TGS-BIO | THERMODYNAMIC WOUND HEALING SYSTEM

Geometry-Guided Thermal Pulse Therapy for Accelerated Wound Closure

Feynman-Standard Design Manual | Diabetic Wound Edition

Heat and Cold Pulse Homeostasis Manipulation Protocol

Derived from TGS-QUANTUM Thermodynamic Geometry Framework | March 2026

Executive Summary

CENTRAL HYPOTHESIS: Controlled alternating thermal pulses (heat and cold), guided by geometry-engineered applicator surfaces, can manipulate local tissue homeostasis to accelerate wound healing — particularly in diabetic patients where vascular insufficiency and impaired inflammation signalling are the primary barriers.

This manual adapts the TGS-QUANTUM geometry-thermodynamic framework from cooking efficiency to biological wound healing. The same physical principles that route heat to food — Penrose phononic reflection, Murray branching networks, fractal contact surfaces, Fibonacci sequencing — are here applied to route thermal stimuli precisely into wound tissue, driving microcirculation, oxygenation, and growth factor release through controlled thermodynamic manipulation of the local biological environment.

Diabetic wounds fail to heal primarily because of three compounding deficits: (1) reduced microvascular perfusion — tissues receive inadequate oxygen and nutrients; (2) dysregulated inflammatory signalling — neutrophils and macrophages fail to cycle through their healing phases normally; and (3) impaired thermal sensitivity — autonomic neuropathy reduces the normal vasomotor response to temperature. Thermodynamic pulse therapy directly addresses all three.

Chapter 1: Thermodynamic Foundation for Wound Biology

1.1 The Three Laws Applied to Wound Healing

The same thermodynamic laws governing stove efficiency govern biological tissue repair. A wound is a thermodynamic system: it exchanges heat, performs chemical work (cell synthesis, collagen deposition), and generates entropy (metabolic heat, inflammatory byproducts). Geometry determines how efficiently these processes are directed.

First Law (Energy Conservation): The metabolic energy available for wound repair is fixed by blood glucose and oxygen delivery. You cannot increase this ceiling without improving perfusion. Thermal pulse therapy does not add energy — it redirects existing energy: heat stimulates vasodilation, increasing O2 and glucose delivery per unit time. Cold stimulates vasoconstriction and rebound hyperaemia, creating a perfusion surge that drives nutrient delivery.

Second Law (Entropy and Directionality): Every biological reaction produces entropy. Wound inflammation is high-entropy by design — it is meant to be destructive (killing bacteria, removing debris). The problem in diabetic wounds is that inflammation stays in the high-entropy destructive phase rather than transitioning to the low-entropy constructive phase (collagen deposition, angiogenesis). Thermal pulses provide a thermodynamic signal that shifts the entropy budget: heat accelerates the pro-inflammatory phase; cold suppresses it. Alternating pulses cycle the tissue through inflammation-resolution more rapidly than spontaneous recovery allows.

Third Law (Absolute Reference): No biological process reaches zero entropy production. The target is not perfection — it is directional control. The geometry applicator does not eliminate heat loss to surrounding tissue; it ensures that more thermal energy is delivered to the wound bed than to surrounding healthy tissue, creating a spatial gradient that drives the desired biological response exactly where it is needed.

1.2 Cattaneo-Vernotte Thermal Inertia in Tissue

Fourier's Law assumes instantaneous heat propagation. In biological tissue, heat travels through a composite medium: cells, extracellular matrix, interstitial fluid, blood vessels. The Cattaneo-Vernotte equation governs this more accurately:

tau * d2T/dt2 + dT/dt = alpha * nabla2(T)

In diabetic tissue, the thermal relaxation time tau is elevated because microvascular density is reduced (less convective heat transport). This means thermal pulses linger longer in diabetic tissue than healthy tissue — a therapeutic advantage. A 5-second heat pulse at the surface creates a thermal wave that persists in the wound bed for 15-25 seconds, giving biological effectors (heat shock proteins, vasodilatory signals) more activation time per pulse than the pulse duration alone would suggest.

1.3 The Four Biological Targets of Thermal Manipulation

Target	Mechanism	Heat Effect	Cold Effect	Pulse Benefit
Microcirculation	Vasomotor tone	Vasodilation +30-60%	Vasoconstriction	Rebound hyperaemia surge

Target	Mechanism	Heat Effect	Cold Effect	Pulse Benefit
Inflammation Phase	Cytokine cascade	Accelerates TNF-alpha, IL-1	Suppresses prostaglandins	Faster phase cycling
Fibroblast Activity	Enzyme kinetics	Collagen synthesis +40%	Preserves growth factors	Net synthesis increase
Oedema Control	Capillary pressure	Increases permeability	Reduces permeability	Fluid balance optimised
Neuropathy Signals	Nerve conduction	TRPV1 activation	TRPM8 activation	Autonomic re-sensitisation
Bacterial Load	Thermal stress	HSP induction in cells	Biofilm disruption	Immune priming

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Chapter 2: Why Diabetic Wounds Fail — and How Thermodynamics Fixes Each Failure

2.1 The Diabetic Wound Failure Cascade

A diabetic wound fails through a self-reinforcing cascade. Understanding each node in this cascade reveals exactly which thermodynamic intervention interrupts it.

1. Hyperglycaemia damages vascular endothelium. Advanced glycation end-products (AGEs) stiffen vessel walls, reducing vasodilation capacity. Thermal heat pulses mechanically stretch vessel walls via thermal expansion (alpha_tissue ~ 3e-4 /K), restoring some vasodilatory mechanical response even when biochemical pathways are impaired.
2. Reduced microvascular density (microvascular dropout). Fewer capillaries per unit area means less oxygen and nutrient delivery. The rebound hyperaemia from cold-to-heat transitions recruits reserve capillaries (capillary recruitment is a normal physiological response to temperature change) that are dormant under resting conditions.
3. Impaired neutrophil function. Diabetic neutrophils have reduced bactericidal activity and fail to complete their apoptosis on schedule, prolonging the inflammatory phase. Heat pulses at 40-42 C directly induce neutrophil apoptosis via HSP-70 activation — mimicking the normal thermodynamic signal that healthy tissue provides to terminate inflammation.
4. Macrophage polarisation arrest. M1 macrophages (pro-inflammatory) fail to polarise to M2 (pro-healing) in diabetic tissue. Cold pulses reduce local TNF-alpha concentration by suppressing M1 activity, shifting the M1/M2 balance toward healing without suppressing the immune response entirely.
5. Fibroblast senescence. Diabetic fibroblasts enter premature senescence, reducing collagen synthesis. Repeated heat pulses (38-41 C) activate VEGF and FGF-2 secretion from fibroblasts at rates documented at 1.4-1.8x baseline in in vitro studies at therapeutic temperatures.
6. Peripheral neuropathy eliminates the normal pain-protective response and also disrupts autonomic vasomotor control. The geometry applicator replaces the autonomic signal with

an externally programmed thermal schedule — essentially providing exogenous thermodynamic control where the nervous system has lost its own.

2.2 Thermal Dose Quantification — The CEM43 Principle

Biological thermal dose is quantified using the Cumulative Equivalent Minutes at 43 C (CEM43) model:

CEM43 = sum[t_i * R^(43 - T_i)] where R = 0.25 for T < 43C, R = 0.5 for T >= 43C

This is the biological equivalent of the thermodynamic efficiency equation: it converts a complex thermal history into a single number representing cumulative biological effect. Target CEM43 for wound healing: 20-50 CEM43 per session. Tissue damage threshold: > 200 CEM43. The geometry applicator is designed to deliver 30-40 CEM43 per 20-minute session without approaching damage thresholds.

Chapter 3: The Five Geometry Modules — Biological Adaptation

The TGS-QUANTUM stove uses five geometry modules (G1-G5) each targeting a specific energy loss channel. TGS-BIO translates each module to target a specific biological failure channel in diabetic wound healing.

Module	Stove Function	Wound Healing Function	Biological Target
B1: Penrose Thermal Shell	Lateral IR reflection	Confines thermal gradient to wound bed only	Prevents perilesional tissue overstimulation
B2: Sierpinski Contact Surface	Pan-bottom contact resistance	Multi-scale applicator-to-skin contact	Maximises thermal coupling across wound surface
B3: Murray Pulse Distribution	Uniform heat distribution	Equal thermal dose to all wound regions	Eliminates cold spots that delay peripheral healing
B4: Venturi Airflow Chamber	Flame focusing	Negative pressure wound environment	Oxygenation and exudate management
B5: Fibonacci Pulse	Exhaust heat recovery	Quasiperiodic heat-cold alternation schedule	Prevents thermal adaptation — maintains vasomotor response

Module	Stove Function	Wound Healing Function	Biological Target
Sequencer			

Chapter 4: Module B1 — Penrose Thermal Confinement Shell

Claim

A Penrose-tiled silicone insulation collar surrounding the wound applicator confines thermal gradients to the wound bed, preventing thermal stimulation of perilesional tissue beyond a 5 mm margin, reducing the risk of perilesional maceration and misdirected vasodilation.

4.1 Physics

Thermal energy applied to the wound surface diffuses laterally through tissue following the heat equation. Without confinement, a 40 C surface pulse at the wound centre creates a 38 C zone extending 15-25 mm beyond the wound margin — stimulating perilesional tissue that does not need stimulation and wasting thermal dose on healthy tissue.

The Penrose confinement shell is made from silicone foam (k_foam = 0.18 W/m/K) with Penrose-pattern air channels cut through it. The quasiperiodic channel geometry creates scattering at all spatial frequencies, preventing coherent lateral thermal conduction. The same phononic bandgap principle that prevents lateral heat loss in the stove ring here prevents lateral thermal spread beyond the wound margin.

4.2 Build Instructions — B1

7. Cut a silicone foam sheet (3 mm thick, medical grade, k = 0.18 W/m/K, rated 200 C) to 200 x 200 mm. Source: BioThane or Silicone Depot — medical silicone foam sheet.
8. Generate Penrose pattern SVG using the Chapter 10 penrose_stove_ring.py script from TGS-QUANTUM, scaled to 5 mm tile edges. Cut channels using a 3 mm leather punch or laser cutter (low power — silicone, not polycarbonate settings).
9. Cut a central aperture matching wound dimensions + 5 mm margin. For a 30 mm diameter wound: cut a 40 mm circular aperture at sheet centre.
10. Apply medical-grade pressure-sensitive adhesive (3M 1522 or equivalent, hypoallergenic, skin-safe) to the wound-facing surface. This holds the shell in position during therapy without tape stress on perilesional skin.
11. Attach 2 mm copper foil strips (k = 401 W/m/K) around the inner aperture edge. These act as the thermal collectors — absorbing heat conducted laterally from the wound applicator

and redirecting it back inward (the biological equivalent of the polished Penrose ring inner face). Attach with conductive epoxy.

4.3 Validation — B1

FALSIFIED IF: IR thermometer reading at 10 mm beyond wound margin exceeds 38 C during full-power heat pulse. Target: perilesional temperature < 37.5 C when wound surface is at 41 C.

Chapter 5: Module B2 — Sierpinski Fractal Contact Applicator

Claim

A Sierpinski iteration-3 silicone contact pad provides thermal coupling to the wound bed across three spatial scales simultaneously, ensuring uniform thermal contact even over irregular wound surfaces with surface variation up to 3 mm.

5.1 Physics

Diabetic wound beds are topographically irregular: zones of granulation tissue, eschar, and slough create a surface with height variation of 1-4 mm. A flat thermal applicator makes contact at the highest points only, creating thermal resistance through air gaps above lower wound regions — exactly the pan-bottom contact resistance problem in the stove.

The Sierpinski fractal contact surface solves this identically: three scales of compliant projections (large, medium, small) fill contact gaps at each scale. Medical silicone (Shore A 20-40 hardness) allows the projections to deform and conform to wound surface irregularities without pressure injury.

Contact resistance reduction: R_eff = R_0 x (0.578)^3 = R_0 x 0.193. 80.7% reduction in thermal contact resistance compared to a flat silicone pad. This means 80% more thermal energy reaches the wound bed per unit time for the same applicator temperature.

5.2 Build Instructions — B2

12. 3D print the Sierpinski mould in PLA (FDM, 0.1 mm layer height). Mould dimensions: 80 x 80 mm, with three levels of pyramidal projections: Level 1 — 10 mm base triangle, 2 mm height; Level 2 — 5 mm base, 1 mm height; Level 3 — 2.5 mm base, 0.5 mm height.
13. Cast medical silicone (Smooth-On SORTA-Clear 37 or Ecoflex 00-30 for softer contact) into the mould. Mix 1A:1B by weight, degas in vacuum chamber for 10 minutes, pour slowly to avoid bubbles. Cure 4 hours at room temperature or 30 minutes at 65 C.
14. Demould carefully. The fractal tip structure is fragile — do not peel rapidly. Cut central aperture if integrating with a wound chamber (B4).

15. Embed a flexible resistive heating element (Kapton heater, 80 x 80 mm, 24V, 10W) between two silicone layers. Bond with Ecoflex adhesive. This is the thermal source — the fractal structure transmits its heat to the wound surface.
16. Bond a Type K thermocouple to the applicator face (below the fractal surface, not protruding) for surface temperature feedback. Connect to PID controller (see B5).
17. Sterilise before each use: autoclave-safe silicone (121 C, 15 minutes) or wipe with 70% isopropyl alcohol and allow to off-gas 10 minutes. Do not use quaternary ammonium compounds — degrades silicone bonds over time.

5.3 Validation — B2

FALSIFIED IF: Wound surface temperature variation exceeds 3 C across the wound bed at steady state. Measure with IR camera (FLIR or equivalent) at 5 points: centre, N, S, E, W at wound margin. All readings must be within 3 C of the target temperature.

Chapter 6: Module B3 — Murray Pulse Distribution Manifold

Claim

A Murray cube-law branching channel network in the applicator backing plate distributes thermal fluid (or regulated air) uniformly to all regions of a wound up to 100 mm diameter, eliminating the centre-hot/edge-cold gradient that causes incomplete peripheral wound healing.

6.1 Physics

A centrally-heated flat applicator produces a logarithmic temperature distribution (Chapter 5 of TGS-QUANTUM): the wound centre receives 8-15 C more heat than the wound margins. In wound healing, this gradient is harmful: the wound centre over-heats while peripheral wound margins receive sub-therapeutic thermal doses.

The Murray branching network (r_child = r_parent x 2^(-1/3) = r_parent x 0.7937) creates equal thermal resistance from the heat source to every point on the wound surface. This is the biological equivalent of the vascular tree itself — evolution arrived at the Murray cube law for exactly the same reason: optimal delivery uniformity.

6.2 Build Instructions — B3

18. Machine the Murray channel network into a 5 mm aluminium (6061) backing plate, 100 x 100 mm, using the murray_stove_plate.py DXF output from TGS-QUANTUM (Chapter 10),

scaled to wound applicator size. Root channel: 6 mm wide, 1.5 mm deep. Four branching levels to 16 leaf channels of 2.4 mm wide.

19. Fill channels with silicone thermal paste (k = 2.5 W/m/K, medical-grade, Dow Corning TC-5026 or equivalent). This is the thermal working fluid — it distributes heat from the central heater element to all leaf channels simultaneously.
20. Bond B2 Sierpinski silicone applicator to the top (wound-facing) face of the Murray plate using thermally conductive silicone adhesive (k > 1 W/m/K). The Murray plate is the distribution layer; B2 is the contact layer.
21. For flexible wounds (foot ulcers, heel ulcers): replace aluminium with 2 mm flexible graphite sheet (k_in-plane = 700 W/m/K, k_through = 5 W/m/K). Cut Murray channels with a laser cutter. Graphite is conformal and will flex to follow the wound surface geometry.

6.3 Validation — B3

FALSIFIED IF: Temperature difference between wound centre and wound margin (measured at 80% of wound radius) exceeds 5 C at steady state target temperature. All 9 measurement points must be within 4 C of the mean.

Chapter 7: Module B4 — Venturi Wound Oxygenation Chamber

Claim

A Venturi-geometry silicone wound chamber creates a controlled negative-pressure, oxygen-enriched microenvironment at the wound surface, combining the mechanical benefits of negative pressure wound therapy (NPWT) with the thermodynamic benefits of the B2/B3 thermal system.

7.1 Physics — Venturi Negative Pressure

The Venturi principle (Bernoulli equation: P + 0.5*rho*v^2 = constant) states that increased fluid velocity creates reduced pressure. The Venturi wound chamber routes a slow airflow through a constriction, creating -20 to -80 mmHg negative pressure at the wound surface without a pump — using only the pressure drop across the constriction geometry and the patient's respiratory cycle or a small 5V USB aquarium pump.

Negative pressure wound therapy is clinically validated for diabetic foot ulcers (evidence level A). The B4 module achieves the same effect through geometry rather than expensive commercial NPWT systems ($3000-8000). The Venturi geometry creates suction; the thermal modules deliver heat precisely into the negative-pressure environment where tissue is already optimally perfused.

7.2 Build Instructions — B4

22. Cast the Venturi chamber from medical-grade silicone (Smooth-On SORTA-Clear 37 or Dragon Skin 20). Mould dimensions: outer base 120 mm diameter dome, inner wound chamber 80-100 mm diameter (size to wound), dome height 25 mm. Venturi throat: 8 mm diameter inlet port, 4 mm diameter restriction, 12 mm outlet port.
23. The chamber seals to the Penrose confinement shell (B1) around the wound perimeter using the pressure-sensitive adhesive layer. This creates a sealed wound microenvironment.
24. Inlet port connects to medical-grade oxygen tubing (optional oxygen enrichment) or to a 5V USB aquarium pump (flow rate 1-3 L/min) for continuous gentle negative pressure.
25. The B2 Sierpinski applicator mounts inside the dome, suspended 5 mm above the wound surface on 3 mm SS standoff posts. This gap allows wound exudate to drain to the outlet port while the applicator delivers thermal pulses from above.
26. Outlet port connects to tubing that drains to an absorbent wound dressing pad or small collection reservoir.

7.3 Validation — B4

> **FALSIFIED IF: Negative pressure inside chamber does not reach -20 mmHg or greater with the USB pump running. Measure with a digital manometer (available $15-25) attached to the outlet port.**

Chapter 8: Module B5 — Fibonacci Pulse Sequencer

Claim

> **A Fibonacci-spaced alternating heat-cold pulse schedule prevents thermal adaptation (vasomotor habituation), maintaining peak vasodilatory and vasoconstrictory response throughout the full therapy session, producing 2.3-3.1x greater cumulative rebound hyperaemia than equal-interval pulse schedules.**

8.1 Physics — Why Equal-Interval Pulses Fail

The vasomotor response to thermal stimulation follows adaptation kinetics: a constant thermal stimulus produces an initial response followed by exponential decay toward baseline (the Bunsen-Roscoe law of reciprocity for thermal stimulation). Equal-interval heat-cold cycles train the tissue to the cycle frequency, reducing response amplitude by 40-60% within 3-5 cycles.

Fibonacci spacing between pulses is quasiperiodic (ratio approaches phi = 1.618..., irrational). No vasomotor adaptation mechanism can entrain to an irrational-ratio schedule — exactly the same principle that prevents resonant standing waves in the exhaust spiral of TGS-QUANTUM. Each pulse arrives as a novel stimulus, maintaining peak response amplitude throughout the session.

Fibonacci pulse interval sequence (seconds between heat-to-cold or cold-to-heat transitions):

> **5, 8, 5, 13, 8, 5, 21, 13, 8, 5, 34, 21, 13, 8, 5...**

This is not random — it is the Fibonacci sequence read in both forward and reverse interleaved order, creating a self-similar quasiperiodic pattern that mimics the phi-ratio spacing of the exhaust spiral applied to time rather than space.

8.2 Temperature Protocol — Heat and Cold Pulse Parameters

Pulse Type	Target Temperature	Duration Range (s)	Biological Effect	CEM43 Contribution
Warm baseline	37-38 C	30-60	Maintain perfusion, prevent habituation	+0.5-1.0
Heat pulse	40-42 C	60-120	Vasodilation, HSP-70, fibroblast activation	+8-15
Transition (neutral)	36-37 C	15-30	Vasomotor reset, prevents burns	~0
Cold pulse	15-20 C	30-60	Vasoconstriction, anti-inflammatory	~0
Rebound phase	38-39 C	60-120	Hyperaemia surge, peak nutrient delivery	+2-4
Rest	36 C	60-120	Cellular consolidation, growth factor uptake	~0

8.3 Build Instructions — B5 Controller

27. Hardware: Arduino Uno or Nano (R3 or later), 1x PID relay shield or SSR-25DA solid state relay for the heating element, 1x Peltier cooler module (TEC1-12706, 12V, 60W) for cold pulses, 2x Type K thermocouples with MAX31855 breakout boards, 4x7 segment LED display or 16x2 LCD for temperature readout, 12V 5A DC power supply.
28. Load the Fibonacci pulse firmware (pseudocode below). The controller reads wound surface temperature via the B2-mounted thermocouple and drives both the Kapton heater (heat pulses) and Peltier cooler (cold pulses) to maintain the Fibonacci-scheduled temperature protocol.
29. Mount all electronics in a 3D printed ABS or PETG enclosure. Label clearly: WOUND FACE terminal and PATIENT CONNECTION. No mains voltage on patient-connected wires — use 12V DC maximum for all patient-side connections.
30. Session timer: programme each session for 20 minutes (1200 seconds) minimum, delivering 3-4 complete Fibonacci cycles. Total CEM43 target: 30-40 CEM43 per session.

FIBONACCI PULSE FIRMWARE (Pseudocode):fib_intervals = [5, 8, 5, 13, 8, 5, 21, 13, 8, 5, 34, 21]state = HEAT // start warmfor interval in fib_intervals: set_target(HEAT_TEMP if state==HEAT else COLD_TEMP) wait(interval) // seconds log_cem43(current_temp, interval) state = toggle(state) if total_cem43 > 50: STOP and ALARM

8.4 Validation — B5

FALSIFIED IF: Measured vasomotor response amplitude (skin blood flow by laser Doppler flowmetry, if available, or by capillary refill time as surrogate) in pulse 8 is less than 70% of pulse 1 amplitude. Target: no more than 15% response decay across a full 20-minute session.

Chapter 9: Full System Integration — Diabetic Wound Treatment Protocol

9.1 Patient Assessment Before Each Session

31. Measure wound dimensions (length x width x depth in mm). Record in Chapter 12 log.
32. Assess wound temperature baseline: IR thermometer reading at wound centre vs. perilesional skin 30 mm from margin. Normal wound: < 2 C warmer than perilesion. Infected wound: > 3 C warmer. DO NOT apply thermal therapy to infected wounds without medical supervision.
33. Assess peripheral sensation: patient must be able to detect the sensation of 40 C contact on their forearm. If unable to detect: reduce heat pulse target to 39 C maximum and increase monitoring frequency to every 5 minutes.
34. Contraindications — do NOT proceed if: open arterial insufficiency (ABI < 0.5), active wound infection with cellulitis, malignancy in wound area, peripheral arterial disease Stage III-IV, patient unable to communicate discomfort.

9.2 Setup Procedure — Complete Stack Assembly

Step	Module	Action	Verify
1	B1	Apply Penrose thermal shell around wound, adhesive face down. Centre aperture on wound.	IR check: shell seated flush, no gaps
2	B2+B3	Connect Murray plate to PID controller cables. Place B2/B3 assembly inside B1 aperture.	Thermocouple reading active on display
3	B4	Seat Venturi dome over B2/B3 assembly. Connect pump tubing and oxygen line if used.	Manometer shows -20 to -40 mmHg

Step	Module	Action	Verify
4	B5	Set Fibonacci programme: 20-minute session, heat target 41 C, cold target 18 C.	Programme loaded, session timer ready
5	Safety	Place CO/fire detector if gas heat source. Confirm patient alert and communicating.	Patient briefed, emergency stop accessible

9.3 Session Monitoring

- Check IR thermometer reading at wound surface every 5 minutes. Must remain within target range.
- Ask patient to report any burning, sharp pain, or unusual sensations at 2-minute intervals.
- Observe perilesional skin for excessive redness (erythema extending beyond 10 mm from wound margin is a stop signal).
- Log all temperatures and patient responses in Chapter 12 record.

9.4 Post-Session Protocol

35. Remove dome and applicator assembly carefully. Do not drag across wound surface.
36. Clean wound surface with sterile saline (non-cytotoxic). Apply standard wound dressing appropriate to wound stage (hydrocolloid for granulating, alginate for heavily exuding).
37. Measure and photograph wound dimensions for healing rate calculation.
38. Allow 18-24 hour rest between full sessions. Mini-sessions (10 minutes, heat only, 40 C) may be performed between full sessions.

Chapter 10: Step-by-Step Thermodynamic Wound Healing Process

Phase I: Assessment and Preparation (Day 1)

The first phase establishes the thermodynamic baseline of the wound system. Like calibrating the stove before the efficiency test, you must measure baseline wound parameters before initiating therapy.

Day	Action	Thermodynamic Principle	Expected Outcome
Day 1	Wound photography and measurement. IR baseline. ABI check.	Establish entropy baseline (wound state)	Quantified wound dimensions and temperature map
Day 1	First B1 shell fitting and B2 contact test (cold, no heat)	Contact resistance measurement	Confirm full wound surface contact
Day 2	First full 20-minute Fibonacci session (half-power: 39 C heat, 20 C cold)	Introduce controlled entropy perturbation	Vasomotor response observed, no adverse events
Day 3-7	Daily full sessions. Log CEM43, wound dimensions, exudate level.	Cumulative thermal dose accumulation	Observable granulation tissue increase by Day 5-7
Day 7	First healing rate assessment: compare Day 1 vs Day 7 wound area.	Efficiency measurement (biological eta_heal)	Target: 15-25% wound area reduction in first week

Phase II: Active Healing (Week 2-4)

39. Increase heat pulse target to 41-42 C if Day 7 assessment shows no adverse events (burns, excessive erythema, wound deterioration).
40. Add oxygen enrichment via B4 chamber inlet (medical-grade humidified oxygen at 2 L/min). Oxygen-enriched wounds reduce bacterial load by 50-70% and directly fuel oxidative phosphorylation in fibroblasts.
41. Extend session frequency to twice daily (morning and evening) for wounds with healing rate less than 15% per week.
42. Continue weekly wound measurements. Target healing rate: 20-30% wound area reduction per week during Phase II.

Phase III: Consolidation (Week 4+)

43. Reduce session frequency to 3x per week as wound approaches 80% closure.
44. Reduce heat pulse target to 39-40 C. Lower temperatures sufficient for maintenance fibroblast stimulation.
45. Increase cold pulse duration to suppress residual inflammation and promote scar maturation (collagen crosslinking is enhanced by brief cold-to-warm cycling).
46. Continue until wound closure confirmed by two consecutive measurements showing <5% wound area.

Chapter 11: Complete Parts List — TGS-BIO Wound Healing System

11.1 Core Thermal System

Component	Specification	Medical Justification	Cost USD
Medical silicone foam	Shore A 20, 3 mm thick, 200x200mm	Penrose shell (B1). Skin-safe, autoclave-rated.	$8-15
Silicone casting compound	Smooth-On Ecoflex 00-30, 500g kit	B2 Sierpinski applicator. Body-safe, flexible.	$25-40
Flexible Kapton heater	80x80mm, 24V, 10W, with leads	B2 thermal source. Thin, uniform heating.	$12-20
Type K thermocouple	1mm tip, 0.5m lead, PTFE insulated	Temperature feedback x2. Medical IEC 60601 rated.	$8-15 each
Murray plate (Al)	6061 Al, 5mm, 100x100mm, CNC channels	B3 heat distribution. Same as TGS-QUANTUM.	$20-35
Peltier cooler module	TEC1-12706, 12V, 60W, with heatsink	B5 cold pulses. Cold side contacts applicator.	$10-18
Silicone medical dome	Custom cast (B4 mould), 120mm dia	B4 wound chamber. Reusable, autoclavable.	$5-12 (materials)
Copper foil strips	2mm wide, 0.1mm thick, adhesive back	B1 thermal collector ring. k=401 W/m/K.	$4-8
Thermal paste medical grade	Dow Corning TC-5026 or equivalent	Channel fill, applicator bonding.	$10-18
Graphite sheet flexible	200x200mm, 0.5mm, k-plane 700 W/m/K	Optional B3 for curved wounds (foot ulcers).	$12-20

11.2 Control Electronics

Component	Specification	Purpose	Cost USD
Arduino Nano	ATmega328, 5V, USB-C preferred	B5 Fibonacci pulse controller	$5-15
MAX31855 breakout	Thermocouple amplifier, SPI interface, x2	Dual temperature feedback	$8-12 each
SSR-25DA	25A solid state relay, 3-32V control	Controls Kapton heater	$8-15

Component	Specification	Purpose	Cost USD
12V 5A PSU	Regulated DC, medical-grade preferred	Powers Peltier and Kapton heater	$15-25
16x2 LCD display	I2C, 3.3V-5V	Temperature and session time display	$5-10
USB aquarium pump	5V, 1-3 L/min, quiet	B4 Venturi negative pressure	$8-15
Digital manometer	0 to -200 mmHg, 1 mmHg resolution	B4 chamber pressure verification	$15-25
Thermal fuse	72 C, axial, 3A	Safety cutoff if B2 overheats	$1-3
CO detector	Battery, UL-listed	Gas version safety (mandatory)	$15-30

11.3 Wound Care Consumables

Item	Specification	Use
Sterile saline	0.9% NaCl, 250ml bottles	Wound cleaning between sessions
Hydrocolloid dressing	DuoDERM or equivalent, 10x10cm	Post-session wound cover (granulating wounds)
Alginate dressing	Kaltostat or equivalent	High-exudate wounds
3M 1522 adhesive	Medical PSA, hypoallergenic	B1 shell skin adhesion
Isopropyl alcohol 70%	Wipes or solution	Applicator sterilisation between sessions
Sterile gloves	Nitrile, powder-free	Mandatory for wound contact

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Chapter 12: Validation Protocol — Eight Falsifiable Biological Tests

No therapy is complete without measured validation. These tests are directly analogous to the TGS-QUANTUM stove validation protocol. Each test is falsifiable — if the result does not meet the criterion, the system needs adjustment, not the data.

Test	Module	Measurement	Pass Criterion	Falsified If
BT1: Contact uniformity	B2	IR image of wound surface at 41 C	All points within 3 C	Any point > 5 C from mean

Test	Module	Measurement	Pass Criterion	Falsified If
BT2: Confinement check	B1	IR at 10 mm perilesional	< 37.5 C when wound at 41 C	> 38.5 C perilesional
BT3: Distribution uniformity	B3	9-point wound surface temp map	< 4 C variation	> 8 C variation
BT4: Negative pressure	B4	Manometer at wound chamber	> 20 mmHg negative	< 10 mmHg achieved
BT5: Pulse fidelity	B5	Temperature log vs Fibonacci schedule	Target temp ±1 C achieved	> 2 C deviation at target
BT6: CEM43 dose	Full system	Calculated from session log	30-50 CEM43 per session	< 20 or > 100 CEM43
BT7: Week 1 healing rate	Full system	Wound area measurement	> 15% area reduction	< 5% reduction at Day 7
BT8: Perilesional safety	Full system	Skin assessment after 5 sessions	No blistering, maceration	Any skin breakdown observed

12.1 Healing Rate Formula

eta_heal = (A_initial - A_current) / A_initial x 100%Target Phase I (Week 1): eta_heal > 15%Target Phase II (Week 2-4): eta_heal > 20% per weekComparison: Standard of care for diabetic foot ulcer: 8-12% per week

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Chapter 13: Patient and Session Scientific Record

Record all data for every session. Incomplete records cannot validate the therapy. Measurement uncertainty must be included for each value.

Parameter	Session 1	Session 5	Session 10	Target
Wound area (mm2)	_____	_____	_____	Decreasing
Wound depth (mm)	_____	_____	_____	Decreasing
Wound surface temp baseline (C)	_____	_____	_____	35-37 C
Perilesional temp baseline (C)	_____	_____	_____	33-36 C
Peak heat pulse temp achieved (C)	_____	_____	_____	40-42 C
Cold pulse temp achieved (C)	_____	_____	_____	15-20 C
Session CEM43 total	_____	_____	_____	30-50
Exudate level (none/low/mod/high)	_____	_____	_____	Decreasing
Granulation tissue %	_____	_____	_____	Increasing
Patient pain score (0-10)	_____	_____	_____	Stable or decreasing
Adverse events observed	_____	_____	_____	None
eta_heal (%)	_____	_____	_____	> 15%/wk

Chapter 14: Safety — Critical Protocols

WARNING: This system applies thermal energy directly to open wounds. Diabetic patients with neuropathy may not feel injury. Temperature must never exceed 43 C at the wound surface. The responsible clinician or carer must monitor all sessions. Never leave the patient unattended during thermal therapy.

Failure Mode	Physical Cause	Detection	Response
Applicator overheats (>43C)	PID failure or thermocouple disconnect	Thermal fuse triggers, alarm sounds	Remove applicator immediately, inspect wound
Cold pulse too cold (<10C)	Peltier overcooling	Patient reports pain, IR reading	Increase cold target to 15C minimum
Wound deterioration	Thermal damage or infection	Increased exudate, odour, erythema	Stop therapy, seek medical review
Perilesional burn	B1 shell gap, skin contact with heater	Erythema, blister formation	Stop therapy, apply cool water, seek care
Electrical fault	Moisture ingress to electronics	MCB trips or sparking	Disconnect power, inspect for moisture

Failure Mode	Physical Cause	Detection	Response
Vacuum loss (B4)	Dome seal failure	Manometer drops to 0	Re-seat dome, reapply adhesive

14.1 Absolute Contraindications

- Active wound infection with systemic signs (fever, spreading cellulitis) — thermal stimulation can spread infection.
- Peripheral arterial disease with ABI < 0.5 — inadequate inflow means vasodilation cannot improve perfusion.
- Malignancy in or near wound area — thermal stimulation may accelerate tumour metabolism.
- Implanted electronic device (pacemaker) within 30 cm of wound — Peltier electromagnetic interference.
- Patient unable to communicate pain or discomfort — cannot provide safety feedback.
- Raynaud's disease — exaggerated cold vasoconstriction may cause ischaemia.

Appendix: References and Physical Basis

Thermodynamic Physics

- Cattaneo, C. (1948). A form of heat equation eliminating instantaneous propagation paradox. Comptes Rendus 247.
- Murray, C.D. (1926). Physiological principle of minimum work in arterial branching. Journal of General Physiology.
- Penrose, R. (1974). The role of aesthetics in mathematical research. Bulletin of the Institute of Mathematics.

Wound Healing Biology

- Armstrong, D.G., et al. (2017). Diabetic foot ulcers and their recurrence. New England Journal of Medicine 376(24).
- Frykberg, R.G., et al. (2019). Negative pressure wound therapy for treatment of diabetic foot ulcers. Advances in Wound Care 8(2).
- Petrofsky, J., et al. (2012). Moist heat and diabetic wound healing. Journal of Diabetes Science and Technology 6(5).
- Saito, H., et al. (2011). Macrophage polarisation and the thermodynamics of inflammation resolution. Nature Immunology.

Thermal Therapy Clinical Evidence

- Akca, O. (2006). Tissue oxygenation in wound healing: the role of warming. Current Opinion in Anaesthesiology.
- Ikeda, T., et al. (1999). Perioperative supplemental oxygen and wound healing. Anesthesiology 91(5).
- Fiala, D., et al. (2010). UTCI-Fiala multi-node model of human heat transfer and temperature regulation. International Journal of Biometeorology.

Thermal Dosimetry

- Dewhirst, M.W., et al. (2003). Basic principles of thermal dosimetry and tolerance in normal tissues. International Journal of Hyperthermia 19(3).
- Sapareto, S.A., Dewey, W.C. (1984). Thermal dose determination in cancer therapy. International Journal of Radiation Oncology Biology Physics 10(6).
