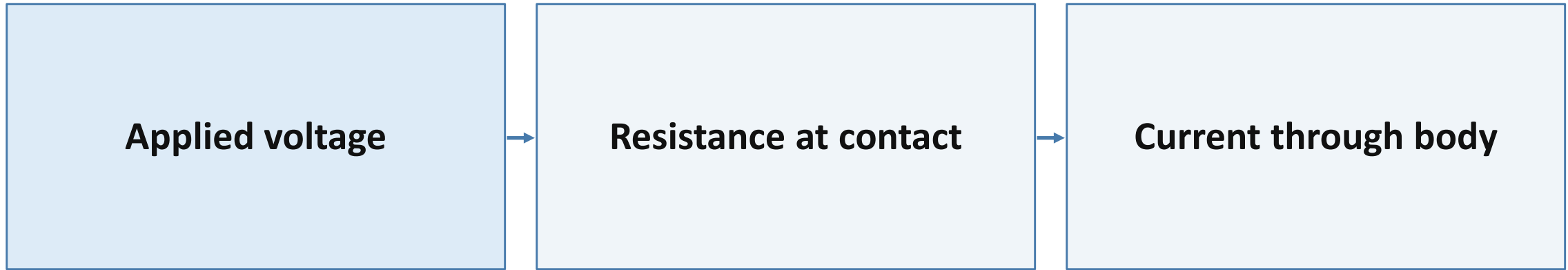
Common exposure environments

- Low-voltage contacts can occur in domestic and occupational environments.
- High-voltage power lines are a separate hazardous exposure setting.
- Lightning is a classic high-voltage source.
- Exposure category alone does not specify the final physiologic injury.

Low-voltage does not mean innocuous

Household / workplace voltage	High-voltage sources
Robbins cites typical supplies of 120 or 220 V.	Power lines and lightning are examples.
Wet contact can permit sufficient current for ventricular fibrillation.	Larger currents more often produce extensive burns.
Alternating current may prolong contact through tetany.	Medullary center paralysis is emphasized.

Why voltage alone cannot describe risk



When resistance is low, ordinary supply voltage may permit a dangerous current.

Three determinants of severity

Electrical exposure

Current strength (amperage)

Duration of flow

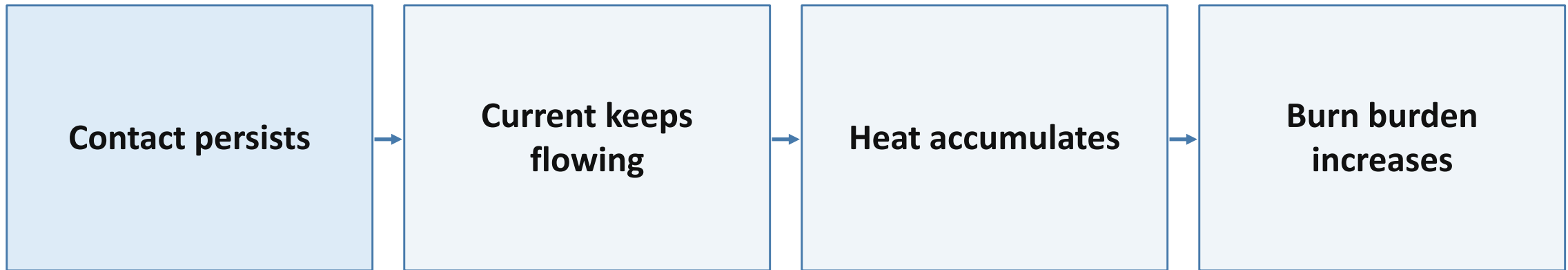
Path through the body

These jointly influence injury type and the severity and extent of burns.

Amperage: The magnitude of flowing current

- The strength of the electrical current is expressed as amperage.
- Current strength is a determinant of the resulting tissue damage.
- High-voltage sources can produce large current flows.
- A source voltage is not equivalent to the amount of current passing through tissue.

Duration compounds current-related injury

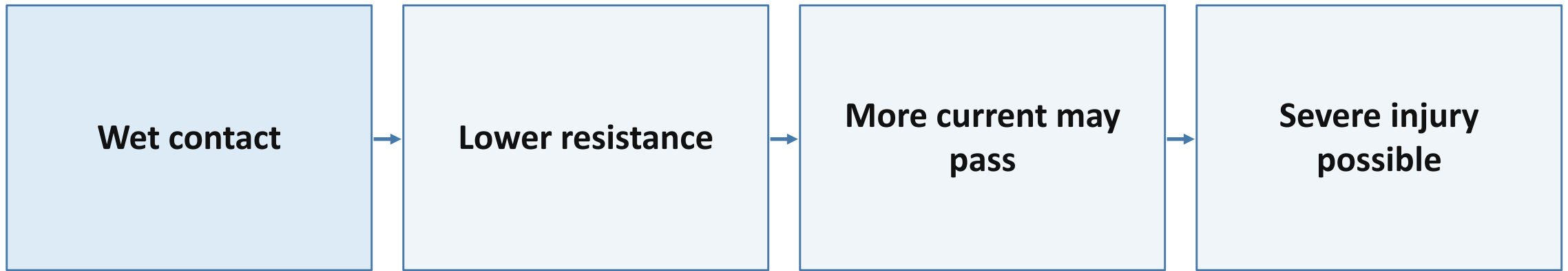


Robbins explicitly links sustained flow to generated heat and burns.

Current path determines exposed structures

- The path within the body as a determinant of injury.
- Entry and exit sites may be burned.
- Internal organs along a current route may also sustain burns.
- Clinical interpretation requires attention to affected tissues, not voltage alone.

Wet skin changes the hazard

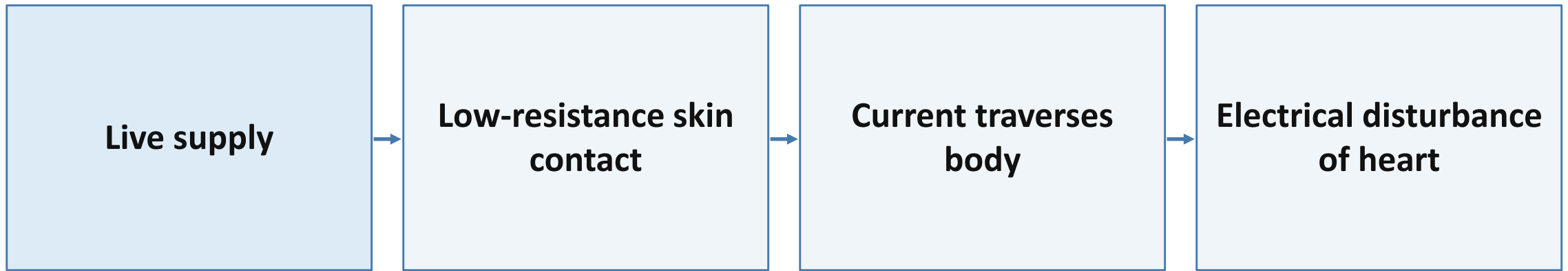


This pathway is the specific household-contact example given.

A 120- or 220-volt contact can be fatal

- Robbins explicitly describes these voltages in homes and workplaces.
- With low contact resistance, current may be sufficient for ventricular fibrillation.
- Visible charring is not a prerequisite in this electrical mechanism.
- Judging danger solely from nominal supply voltage is inadequate.

A domestic contact: From exposure to fibrillation

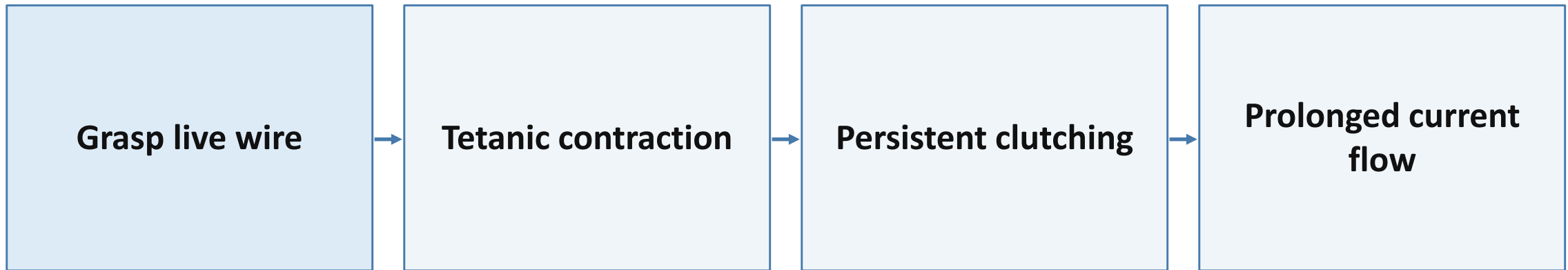


Conceptual sequence: The electrical endpoint includes ventricular fibrillation.

Alternating current changes the duration of contact

- Most domestic electrical supplies use alternating current (AC).
- AC may trigger tetanic skeletal-muscle contraction.
- Tetany can interfere with voluntary release of a grasped conductor.
- The duration of electrical exposure may therefore increase.

Alternating current: The grasp–tetany loop

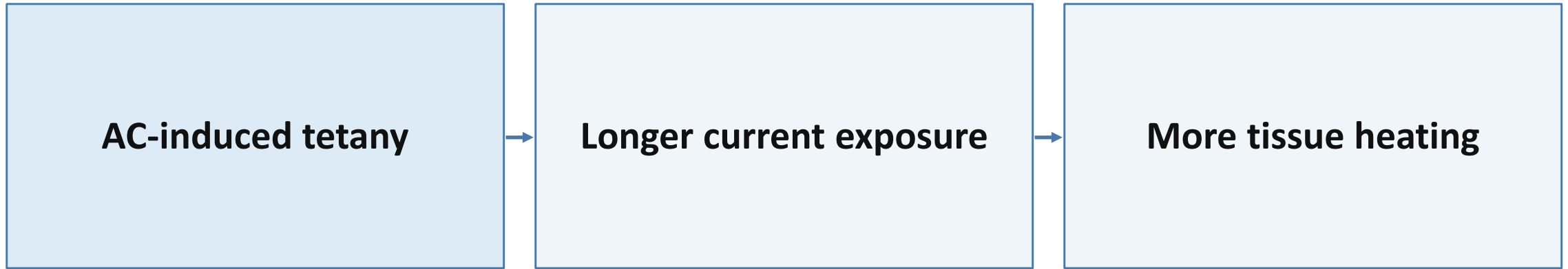


The source describes irreversible clutching as likely, not inevitable, when a live wire or switch is grasped.

Tetany is a mechanical amplifier of exposure

- The muscle contraction is itself an electrical effect.
- Loss of voluntary release extends the time of current passage.
- Sustained current can increase heat generation.
- The physiologic effect can therefore intensify the thermal injury.

One exposure produces two coupled pathways



Parallel: Chest-wall tetany can impair ventilation; Prolonged exposure increases burn risk.

Chest-wall muscle spasm can cause asphyxia

- Alternating current can induce sustained spasm of respiratory muscles.
- Impaired chest-wall movement can prevent effective breathing.
- Fatal asphyxia as a possible result.
- This mechanism differs from failure of central respiratory drive.

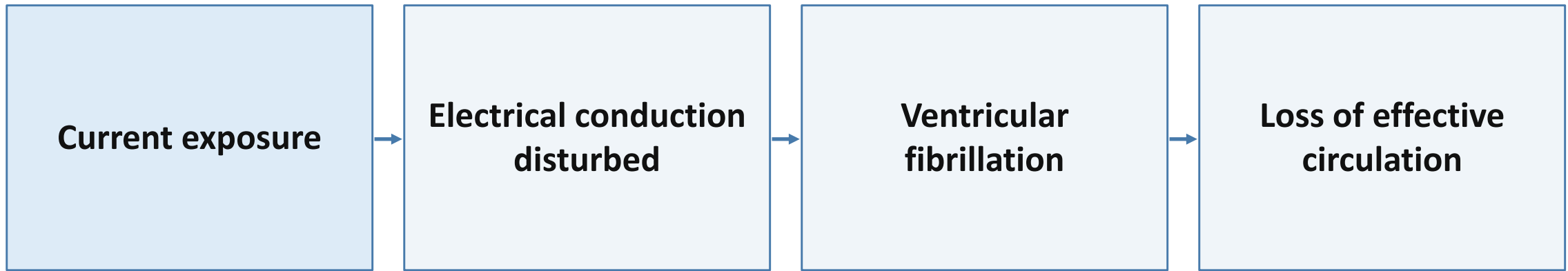
Two mechanisms of respiratory failure

Chest-wall tetany	Respiratory center failure
Peripheral muscular mechanism	Central conduction / control mechanism
AC-associated spasm restricts respiratory movement.	Electrical injury disrupts neural impulse conduction.
Asphyxia can be the terminal event.	Center failure can be the terminal event.

Impulse-conduction disruption causes functional injury

- Electrical injury need not act through thermal tissue destruction.
- Interference with nerve impulse conduction is a separate mechanism.
- Cardiac and respiratory centers may fail.
- This accounts for life-threatening physiology even when burns are not the dominant feature.

Pathway to ventricular fibrillation



Final box is physiologic interpretation of ventricular fibrillation, not quoted Robbins language.

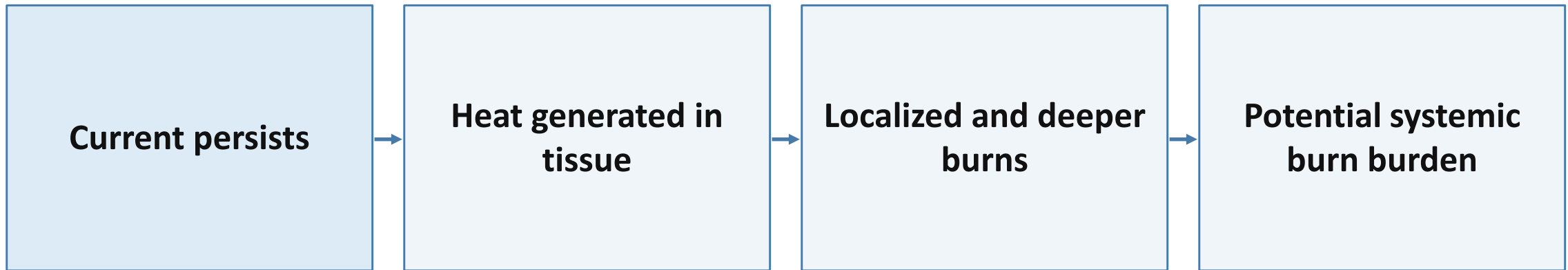
Ventricular fibrillation is an electrical endpoint

- Ventricular fibrillation among principal fatal events.
- Low-resistance contact at household voltage may provide enough current.
- Cardiac electrical failure must be distinguished from tissue heating.
- The outcome depends on the particular current exposure, not simply its category.

Immediate electrical events versus heat injury

Functional / conduction injury	Structural / thermal injury
Ventricular fibrillation	Entry and exit burns
Cardiac or respiratory center failure	Internal-organ burns
Chest-wall tetany and asphyxia	Extensive tissue destruction with sustained flow

Sustained current generates thermal injury

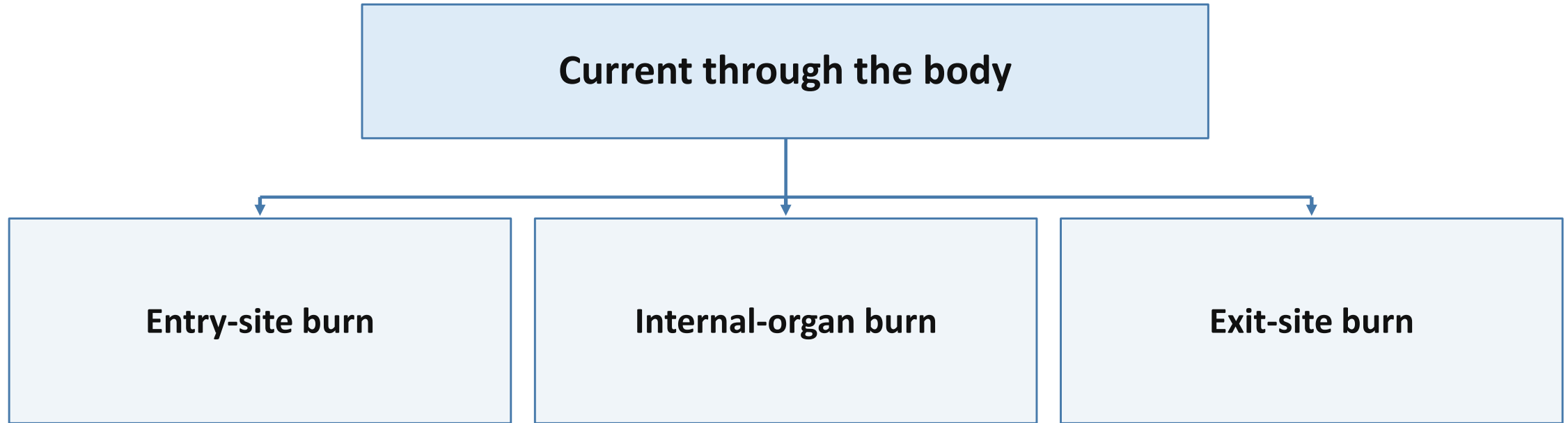


Last step represents possible effects of extensive burns, covered in adjacent thermal-burn text.

Electrical burns may involve entry and exit sites

- Burns at the sites where current enters and exits.
- The contact points represent only part of the possible injury distribution.
- Sustained current increases the potential for heating along the route.
- Do not infer a complete injury pattern from one skin contact mark.

A current route may injure more than skin



Schematic only: Actual paths vary; The textbook does not prescribe a fixed anatomical route.

Internal-organ burns are an essential possibility

- Electrical heating is not restricted to exposed epidermis.
- Robbins explicitly describes internal-organ burns with sustained flow.
- The current path becomes important when interpreting internal damage.
- External burn appearance cannot establish the absence of deeper injury.

Thermal and electrical injury: Overlap and distinction

Thermal-burn framework	Electrical-specific mechanisms
Describes depth and tissue necrosis.	Explains current path and contact conditions.
Explains edema, shock and infection in extensive burns.	Includes ventricular fibrillation and nerve conduction failure.
Useful when electrical injury causes major burns.	Cannot be replaced by burn grading alone.

High voltage often produces larger current flows

- High-voltage exposures as producing similar types of damage.
- Their large current flows make extensive burns more likely.
- Paralysis of medullary centers is also more likely.
- This is a tendency, not a claim that low-voltage events are harmless.

High-voltage exposure: Dominant hazards

High-voltage electrical exposure

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graph TD; A[High-voltage electrical exposure] --> B[Large current flows]; A --> C[Extensive burns]; A --> D[Medullary-center paralysis];
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Large current flows

Extensive burns

Medullary-center paralysis

Extensive burns and medullary paralysis are parallel possible effects, not a sequential cascade.

Medullary center paralysis

- Robbins specifically associates high-voltage sources with medullary center paralysis.
- The medulla contains vital autonomic control structures.
- Loss of central control can produce catastrophic cardiorespiratory failure.
- This is mechanistically different from a burn confined to contact skin.

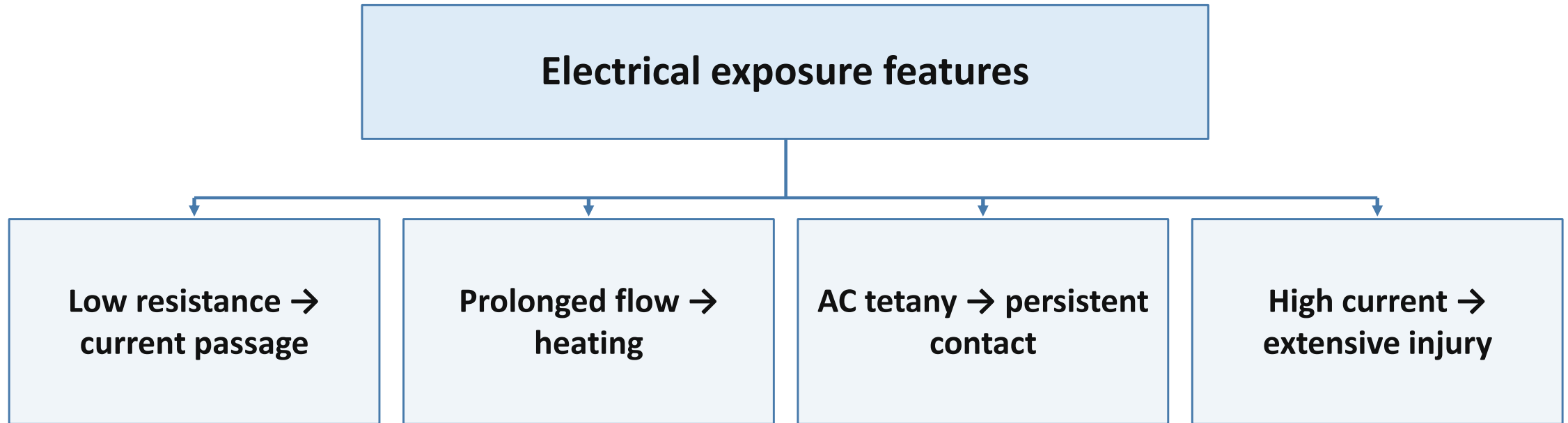
Electrical injury across exposure categories

Low-voltage exposure	High-voltage exposure
Household and workplace contact	Power lines and lightning
May cause ventricular fibrillation, especially with low skin resistance	More often causes extensive burns with large current flows
AC grasp tetany can prolong contact	Medullary paralysis more likely
Severe injury possible	Similar mechanisms, often greater tissue injury

Lightning as high-voltage exposure

- Lightning is a classic example of high-voltage electrical injury.
- Lightning within the same major electrical injury category.
- Potential endpoints remain burns and electrical disruption of excitable tissues.
- No lightning-specific lesion morphology is specified in the supplied excerpt.

Link determinants to their pathologic effects

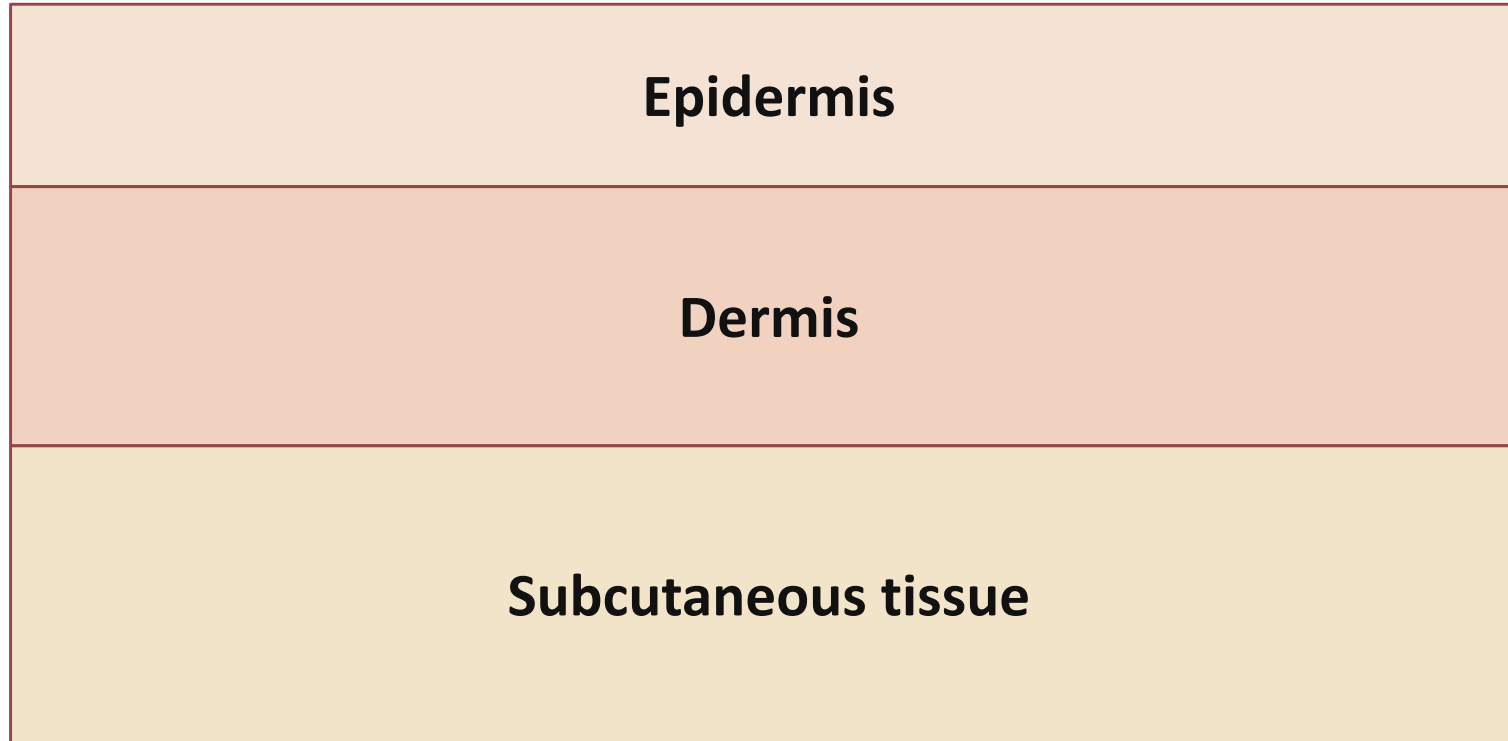


These arrows express source-supported causal relationships, not quantitative risk estimates.

Extensive electrical burns enter the burn-injury framework

- Tissue heating from sustained current can produce genuine burns.
- The adjacent Robbins thermal-burn section defines burn depth and complications.
- Those general consequences provide context when electrical burns are extensive.
- They do not replace assessment of the electrical mechanism itself.

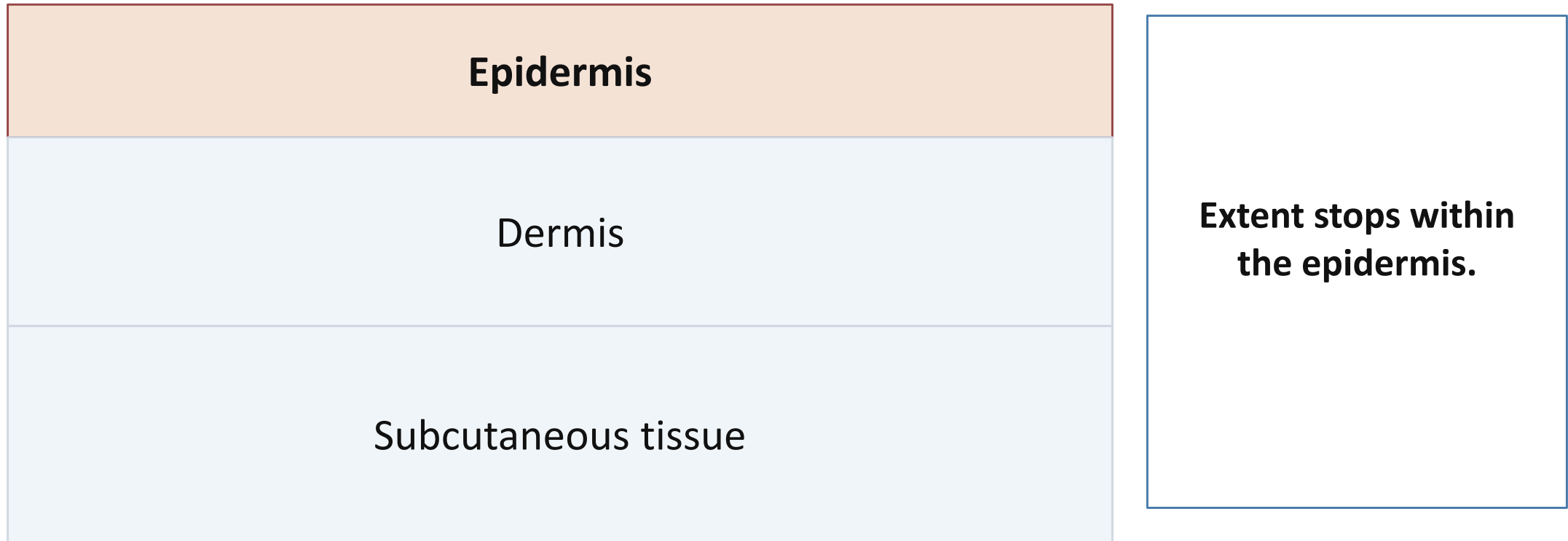
Burn depth is a tissue-anatomic classification



**Compare the
anatomic layer
reached by each burn
depth.**

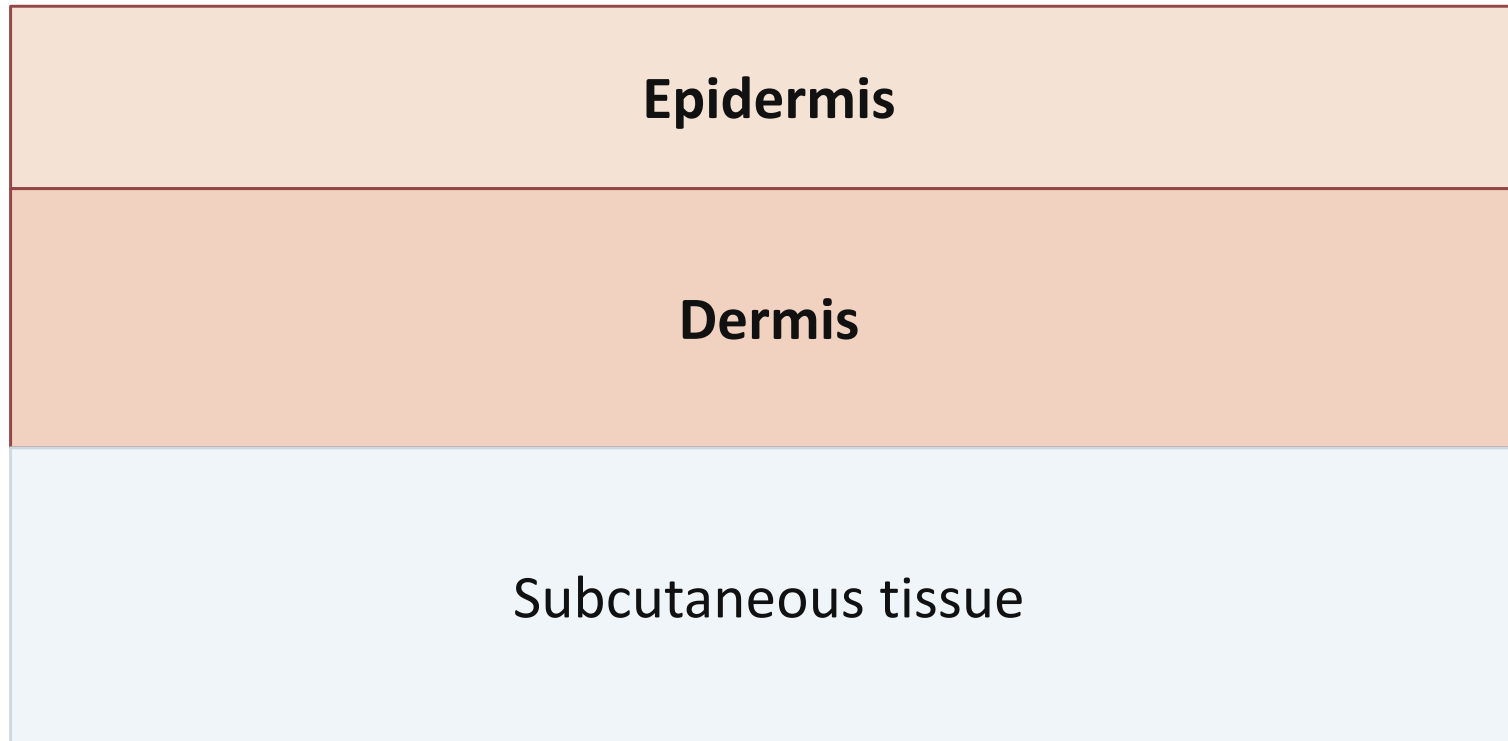
Superficial: Epidermis • Partial-thickness: Dermis • Full-thickness: Subcutaneous tissue

Superficial burn: Injury confined to epidermis



A superficial burn remains within the epidermis; Deeper layers are spared in this classification.

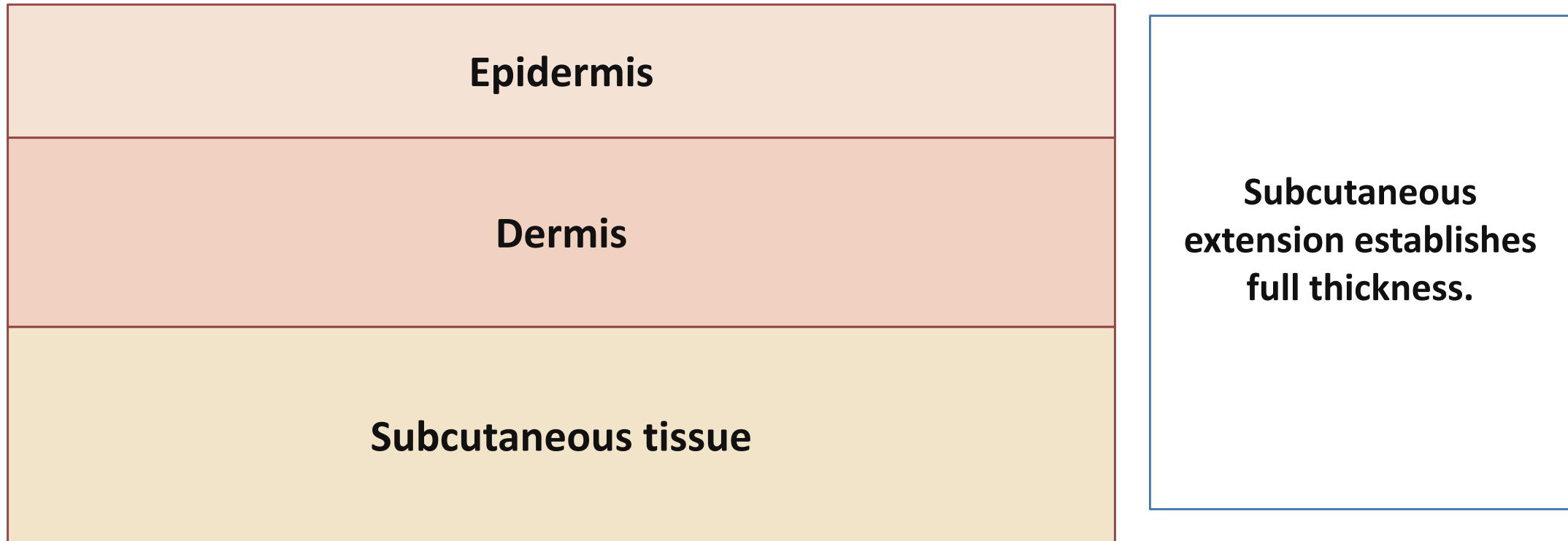
Partial-thickness burn: Dermis involved



**Dermal involvement
makes this a partial-
thickness injury.**

Partial-thickness injury extends into the dermis, exceeding a superficial epidermal burn.

Full-thickness burn: Into subcutaneous tissue



Full-thickness injury extends into subcutaneous tissue and may also involve underlying muscle.

Burn depth and gross appearance

Partial-thickness burn	Full-thickness burn
Pink or mottled appearance	White or charred appearance
Blisters may be present	Dry surface
Painful	Painless if cutaneous nerve endings destroyed
Features vary with depth	Deep tissue loss and possible muscle involvement

Burned tissue shows coagulative necrosis

- The adjacent thermal-burn morphology section describes coagulative necrosis in devitalized tissue.
- Tissue architecture can persist transiently despite cell death.
- Microscopy addresses structural heat damage—not ventricular fibrillation.
- This framework applies to thermal components of electrical injury.

At the burn edge: Necrosis and inflammation

Heat-damaged tissue interface

**Devitalized tissue: Coagulative
necrosis**

**Adjacent living tissue:
Leukocytes**

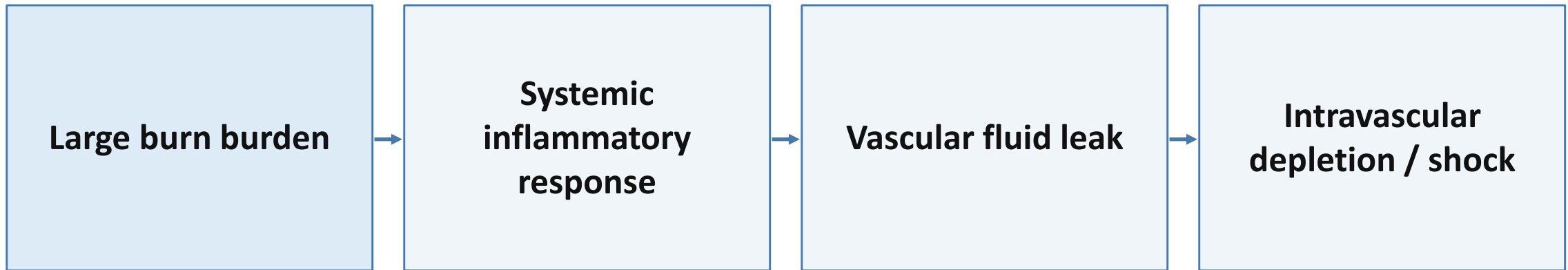
**Marked inflammatory
exudation**

Necrosis and adjacent inflammation are related morphologic findings, not distinct sequential stages.

Depth does not equal total clinical severity

- Burn depth is one component of severity.
- Robbins also identifies total body-surface involvement and associated internal injury.
- Fluid/electrolyte support and infection prevention influence outcomes of extensive burns.
- Electrical cardiopulmonary effects remain additional, distinct hazards.

Extensive burns can precipitate shock



General thermal-burn physiology; Not a claim that every electrical burn produces shock.

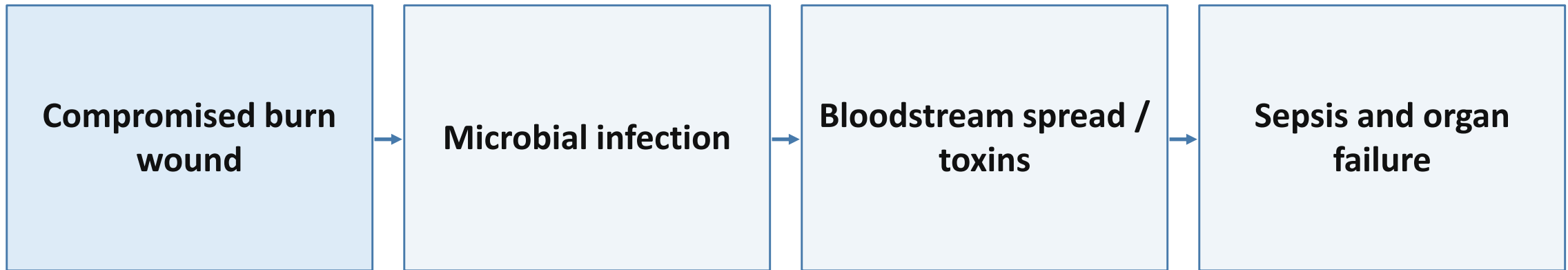
Vascular leak explains widespread edema

- Severe burns may produce a systemic inflammatory response.
- Fluid shifts to interstitial tissues through increasingly permeable vessels.
- Generalized edema can be prominent; Pulmonary edema may occur.
- This is a secondary injury mechanism in extensive burn patients.

Burn wounds provide a niche for microorganisms

- The burn surface contains proteinaceous exudate and tissue debris.
- Compromised local blood supply can weaken effective host responses.
- Colonization is common; Invasive infection can progress beyond the wound.
- Infection is a later hazard distinct from the initial electric exposure.

Burn infection can become a systemic problem



General thermal-burn section connects wound infection to pneumonia, shock, renal failure and ARDS.

Three major complications of extensive burns

Complication	Mechanistic association
Shock	Inflammation and systemic fluid leakage
Sepsis	Wound microbial invasion and dissemination
Respiratory insufficiency	Pulmonary edema, infection or associated airway injury

Major burns impose increased metabolic demands

- A hypermetabolic state after extensive burns.
- Heat loss and heightened energy requirements contribute to this response.
- Systemic burn care must therefore look beyond the local skin wound.
- This observation is not a specific biomarker of electrical injury.

Recovery may leave hypertrophic scars

- Hypertrophic scarring can follow major burn injuries.
- Excess collagen deposition characterizes the scar.
- Itching and scar symptoms can persist long after the exposure.
- This is a healing consequence, not a defining electrical diagnostic sign.

Immediate and later complications are not interchangeable

Immediate electrical hazard	Potential consequence of extensive burn
Ventricular fibrillation	Circulatory shock from vascular leak
Cardiac / respiratory center failure	Wound infection and sepsis
AC-associated chest-wall tetany	Hypertrophic scarring during recovery

A clinicopathologic interpretation checklist

- Identify the likely source: Domestic, workplace, power line or lightning.
- Consider contact resistance, duration, current strength and trajectory.
- Separate conduction-related outcomes from heat-mediated injury.
- For extensive burns, use depth, extent and systemic complications as additional context.

Case 1 — Wet-contact household exposure

Clinical scenario

An adult touches a household electrical fitting with wet hands. The source is a conventional domestic supply.

Why can this apparently low-voltage contact produce a fatal event?

- A. Wet skin raises electrical resistance.
- B. Lower skin resistance permits greater current passage.
- C. Only high-voltage sources can affect the heart.

Case 1 — Mechanistic resolution

B. Lower skin resistance permits dangerous current passage.

- Robbins explicitly uses wet skin as a low-resistance example.
- Domestic 120/220 V exposure can allow sufficient current for ventricular fibrillation.
- Electrical lethality is not determined from voltage label alone.

Case 2 — Grasping a live conductor

Clinical scenario

A worker grasps a live AC conductor. The hand remains tightly clenched around it despite an attempt to release.

Which sequence best accounts for increasing tissue injury?

A. Tetany → sustained contact → longer current flow → heating

B. Thermal necrosis → lower voltage → no further exposure

C. Medullary paralysis → reduced current through the limb

Case 2 — Mechanistic resolution

A. Tetany extends electrical contact.

- Alternating current can cause sustained muscle spasm.
- Persistent clutching prolongs the period of current flow.
- Longer exposure increases the opportunity for extensive burns.

Case 3 — High-voltage exposure

Clinical scenario

A person contacts a high-voltage power source and develops extensive tissue injury together with abrupt cardiorespiratory dysfunction.

Which pattern is most consistent with Robbins?

- A. High voltage produces burns but never affects neural centers.
- B. High current flows favor extensive burns and medullary paralysis.
- C. Current trajectory and exposure duration have no role.

Case 3 — Mechanistic resolution

B. High current flow can injure tissue and vital centers.

- High-voltage sources can generate large current flows.
- Extensive burns are more likely.
- Paralysis of medullary centers is an additional recognized hazard.

Case 4 — Deep burn and systemic deterioration

Clinical scenario

A survivor of electrical exposure has sustained large, deep burns. Over the subsequent course, generalized edema and systemic infection become concerns.

Which mechanistic framework is appropriate?

A. Only electrical arrhythmias are relevant after exposure.

B. Major-burn inflammatory leak and infection pathways may apply.

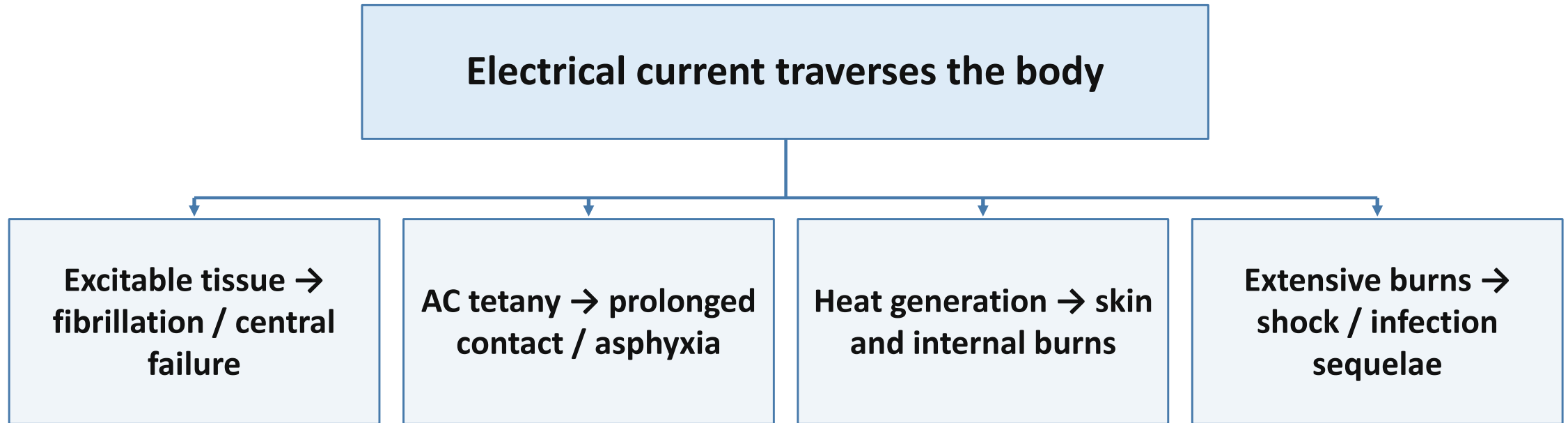
C. Tissue depth provides no useful pathologic information.

Case 4 — Mechanistic resolution

B. Extensive burns have important downstream pathophysiology.

- Systemic inflammatory vascular leak can contribute to edema and shock.
- Devitalized burn wounds are vulnerable to infection and sepsis.
- Electrical conduction-related complications must still be considered independently.

One integrated map of electrical injury



All branches are potentially relevant; Their relative prominence depends on exposure.

Essential clinicopathologic conclusions

- Electrical injury operates through both heat generation and disruption of impulse conduction.
- Amperage, exposure duration and current path are central determinants.
- Wet contact and AC grasp tetany explain why household exposures can be lethal.
- High-voltage currents favor extensive burns and medullary-center injury.
- Extensive electrical burns can share the complications of major thermal burns.

Reference and source boundary

Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th edition (2026).

- Electrical Injury (principal section)
- Injury by Physical Agents (parent context)
- Thermal Burns (adjacent general burn pathology)