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Simulation of Heat Transfer in the “Biological Tissue – Thermoelectric Sensor – Environment” System for the Diagnosis of Inflammatory and Destructive Processes

The paper presents a physical and mathematical model of the complete measuring system formed by multilayer biological tissue, a contact thermoelectric heat flux sensor and the environment. The model is based on the Pennes bioheat equation, which accounts for blood perfusion and metabolic heat generation, and it includes the sensor itself as a set of thermally resistive layers, so that the perturbation introduced by the instrument into the quantity it measures can be evaluated quantitatively. Pathological states are described by local changes of two physiological parameters only: the volumetric blood perfusion rate and the specific metabolic heat generation. The boundary value problems were solved by the control volume method implemented in Python with the NumPy and SciPy scientific libraries; the implementation was verified against an analytical solution, by grid convergence and by cross-comparison of independent one- and two-dimensional formulations. It is shown that under standardized conditions the undisturbed heat flux density from the skin surface is 13.5 mW/cm², and that blood perfusion is the dominant physiological driver of this flux – about thirty times more influential than volumetric metabolic heat generation (26.7 % against 0.8 % of the flux, switch-off test) – so that a pure-conduction model is inadmissible for this class of problems. For a pathological region extending laterally under the whole sensor the flux changes by up to +24 % (inflammation), +42 % (proliferating tumour, Gautherie relation) and –20 % (ischaemia); for a realistic localized focus of 8 mm radius the tumour signal is smaller and depth-dependent, from +15.9 % at 1 mm depth to +3.0 % at 9 mm. Heat flux measurement is shown to be markedly

Citation: R.R. Kobylanskyi, V.V. Boichuk (2026). Simulation of Heat Transfer in the “Biological Tissue – Thermoelectric Sensor – Environment” System for the Diagnosis of Inflammatory and Destructive Processes. *Journal of Thermoelectricity*, (3), 91–117. <https://doi.org/10.63527/1607-8829-2026-3-91-117>

Received: 18.08.2026; Revised: 10.09.2026; Published: 30.09.2026

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more sensitive to these pathological changes than surface thermometry, whose useful signal (1.9–2.3 °C) is only a few times the resolution of clinical thermography, whereas the flux changes above are five to eight times the instrument's own passport error. The methodical error of a contact sensor of 14 × 14 × 3 mm – arising in comparable measure from the thermal resistance of its own layer stack, from the emissivity mismatch between its top face and skin, and, to the smallest extent, from the lateral bypass of flux around it – is –4.6 % under ideal contact and grows to –12.5 % for poor thermal contact; the sensor settling time is 1.5–2 min. The results define the physical limits of thermoelectric heat flux diagnostics and justify the requirements to sensor design and measurement procedure.

Keywords: thermoelectricity, heat flux density, thermoelectric sensor, bismuth telluride, Pennes bioheat equation, blood perfusion, computer simulation, control volume method, medical diagnostics, inflammation, tumour, ischaemia.

Introduction

General characterization of the problem. The temperature of the human body surface and the density of the heat flux leaving it are among the most informative physical quantities that reflect the state of the organism. Local thermal characteristics of biological tissue are determined by the intensity of metabolic processes and by the state of blood supply and microcirculation. Consequently, practically any pathological change – an inflammatory reaction, a vascular disorder, a neurological or neurotrophic lesion, a destructive or an oncological process – alters the local heat generation, and these alterations frequently precede the appearance of clinical symptoms [1–3]. Detecting them early opens the way to non-invasive and objective diagnostics of a wide range of diseases.

Of the two measurable thermal quantities, the heat flux density is the physically richer one. Temperature is a state variable of the surface; it is established by the balance between the heat delivered from the depth of the body and the heat removed into the environment, and the same surface temperature may correspond to appreciably different heat fluxes depending on the conditions of heat exchange. The heat flux density, in contrast, is an energy quantity: it measures directly how much thermal power the tissue delivers per unit area, and it is therefore linked to the physiological processes that generate and transport that power. This distinction is not merely qualitative, and one of the aims of the present work is to express it in numbers.

Semiconductor thermoelectric converters based on bismuth telluride are the natural instrument for such measurements. Their figure of merit is maximal precisely in the physiological temperature range, they are passive (the sensor introduces no energy into the tissue), compact, and their output is an electrical signal directly proportional to the heat flux passing through them [4–7]. Devices of this type, developed at the Institute of Thermoelectricity of the NAS and MES of Ukraine, have been applied to the diagnosis of degenerative-dystrophic diseases of the spine [2, 3], to neurotrophic injuries of the lower extremities [1], to perioperative monitoring in vitreoretinal surgery [8, 9], to the objective control of rehabilitation in locomotor therapy [10], and to experimental studies of localized heat release in inflammatory and oncological conditions [11]. The technological modes of manufacturing such sensors and the

procedure for their calibration have been established in [12, 13], and computer-aided design methods for thermoelectric microcalorimetric sensors have been developed in [14].

Analysis of the state of the art. Modelling of heat transfer in living tissue has been dominated for more than seven decades by the equation proposed by Pennes in 1948 [15], in which the blood supply is represented as a distributed volumetric heat source proportional to the difference between the arterial blood temperature and the local tissue temperature. Its simplicity and its reasonable accuracy on the scale of centimetres and minutes have kept it in use, and it remains the working tool of quantitative bioheat physics; recent reviews summarize its applications to thermal therapy and thermal comfort and the directions in which it is being refined [16]. Its principal simplification – the assumption of isotropic, spatially uniform perfusion that neglects the directionality of blood flow and the counter-current heat exchange in paired vessels – has motivated a number of modified formulations, among them models with heterogeneous perfusion fields [17]. For the spatial and temporal scales relevant to surface heat flux measurements the differences between these formulations lie within a few per cent, which is smaller than the inter-individual variability of tissue properties, so that the classical Pennes formulation remains an adequate and practical choice.

Interest in contact measurements of the heat flux from the skin has grown markedly in recent years, driven by wearable and non-invasive monitoring: flexible multimodal devices that infer the core body temperature from surface heat transfer [18] and wearable skin calorimeters that measure heat flux together with thermal resistance and heat capacity [19] have been reported. Developments in bismuth-telluride device technology, including medical and wearable applications, have been reviewed in [20].

What is missing from this picture is the measuring instrument itself. In the bioheat literature the sensor is normally either absent – the undisturbed flux at a free surface is computed - or treated as ideal, with zero thermal resistance. Yet a contact sensor placed on the skin is a thermally resistive body: it changes the local conditions of heat removal, part of the heat flux bypasses it through the adjacent skin, and the instrument therefore systematically reads less than the flux it was intended to measure. Without a quantitative description of this perturbation the methodical error of contact thermometry of heat flux remains unknown, and the requirements to sensor geometry, to the quality of the thermal contact and to the conditions of measurement cannot be substantiated physically.

The aim of the present work is to construct a physical and mathematical model of the complete “biological tissue – thermoelectric sensor – environment” system on the basis of the Pennes bioheat equation, to establish by computer simulation how inflammatory, oncological and ischaemic processes change the heat flux at the body surface, to evaluate quantitatively the perturbation introduced by the sensor, and thereby to substantiate physically the applicability of thermoelectric heat flux sensors to medical diagnostics and to define the limits of that applicability.

1. Physical and mathematical model

1.1. How a thermoelectric sensor measures a heat flux

The operation of the instrument sets the quantity that the model must predict, so it is worth recalling briefly. A thermoelectric heat flux sensor is a thermopile: a set of N semiconductor thermocouples connected electrically in series and thermally in parallel between two ceramic plates. When a heat flux of density q passes through the thermopile, a temperature difference is established across it,

$$\Delta T = q \frac{\delta}{k_{tp}}, \quad (1)$$

where δ is the height of the legs and k_{tp} the effective thermal conductivity of the thermopile. By the Seebeck effect this difference generates the electromotive force

$$U = N \alpha_{12} \Delta T = N \alpha_{12} \frac{\delta}{k_{tp}} q = S q, \quad (2)$$

where α_{12} is the differential Seebeck coefficient of the material pair and S the volt-watt sensitivity of the sensor, determined individually by calibration against a normalized heat flux [13]. For semiconductor legs based on Bi_2Te_3 the coefficient α_{12} reaches 300–400 $\mu\text{V/K}$ per couple, an order of magnitude above metallic pairs, which is what makes semiconductor thermopiles the practical choice for medical measurements [4, 20]. The reproducibility of the sensitivity within a batch is governed by the technology of leg commutation and by the electrical and thermal contact resistances of the “metal – thermoelectric material” junctions [21], and by the homogeneity of the material microstructure [22].

Two features of (2) matter for what follows. First, the sensor reads the heat flux through itself, not the flux that would exist at the undisturbed skin surface – and these are not the same quantity, because the sensor possesses a finite thermal resistance δ/k_{tp} that is unavoidable, since the same resistance is what creates the measurable signal. Second, the sensitivity grows with δ , so a more sensitive sensor perturbs the field more strongly. The model developed below is precisely the instrument for quantifying this trade-off.

1.2. Geometry of the system

The region of the body where the measurement is performed is represented as a multilayer half-space consisting of four plane layers of biological tissue: epidermis, dermis, subcutaneous fat and muscle, beneath which the deep tissues maintain the stable core temperature $T_b = 37^\circ\text{C}$ (Fig. 1). Each layer i is characterized by its thickness δ_i , thermal conductivity k_i , density ρ_i , specific heat c_i , volumetric blood perfusion rate $\omega_{b,i}$ and specific metabolic heat generation $Q_{m,i}$. The values used (Table 1) are representative of the ranges reported in the bioheat-modelling literature for human skin and subcutaneous tissue, of which [15, 23, 24] are general background rather than a single tabulated source reproduced verbatim; reported values for these parameters, and especially for blood perfusion, vary appreciably between sources and between anatomical sites, and this variability is the dominant source of uncertainty in the absolute value of q_0 (§ 2.3 gives a quantitative sensitivity estimate). The comparative and diagnostic conclusions of § 4, which rest on the asymmetry between two simultaneously measured,

nominally identical regions, are far less sensitive to this uncertainty than q_0 itself, since a consistent shift of the base-line parameters affects both channels alike.

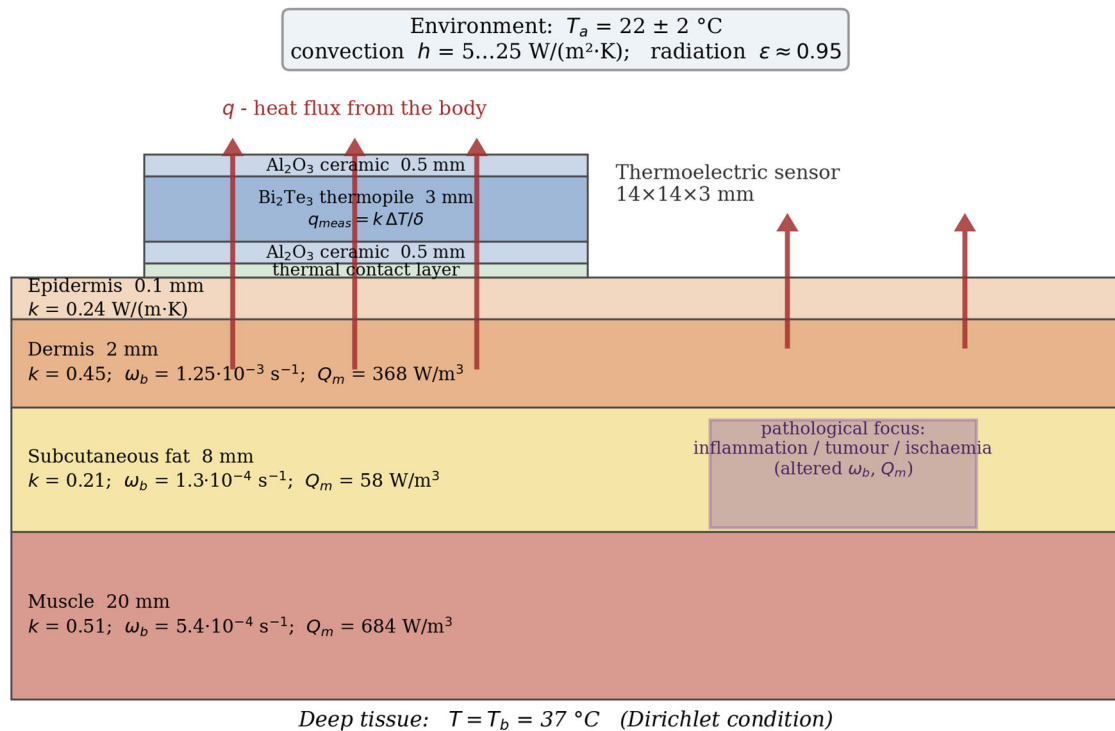


Fig. 1. Physical model of the “biological tissue – thermoelectric sensor – environment” system

Table 1

Thermophysical properties of the tissue layers (normal state)

Layer	δ , mm	k , W/(m·K)	ρ , kg/m ³	c , J/(kg·K)	ω_b , s ⁻¹	Q_m , W/m ³
Epidermis	0.10	0.24	1200	3590	0	0
Dermis	2.0	0.45	1200	3300	$1.25 \cdot 10^{-3}$	368
Subcutaneous fat	8.0	0.21	920	2500	$1.3 \cdot 10^{-4}$	58
Muscle	20.0	0.51	1085	3800	$5.4 \cdot 10^{-4}$	684

For blood, $\rho_b = 1060 \text{ kg/m}^3$, $c_b = 3770 \text{ J/(kg}\cdot\text{K)}$ and $T_b = 37 \text{ }^\circ\text{C}$ are adopted. The epidermis contains no blood vessels, so its perfusion and metabolic generation are zero; the main heat production is concentrated in the muscle layer and in the dermis.

The thermoelectric sensor is modelled as a stack of layers placed on the skin: a contact layer (gel or the natural microscopic gap, $\delta = 0.05 \text{ mm}$, $k = 0.30 \text{ W/(m}\cdot\text{K)}$), a lower Al₂O₃ ceramic plate (0.5 mm, $25 \text{ W/(m}\cdot\text{K)}$), a Bi₂Te₃ thermopile (2–3 mm; effective thermal conductivity $k_{tp} \approx 1.2 \text{ W/(m}\cdot\text{K)}$ with allowance for the leg filling factor) and an upper ceramic plate. These dimensions correspond to the experimental sensor of $14 \times 14 \times 3 \text{ mm}$ with a $10 \times 10 \times 3 \text{ mm}$ thermopile used in the multichannel heat meter [1].

1.3. The Pennes bioheat equation and the meaning of its terms

Heat transfer in perfused biological tissue is described by the equation proposed by Pennes [15]:

$$\rho c \frac{\partial T}{\partial t} = \nabla \cdot (k \nabla T) + \rho_b c_b \omega_b (T_b - T) + Q_m. \quad (3)$$

The left-hand side is the rate of change of the thermal energy stored in a unit volume of tissue. The first term on the right is ordinary conduction. The two remaining terms are what distinguishes living tissue from an inert body, and their physical content deserves to be stated explicitly, because the whole diagnostic argument of this paper rests on them.

The term $\rho_b c_b \omega_b (T_b - T)$ describes the convective heat supply by blood. The volumetric perfusion rate ω_b (s^{-1}) is the volume of blood passing through a unit volume of tissue per unit time in the exchange vessels – the capillary bed, where blood is in thermal contact with the tissue. Arterial blood arrives at the core temperature $T_b = 37^\circ C$, which is higher than the temperature of the superficial layers (about $32^\circ C$ at the skin surface), gives up heat to the tissue in proportion to the local temperature difference, and leaves cooled. Blood therefore acts as a distributed heater of the superficial tissues, short-circuiting their high thermal resistance. The Pennes formulation treats this exchange as isotropic and spatially uniform within a layer, which is the simplification discussed in the Introduction.

The term Q_m is the specific metabolic heat generation – the thermal power released per unit volume by the biochemical processes in the cells themselves.

The essential point for diagnostics is that these two terms respond to pathology in a characteristic and physiologically well-understood way, so that a pathological state can be introduced into the model by altering them alone, without changing the structure of the equation.

1.4. Modelling of pathological states

A pathological focus is represented as a local region – a layer in the one-dimensional formulation or a cylindrical region in the two-dimensional one – in which the perfusion and the metabolic generation differ from their normal values (Table 2).

Table 2

Models of pathological states of biological tissue

State	Perfusion ω_b	Metabolism Q_m	Physiological basis
Inflammation	$\times(2\dots5)$	$\times(5\dots10)$	hyperaemia, intensified metabolism [2, 11]
Tumour (proliferating)	$\times 2$	by (4)	neoangiogenesis, abnormal metabolism [25]
Ischaemia, thrombosis, destruction	$\times(0.1\dots0.3)$	$\times 0.5$	suppressed blood supply [11]

In acute inflammation the vascular reaction follows a typical sequence: a brief arteriolar spasm gives way to sustained arterial hyperaemia with dilatation of the resistance vessels and opening of reserve capillaries, so that the local blood flow rises several-fold; in parallel,

inflammatory mediators intensify cellular metabolism. Both mechanisms act in the same direction – an increase of local heat generation – which makes an inflammatory focus the most contrasted object of heat flux diagnostics.

In ischaemia, thrombosis and destructive processes the blood supply of the affected region is suppressed, the perfusion falls to 10–30 % of normal and the metabolic activity declines, so the heat flux decreases.

Malignant neoplasms behave in a more complex way. The metabolic heat generation of actively proliferating tumour tissue is related to the doubling time of the tumour volume by the empirical relation established by Gautherie [25]:

$$Q_m^{tum} = \frac{3.27 \cdot 10^6}{\tau_d} \text{ W/m}^3, \quad (4)$$

where the constant $3.27 \cdot 10^6$ carries units of $\text{W} \cdot \text{day/m}^3$, so that with τ_d expressed in days the right-hand side is dimensionally a volumetric power in W/m^3 ; the same value and the same primary source are used elsewhere in the tumour bioheat-modelling literature [26]. Reported tumour volume doubling times span a wide range – from about ten days for the most rapidly progressing lesions to several years for slowly growing ones [27] – but the range $\tau_d = 50 \dots 300$ days adopted here is representative specifically of the actively proliferating, thermally detectable tumours that are the object of this study, rather than of the whole clinical spectrum; it gives $Q_m^{tum} \approx 11 \dots 65 \text{ kW/m}^3$, one to two orders of magnitude above healthy tissue. Relation (4) describes such an actively proliferating, well-vascularized tumour. Foci with a necrotic core and impaired blood supply, which were observed in medical trials of thermoelectric sensors [11] and which showed reduced heat generation, are described within the same model by the ischaemic regime of Table 2. The model thus reproduces both experimentally observed signs of the thermal anomaly of oncological foci, and the sign is determined by the balance between the metabolic activity of the focus and the state of its perfusion.

1.5. Boundary conditions

On the free skin surface and on the upper surface of the sensor a convective-radiative heat exchange with the environment is prescribed:

$$-k \frac{\partial T}{\partial n} = h (T_s - T_a) + \varepsilon \sigma (T_s^4 - T_a^4), \quad (5)$$

where h is the convective heat transfer coefficient ($5\text{--}25 \text{ W}/(\text{m}^2 \cdot \text{K})$ depending on air movement; the base value $8 \text{ W}/(\text{m}^2 \cdot \text{K})$ corresponds to still air), ε the surface emissivity (0.95 for skin, 0.90 for the sensor) and $\sigma = 5.67 \cdot 10^{-8} \text{ W}/(\text{m}^2 \cdot \text{K}^4)$ the Stefan–Boltzmann constant. The base ambient temperature $T_a = 22 \text{ }^\circ\text{C}$ corresponds to the standardized conditions of medical measurements ($22 \pm 2 \text{ }^\circ\text{C}$). At the lower boundary of the region the condition of the first kind $T = T_b = 37 \text{ }^\circ\text{C}$ is imposed.

Beneath the sensor the boundary condition expresses the heat transfer through the whole sensor stack:

$$-k \frac{\partial T}{\partial z} = U_c (T - T_a), \quad z = 0, \quad r \leq a; \quad U_c = (R_c + \sum_i \frac{\delta_i}{k_i} + \frac{1}{h_{up}})^{-1}, \quad (6)$$

where a is the sensor radius, R_c the thermal resistance of the “skin–sensor” contact, the sum is

taken over the sensor layers and h_{up} is the effective heat transfer coefficient from the upper surface of the sensor. Condition (6) is the formal expression of the fact that the sensor is a thermally resistive body, and it is what makes the quantitative analysis of the methodical error possible. All two-dimensional computations reported below use the lumped form (6), i.e. the sensor stack of § 1.2 is collapsed into the single series resistance $\sum_i \delta_i / k_i$ applied as a uniform Robin condition at $z = 0$, $r \leq a$; the individual layers are not resolved as separate finite-thickness bodies inside the two-dimensional domain. This is a conservative simplification for the lateral bypass estimate of § 4.2: because the alumina plates of the stack have a thermal conductivity about two orders of magnitude above that of tissue, an explicitly resolved stack would spread heat laterally within the sensor itself and could either partly compensate or add to the bypass captured by the tissue-side field alone. The infinite-radius control computation of § 4.2, however, isolates the non-bypass part of the error by construction (bypass is geometrically impossible there regardless of how the stack is discretized), so the conclusion that lateral bypass is a minority contributor to the total error does not depend on this simplification; only the exact numerical split between the stack-resistance and bypass contributions could shift with a fully resolved sensor geometry.

In the one-dimensional formulation (coordinate x directed into the body) the steady-state problem takes the form

$$\frac{d}{dx} \left(k(x) \frac{dT}{dx} \right) + \rho_b c_b \omega_b(x) (T_b - T(x)) + Q_m(x) = 0, \quad 0 < x < L, \quad (7)$$

with $L = 30.1$ mm, the sum of the layer thicknesses of Table 1. In the two-dimensional axisymmetric formulation (coordinates r, z , the axis z passing through the centre of the sensor) the steady-state Pennes equation reads

$$\frac{1}{r} \frac{\partial}{\partial r} \left(k r \frac{\partial T}{\partial r} \right) + \frac{\partial}{\partial z} \left(k \frac{\partial T}{\partial z} \right) + \rho_b c_b \omega_b (T_b - T) + Q_m = 0 \quad (8)$$

in the region $0 \leq r \leq R = 40$ mm, $0 \leq z \leq L$, with symmetry conditions $\partial T / \partial r = 0$ at $r = 0$ and at $r = R$ (far field), condition (5) on the free skin surface at $r > a$ and condition (6) beneath the sensor at $r \leq a$.

The choice between the two formulations is dictated by the physics of the effect studied. The one-dimensional problem is adequate where the characteristic lateral dimensions are large compared with the depth of penetration of the disturbance, and it is used here for the steady thermal states of tissue and for transient processes, its advantage being computational economy that permits massive parametric studies. The two-dimensional axisymmetric problem is indispensable where the lateral effects are decisive - the bypass of heat flux around a sensor of finite size, edge effects, localized foci – and it is used for the analysis of the methodical error and of the spatial resolution.

2. Numerical method and computational implementation

2.1. Discretization by the control volume method

Equations (7) and (8) were discretized by the control volume method, which was chosen because it guarantees the exact satisfaction of the energy balance on every cell of the grid and the correct conjugation of heat fluxes at the interfaces between layers of different thermal

conductivity – a requirement that is essential here, since the conductivities of the adjacent media differ by up to two orders of magnitude (0.21 W/(m·K) for fat against 25 W/(m·K) for the ceramic plate).

Integration of (7) over the j -th cell of thickness Δx_j gives the balance equation

$$g_{j-1/2} (T_{j-1} - T_j) + g_{j+1/2} (T_{j+1} - T_j) + \rho_b c_b \omega_{b,j} \Delta x_j (T_b - T_j) + Q_{m,j} \Delta x_j = 0, \quad (9)$$

in which the inter-cell conductances are evaluated as harmonic means,

$$g_{j+1/2} = \left(\frac{\Delta x_j}{2k_j} + \frac{\Delta x_{j+1}}{2k_{j+1}} \right)^{-1}, \quad (10)$$

which ensures the continuity of the heat flux across an interface between dissimilar layers; an arithmetic mean would violate it and would introduce a systematic error precisely at the sensor–skin boundary, where the accuracy matters most. The boundary condition (5) is implemented through the effective conductance of the near-surface half-cell, $G_0 = (\Delta x_1/2k_1 + 1/h_{eff})^{-1}$, where $h_{eff} = h + h_{rad}$ and the radiative part is linearized as

$$h_{rad} = \varepsilon \sigma (T_s^2 + T_a^2)(T_s + T_a) \quad (11)$$

and refined iteratively, convergence to 10^{-4} °C in the surface temperature being reached in three to four iterations.

In the two-dimensional axisymmetric problem the control volumes are rings of rectangular cross-section; the coupling coefficients are built from the same harmonically averaged conductances multiplied by the areas of the lateral ($2\pi r \Delta z$) and end ($\pi(r_{out}^2 - r_{in}^2)$) faces of each volume.

For the transient problem a fully implicit (backward Euler) scheme was used: the term $\rho_j c_j \Delta x_j (T_j^{n+1} - T_j^n)/\Delta t$ is added to the left-hand side of (9). The scheme is unconditionally stable, so the time step is dictated by accuracy rather than by stability; a step of 0.5 s was adopted, which resolves the settling process with a margin.

2.2. Software implementation

The model was implemented as a set of original modules in Python 3.11 using the NumPy 2.4 and SciPy 1.17 scientific libraries; the graphical output was produced with Matplotlib. No commercial finite-element package was used: the direct implementation keeps every stage of the computation - the construction of the grid, the assembly of the coefficients, the solution and the post-processing - explicit and open to inspection and reproduction.

In the one-dimensional model a non-uniform grid is generated with refinement in the thin layers (no fewer than six cells per layer); with a target step of 0.05 mm this gives 606 control volumes for the tissue package. The resulting system of algebraic equations is tridiagonal and is solved by the Thomas algorithm in $O(N)$ operations; a single steady-state solution takes about 2.4 ms, which makes massive parametric series practicable.

In the two-dimensional model a structured grid with refinement near the body surface and near the sensor edge is used; the working grid of 140×120 control volumes corresponds to 16 800 unknowns. The assembled sparse five-diagonal system is stored in the scipy.sparse format and solved by the direct method `scipy.sparse.linalg.spsolve`; one solution with three iterations of the radiative refinement takes about 0.62 s. The complete set of computations reported below

– more than seventy two-dimensional solutions, tallying the field and profile plots, the four parametric sweeps of Fig. 5 and the 5×7 focus-position map of Fig. 7 – is executed on an ordinary personal computer in a few minutes, so the model is a practical engineering tool for the design of new instruments rather than a one-off calculation.

The signal of the sensor is extracted from the solution as the mean heat flux through the thermopile layer, in accordance with (1)–(2); the “measured” flux q_{meas} in the two-dimensional model is the heat flux through the surface $z = 0$ averaged over the sensor footprint $r \leq a$, with $a = 7$ mm the radius of the disc of area equal to the sensor’s 14×14 mm square footprint (7.90 mm would instead be exactly area-equivalent; the 2 % difference in radius is immaterial to the axisymmetric approximation adopted). The physical signal, strictly, is generated only by the 10×10 mm thermopile at the centre of this footprint (area-equivalent radius 5.64 mm); averaging over that smaller area instead changes q_{meas} by less than 0.1 pp of error (§ 4.2), so the footprint-averaged definition is retained throughout for simplicity without materially affecting any of the reported values. The undisturbed flux q_0 is obtained from the solution of the same problem with the sensor removed. The methodical error of the contact measurement is accordingly defined as

$$\eta_q = \frac{q_{meas} - q_0}{q_0} \cdot 100 \%. \quad (12)$$

2.3. Verification

The correctness of the implementation was established at three independent levels.

Comparison with an analytical solution. For the degenerate case of a single layer without perfusion and metabolism ($k = 0.4$ W/(m·K), $L = 20$ mm, $h = 10$ W/(m²·K) without the radiative component, $T = 37$ °C at the deep boundary and $T_a = 22$ °C) the exact solution gives $q = (37 - 22)/(L/k + 1/h) = 100.0000$ W/m², which the numerical solution reproduces with a discrepancy below 10^{-4} %. Because the control-volume scheme with harmonic-mean interface conductances reproduces a pure series thermal resistance exactly, this case tests only the assembly of the boundary condition and the linear solve and does not by itself exercise the perfusion source term. A second case therefore retains the perfusion term at constant properties ($k = 0.5$ W/(m·K), $\omega_b = 1.0 \cdot 10^{-3}$ s⁻¹, $L = 30$ mm, $h = 10$ W/(m²·K), no radiation), for which the steady Pennes equation has the closed-form solution $T(x) - T_b = C \sinh[m(L - x)]$ with $m = \sqrt{\rho_b c_b \omega_b / k}$, giving $q = 122.78697$ W/m²; the discretized scheme reproduces it with a discrepancy of 0.011 % at a step of 0.8 mm, falling to 0.000045 % at 0.05 mm, and the observed rate of convergence is second order ($p \approx 2.00$ between successive halvings of the step), as expected for the scheme used. Together the two cases confirm the correctness of the discretization and of the boundary conditions for both the conductive and the source-term parts of the operator.

Grid convergence. For the one-dimensional model the computed value of q_0 stabilizes to four significant figures already at a step of 0.1 mm (13.5192, 13.5193, 13.5193 and 13.5193 mW/cm² for steps of 0.200, 0.100, 0.050 and 0.025 mm). Because the rule of no fewer than six cells per layer pins the resolution of the thinnest layer (the 0.1 mm epidermis) regardless of the

nominal step, the actual minimum cell size is 0.0167 mm at every one of these four steps; the apparent flatness of the sequence is a consequence of this floor and is not evidence that a coarser epidermal resolution would also suffice. For the two-dimensional model the change of q_{meas} between the grids of 120×140 and 160×180 control volumes does not exceed 0.07 % (12.909 against 12.901 mW/cm²), which is two orders of magnitude below the effects under study.

Cross-verification of independent formulations. The undisturbed heat flux density from the free skin surface computed independently by the one- and two-dimensional models is 13.52 and 13.53 mW/cm² respectively, a discrepancy below 0.1 %. Since the two implementations share neither the grid generator, nor the assembly of coefficients, nor the linear solver, their agreement is a meaningful test of both.

Parameter sensitivity of q_0 . The three verifications above establish that the numerical scheme solves the stated equations correctly; they say nothing about the uncertainty carried by the input parameters themselves, which – as noted in § 1.2 – is the dominant source of uncertainty in the absolute value of q_0 . Table 1 was therefore perturbed one parameter at a time, over a plausible range of literature variability (a factor of two for perfusion and metabolism, ± 20 % for conductivity), keeping all other parameters fixed:

Table A

Sensitivity of the undisturbed flux q_0 to individual tissue parameters

Parameter varied	$\times 0.5$ (or $\times 0.8$)	$\times 2.0$ (or $\times 1.2$)
Dermal perfusion ω_b	–6.9 %	+9.8 %
Fat conductivity k	–2.6 %	+2.0 %
Muscle perfusion ω_b	–1.1 %	+1.4 %
Dermal conductivity k	–0.7 %	+0.5 %
Muscle metabolism Q_m	–0.3 %	+0.6 %

Dermal perfusion dominates, consistent with the dermis being the shallowest perfused layer and therefore the one most directly coupled to the surface flux; a conservative combined estimate is $q_0 = 13.5$ mW/cm² with a parameter-driven uncertainty of the order of ± 10 %. This uncertainty attaches to the absolute value of q_0 quoted throughout the paper; it does not carry over in the same proportion to the relative pathological contrasts of Table 3 or to the asymmetry criterion of § 5, both of which compare two regions modelled with the same nominal parameters and are correspondingly more robust.

Adequacy of the domain size. The choice of $L = 30.1$ mm for the deep boundary and $R = 40$ mm for the far-field radius was checked directly rather than assumed. The characteristic perfusion length of the muscle layer, $\sqrt{k/(\rho_b c_b \omega_b)} \approx 15.4$ mm, sets the natural decay scale of a thermal disturbance in that layer; extending the muscle layer from 20 to 120 mm while keeping all other layers unchanged shifts q_0 by only –0.5 %, confirming that the Dirichlet condition at $L = 30.1$ mm (about 1.3 perfusion lengths into the muscle) does not materially affect the result. Similarly, the two-dimensional field is undisturbed by the sensor to within 0.05 % beyond $r \approx 19$ mm (§ 4.1), well inside the domain radius $R = 40$ mm used throughout.

3. Heat transfer in normal and pathological tissue

3.1. Steady thermal states

Fig. 2, a shows the steady temperature profiles in tissue for four states – normal, inflammation, tumour and ischaemia – computed by the one-dimensional model (7) for free skin under standardized conditions ($T_a = 22\text{ °C}$, $h = 8\text{ W/(m}^2\cdot\text{K)}$); Fig. 2, b gives the corresponding heat flux densities at the surface. The numerical characteristics are collected in Table 3.

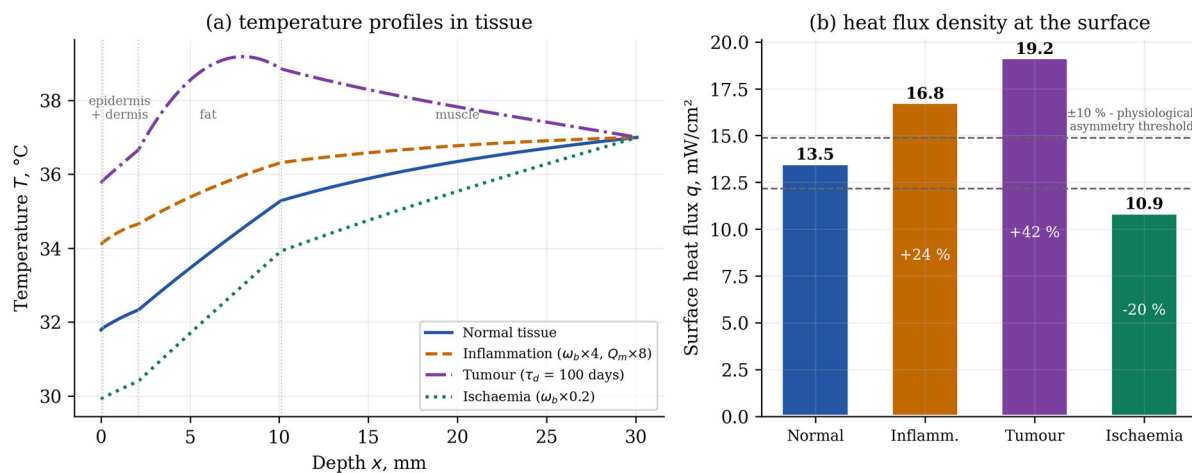


Fig. 2. Steady thermal state of biological tissue: (a) temperature profiles $T(x)$;
(b) heat flux density at the surface

Table 3

Computed thermal characteristics of the body surface for the base scenarios, one-dimensional model, pathology occupying the whole area of the affected layers under the sensor ($T_a = 22\text{ °C}$, $h = 8\text{ W/(m}^2\cdot\text{K)}$, free skin). These are upper-bound estimates for a laterally extended lesion; § 4.4 and Fig. 7 give the corresponding values for a realistic, laterally localized focus, which are smaller and depend on its depth

Scenario	Layers affected	T_{skin} , °C	ΔT to normal, °C	q , mW/cm ²	$\Delta q/q_0$, %
Normal tissue	—	31.8	—	13.5	—
Inflammation ($\omega_b \times 4$, $Q_m \times 8$)	dermis + fat	34.1	+2.3	16.8	+24.1
Tumour ($\tau_d = 100$ days, $\omega_b \times 2$)	fat, 2–10 mm deep	35.8	+4.0	19.2	+41.8
Ischaemia ($\omega_b \times 0.2$, $Q_m \times 0.5$)	dermis + fat + muscle	29.9	−1.9	10.9	−19.5

In the normal state the temperature falls monotonically from 37 °C in the deep tissues to 31.8 °C at the surface, the steepest gradient being concentrated in the subcutaneous fat, which is the principal thermal resistance of the tissue package; the heat flux density at the surface is

$q_0 = 13.5 \text{ mW/cm}^2$. An inflammatory focus occupying the whole area of the dermis and the fat draws the temperature profile towards the arterial value: the surface temperature rises to 34.1°C and the heat flux to 16.8 mW/cm^2 , that is by 24 % relative to normal. A proliferating tumour in the fat layer at a depth of 2–10 mm ($\tau_d = 100$ days, $Q_m = 32.7 \text{ kW/m}^3$ by (4)) creates a local temperature maximum of 39.2°C at a depth of about 8 mm and, likewise occupying the whole layer, raises the surface flux by 42 %; a realistic, laterally confined tumour of the same activity produces a substantially smaller signal, as § 4.4 shows. Ischaemia of the dermis, fat and muscle together lowers the surface temperature to 29.9°C and the flux to 10.9 mW/cm^2 , that is by 19.5 %; ischaemia confined to the dermis and fat alone, without involving the muscle, gives –15.3 %.

These whole-layer figures have a direct diagnostic meaning as upper bounds: the deviations for inflammation (+24 %) and ischaemia (–19.5 %) clear the threshold of physiological asymmetry between symmetric regions of the body adopted in clinical practice (10 % [1, 11]) with a comfortable margin, so foci of this lateral extent are reliably detected by a multichannel instrument comparing the affected and the contralateral regions. For a realistic, laterally localized focus the margin is narrower and depends on its size and depth, as quantified in § 4.4.

The computed absolute value $q_0 = 13.5 \text{ mW/cm}^2$ itself is worth relating to published measurements rather than left as a self-contained model output. Rodríguez de Rivera et al. [19] report skin heat flux of about $35\text{--}42 \text{ mW/cm}^2$ measured with a wearable calorimeter thermostatted to 28°C in a $22\text{--}24^\circ\text{C}$ room; this is not directly comparable to the present passive-convection value, since an actively thermostatted sensor imposes a driving temperature difference several times larger than the passive skin-to-air difference modelled here, and the comparison mainly illustrates the sensitivity of the absolute flux to the measurement's own thermal boundary condition, consistent with the ambient-temperature dependence quantified in § 4.2. A direct comparison against the group's own clinical measurements [1–3, 11] – which already supply the 10 % asymmetry threshold and the 3–5 % passport error used throughout this paper as empirical inputs – would be the natural next step and is identified as such in the Conclusions; it has not been attempted here because those studies report asymmetry and calibrated instrument readings rather than the absolute undisturbed flux under the specific standardized conditions ($T_a = 22^\circ\text{C}$, free skin, no sensor) that the present model addresses.

3.2. What the heat flux is generated by

The most instructive result of the model is obtained not from the pathological scenarios themselves but from a term-by-term analysis of the Pennes equation. Table 4 shows what remains of the surface heat flux when the perfusion and the metabolic terms of (3) are switched off in turn.

The result is unambiguous: the perfusion term is decisive – switching it off lowers the heat flux by almost 27 % and the surface temperature by 2.6°C – whereas the direct volumetric metabolic heat generation contributes only 0.8 %. The physical reason is that blood arriving at the core temperature of 37°C effectively heats the superficial layers, short-circuiting their high thermal resistance, while the intrinsic metabolic heat release of soft tissues at rest is small compared with this convective supply.

Table 4

*Contribution of the terms of the Pennes equation to the formation of the heat flux
from the skin surface (normal tissue, standardized conditions)*

Model variant	q , mW/cm ²	T_{skin} , °C	Deviation from the full model, %
Full Pennes model	13.52	31.79	–
Without metabolism ($Q_m = 0$)	13.41	31.71	–0.8
Without perfusion ($\omega_b = 0$)	9.91	29.21	–26.7
Pure conduction ($\omega_b = 0$, $Q_m = 0$)	9.68	29.05	–28.4

Two conclusions follow. The first is methodological: the use of a simplified pure-conduction model for problems of surface heat flux measurement is inadmissible, since it underestimates the flux by more than a quarter - more than all the diagnostic effects under study taken together. The Pennes equation is thus the minimally sufficient model for this class of problems. The second conclusion is diagnostic, and it is the physical foundation of the whole method: perfusion is about thirty times more influential on the measured flux than volumetric metabolism (26.7 % against 0.8 %, and the comparison if anything understates perfusion’s role, since the deep boundary condition is itself sustained by the systemic circulation), so that thermoelectric heat flux measurement is primarily a method of assessing the state of tissue blood supply. This explains why the method is highly sensitive to vascular and neurotrophic disorders, which alter perfusion, and why the clinical observations of the greatest contrast were obtained precisely for hyperaemic (inflammatory) and hypoperfused (ischaemic, thrombotic) states [1–3, 11].

3.3. Why heat flux rather than temperature

The data of Table 3 permit a comparison of the two measurable quantities as diagnostic parameters, but the comparison must be made with care: expressing the temperature change as a percentage of a Celsius reading is not physically meaningful, since it depends on the arbitrary zero of that scale. For inflammation the surface temperature rises by 2.3 °C – which is 7 % of the Celsius reading 31.8 °C, but only 0.75 % of the absolute temperature 305 K – while the heat flux rises by 24 %; for ischaemia the corresponding figures are 1.9 °C (6 % Celsius, 0.6 % absolute) and 19.5 %. A “factor of three to four” built on the Celsius percentage is accordingly an artefact of the scale, not a physical result, and the two pathologies in fact give a closely similar ratio (≈ 3.2 – 3.3) once compared this way.

The physically meaningful comparison is each modality’s signal against its own noise floor rather than against an arbitrary reference point. Clinical infrared thermography resolves temperature to about 0.05–0.1 °C, so the 1.9–2.3 °C changes studied here are twenty to fifty times its resolution; the thermoelectric sensor’s own passport error is 3–5 % [1, 11], so the 20–24 % flux changes are five to eight times that error. Both modalities therefore register these particular pathologies comfortably, and the advantage of the flux channel established in this work is not a simple multiple on this basis. It is, first, structural: the surface temperature is

bounded from above by the arterial temperature and from below by the ambient temperature, so its whole physiological range of variation is compressed into a few degrees, whereas the heat flux is not bounded in this way and tracks the change of the underlying thermal source directly. Second, and more practically, the flux channel is comparatively insensitive to the ambient-radiative and surface-emissivity artefacts that dominate the error budget of thermography (§ 4.2 quantifies this insensitivity for the sensor itself). Finally, in both modalities the factor that ultimately limits detectability of small or deep anomalies is the 10 % threshold of physiological inter-regional asymmetry (§ 5), which is a property of the organism, common to both channels, rather than an instrumental limit that heat flux measurement removes. This refines, rather than qualitatively changes, the thesis argued mainly qualitatively in [2, 3, 11] that heat flux density is the more robust diagnostic parameter, and it also explains why infrared thermography, which by its physical principle registers only the surface temperature, remains more exposed to these confounds [28, 29].

4. Perturbation introduced by the sensor

4.1. Two-dimensional temperature fields

Fig. 3 shows the two-dimensional temperature field in tissue with a sensor of radius $a = 7$ mm installed, in the presence of a tumour focus (a cylinder $r \leq 6$ mm at a depth of 3–9 mm), computed with the model (8), (6).

The sensor deforms the isotherms appreciably only in a near-surface layer a few millimetres thick: beneath the sensor the surface temperature rises, because the thermal resistance of the sensor stack impedes the flux, while immediately beyond its edge an annular zone of increased heat removal is formed. The pathological focus manifests itself as a characteristic “dome” of isotherms that reaches the surface and produces a registrable anomaly of the flux even through the sensor.

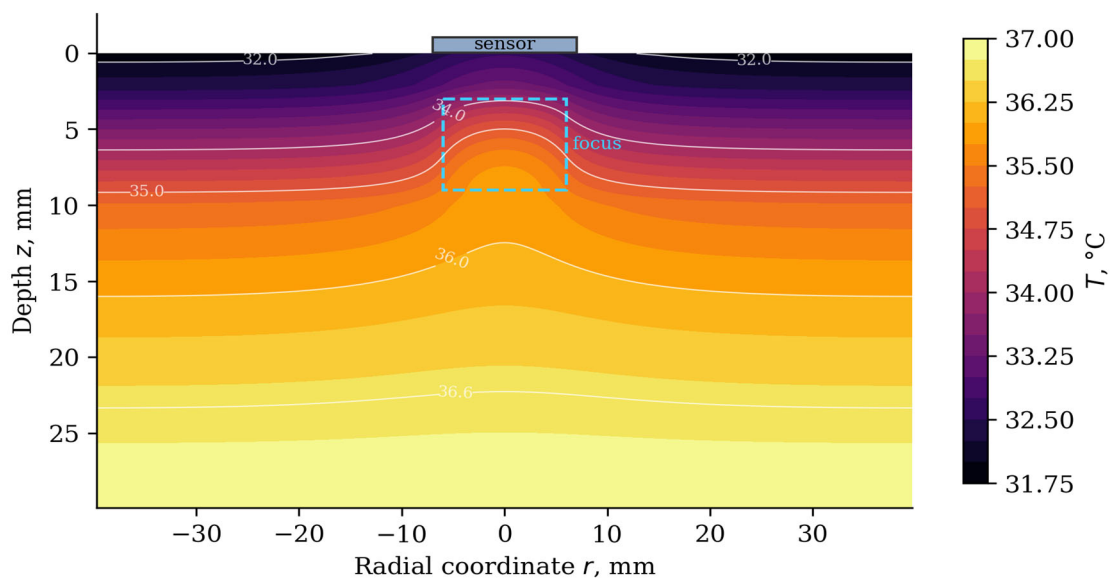


Fig. 3. Temperature field in tissue beneath the sensor in the presence of a tumour focus ($r \leq 6$ mm, 3–9 mm deep, $\tau_d = 100$ days)

The distribution of the heat flux density over the skin surface (Fig. 4) quantifies both effects. Beneath the sensor the flux is reduced relative to the undisturbed value (12.91 against 13.53 mW/cm² for normal tissue, a –4.6 % error); a small peak is observed at the sensor edge; and the field returns to within 0.2 % of its undisturbed value by about 11 mm from the axis (4 mm beyond the sensor edge) and to within 0.05 % by about 19 mm. From this follows a practical recommendation: adjacent sensors of a multichannel instrument should be placed no closer than about 20 mm from one another – a margin that already includes a safety factor over the computed extent of the disturbance – otherwise they perturb each other’s readings.

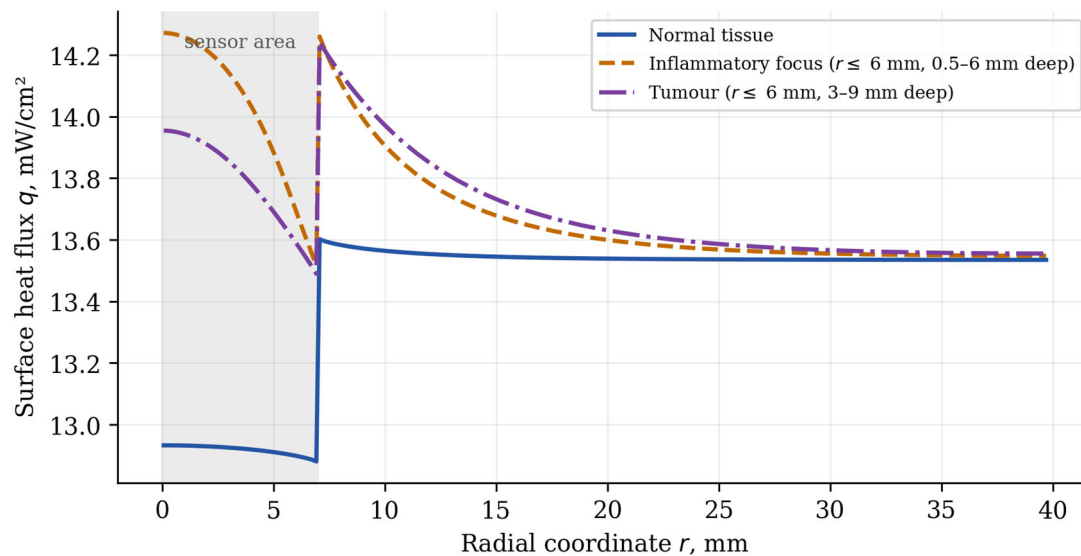


Fig. 4. Distribution of the heat flux density over the skin surface with the sensor installed, for normal tissue and for tissue with pathological foci

A more detailed analysis gives further quantitative characteristics. In the steady state the temperature of the upper surface of the sensor, computed from the converged flux through it as $T_{top} = T_a + q_{meas}/h_{up,eff} \approx 31.6$ °C, is essentially the same as that of the adjacent free skin (31.8 °C) – the small methodical error established below does not translate into an appreciable thermal offset of the sensor’s exposed face, and it is precisely the much smaller temperature drop across the thermopile itself (about 0.3–0.4 °C under normal conditions) that forms the useful signal in accordance with (1). The edge peak of the flux is modest: at most about +0.5 % just outside the sensor rim, decaying to under 0.2 % within a few millimetres (Fig. 4); an energy balance over the whole modelled surface confirms that only about a quarter of the flux deficit under the sensor re-emerges through this route, the remainder simply not being extracted from the body while the sensor is in place (§ 4.2). For sensor design this suggests the usefulness of a guard ring around the measuring zone that would displace even this modest edge effect beyond the limits of the thermopile – a construction realized in the guard elements of the calibration bench [13].

4.2. Methodical error: design and external factors

A systematic study of the methodical error (12) was carried out as a series of two-dimensional computations; the results are collected in Fig. 5.

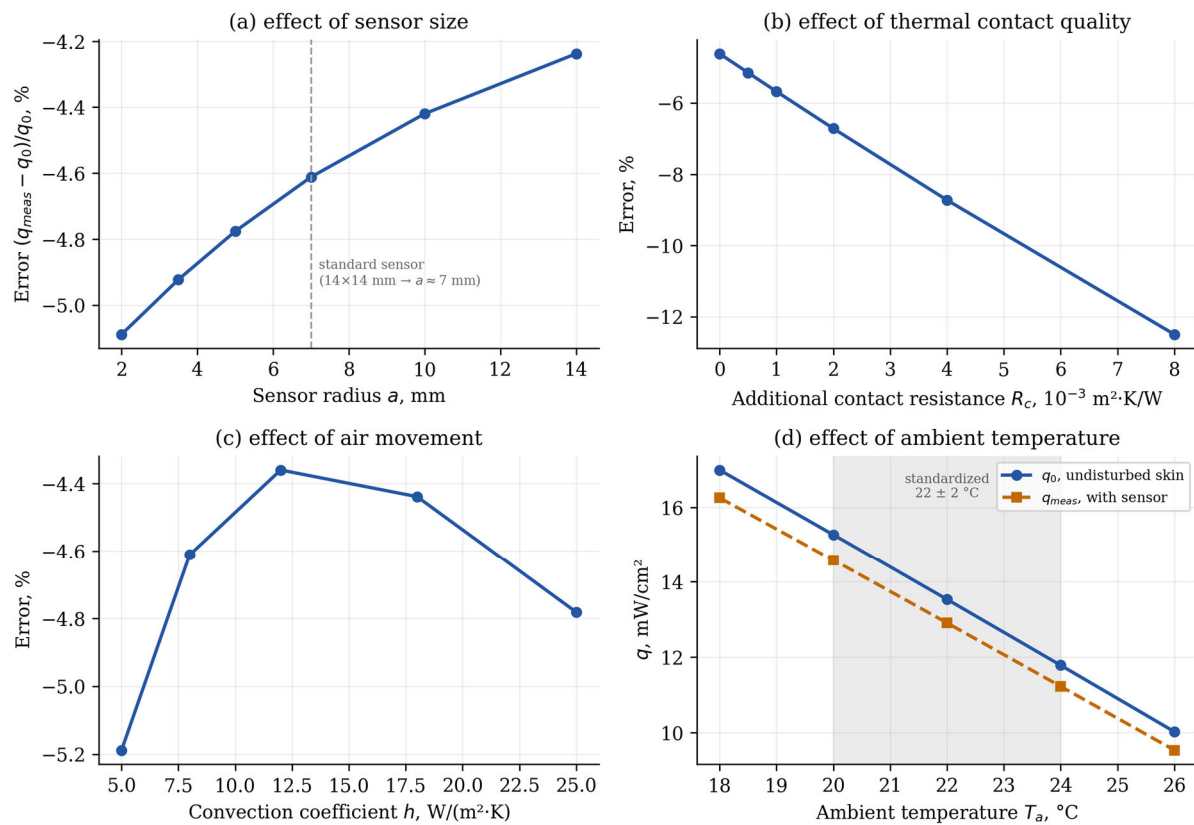


Fig. 5. Methodical error of the contact measurement of heat flux density: (a) effect of sensor size; (b) effect of thermal contact quality; (c) effect of air movement; (d) effect of ambient temperature

Before turning to the parametric sweeps, it is worth establishing what the -4.6% error at the standard sensor size is actually made of, since the mechanism usually invoked – lateral bypass of the flux around the sensor – turns out to be its smallest part. Three variants of the model isolate the three physical contributions: (i) the full two-dimensional problem with $a = 7$ mm and $\varepsilon_{\text{sensor}} = 0.90$ gives the reference error, -4.6% ; (ii) letting the sensor radius grow to the point where lateral bypass becomes geometrically impossible ($a = 39$ mm, the sensor now covers essentially the whole modelled domain) removes the bypass contribution alone, leaving -3.8% ; (iii) further setting $\varepsilon_{\text{sensor}} = 0.95$, equal to skin, in this bypass-free configuration isolates the contribution of the series thermal resistance of the sensor stack on its own, giving -2.3% . Subtracting successively:

Table 3a

Decomposition of the methodical error at $a = 7$ mm, ideal contact

Contribution	Magnitude, pp	Share of total
Series thermal resistance of the sensor stack	-2.3	51 %
Emissivity mismatch of the top face (0.90 vs 0.95)	-1.5	32 %
Lateral bypass around the sensor edge	-0.8	17 %
Total	-4.6	100 %

The bypass contribution is confirmed independently by the energy balance of § 4.1: only about a quarter of the flux deficit under the sensor re-emerges through the surrounding skin, and that quarter is precisely of the same order as the -0.8 pp found here. The practical implication is favourable: over half of the error is attributable to the sensor's own thermal design (a thinner stack, at the cost of sensitivity, which is proportional to the same resistance) and about a third to a coating specification - matching the sensor's top-face emissivity to that of skin - rather than to an unavoidable geometric limitation of contact measurement. Applying the emissivity correction alone, without any change to the sensor's geometry, would reduce the headline error from -4.6 % to about -2.8 %.

Sensor size (Fig. 5, a). The total error is negative and decreases in magnitude with growing radius: from -5.1 % at $a = 2$ mm to -4.2 % at $a = 14$ mm; for the standard sensor ($a \approx 7$ mm) it is -4.6 %. Given the decomposition above, this weak dependence has a correspondingly modest physical origin: the bypass contribution (the only one of the three that depends on a) itself falls from about -1.3 pp at $a = 2$ mm to about -0.4 pp at $a = 14$ mm, while the stack-resistance and emissivity contributions stay essentially constant, since they do not depend on the sensor's lateral size. The practical consequence remains: reducing the sensor to dimensions of about 4×4 mm – which is of interest for paediatrics and for curved surfaces – raises the error by less than 1 pp, so miniaturization encounters no fundamental metrological obstacle.

Quality of the thermal contact (Fig. 5, b). The error grows almost linearly with the additional contact resistance: from -4.6 % for an ideal contact to -12.5 % at $R_c = 8 \cdot 10^{-3}$ m²·K/W, which corresponds to an air gap of about 0.2 mm. This is the strongest design-and-procedure factor affecting the accuracy. It substantiates the requirements of the measurement procedure: elastic fixation of the sensor with uniform pressure, without excessive force (which would itself alter the skin perfusion), and the use of a thin layer of contact gel where necessary. It should be stressed that individual calibration of the sensor on a bench [13] compensates only the intrinsic resistance of the sensor stack and not the variable contact resistance, so the quality of contact must be monitored during every measurement – which can be done algorithmically, from the stability of the signal.

Air movement (Fig. 5, c). In the range $h = 5 \dots 25$ W/(m²·K) the error varies only between -4.4 and -5.2 %; the values of q_0 and q_{meas} themselves, however, change almost twofold. Draughts and forced ventilation therefore distort not so much the measurement error as the diagnostic interpretation of the absolute values, which confirms the requirement to perform measurements in a room without draughts.

Ambient temperature (Fig. 5, d). Both the undisturbed and the measured fluxes fall almost linearly with rising T_a , with a coefficient of about 0.9 (mW/cm²)/°C: from 17.0 mW/cm² at 18 °C to 10.0 mW/cm² at 26 °C. An uncontrolled variation of the room temperature by ± 2 °C therefore changes the measured flux by ± 13 %, which exceeds the diagnostic asymmetry threshold. This quantitatively substantiates the standardization of conditions (22 ± 2 °C) together with the obligatory recording of the actual room temperature and, above all, the use of relative diagnostic criteria - the asymmetry of symmetric regions measured simultaneously

under identical conditions, which is far more robust to variations of T_a .

The overall estimate of the total methodical error under standardized conditions and with satisfactory contact is about 5 %, which agrees with the passport error of heat flux measurement of the developed instrument (3–5 % [1, 11]) and is acceptable for clinical applications with a diagnostic threshold of 10 %.

4.3. Transient process after sensor placement

Transient modelling (Fig. 6) reproduces the beginning of a measurement: a sensor at room temperature (22 °C) is placed on skin having a steady temperature profile. The sensor’s own thermal mass is included explicitly in the transient model as part of the one-dimensional layer stack, each layer contributing its own ρc to the time-derivative term of (9); the areal heat capacities are 0.15, 1.72, 2.72 and 1.72 kJ/(m²·K) for the contact layer, the lower ceramic plate, the thermopile and the upper ceramic plate respectively, totalling 6.3 kJ/(m²·K) for the whole stack. This is comparable to the heat capacity of the top few millimetres of tissue and is what sets the scale of the settling time found below; a sensor built with thinner ceramic plates would settle correspondingly faster, at the cost of the mechanical robustness the plates provide.

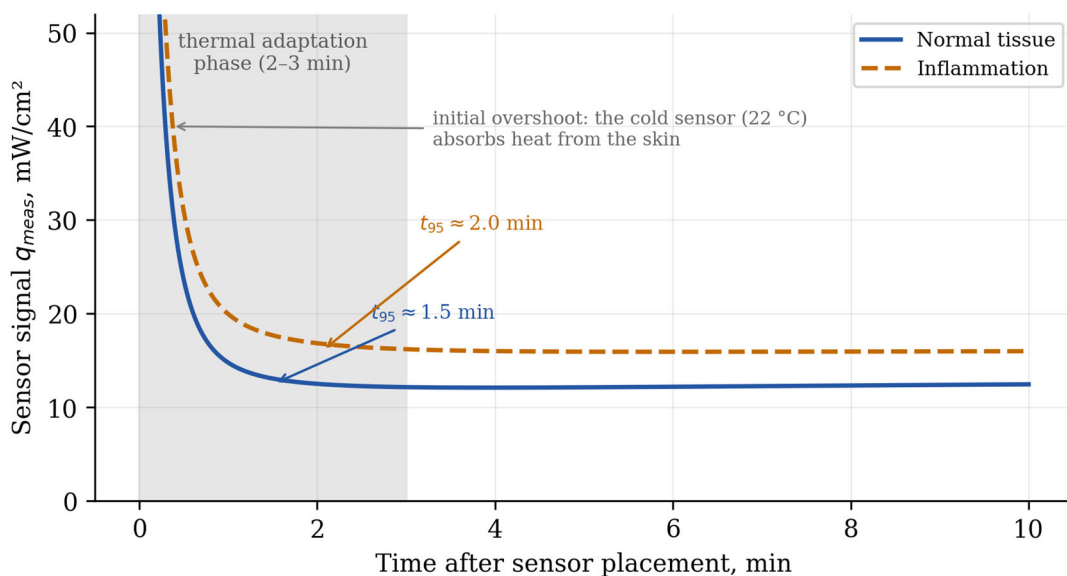


Fig. 6. Transient process of a thermoelectric sensor after placement on the skin

In the first seconds the cold sensor absorbs heat from the skin intensively and the signal exceeds the steady value many times over; an exponential settling then follows. The settling time to within 5 % is $t_{95} \approx 1.5$ min for normal tissue and about 2.0 min for inflamed tissue. This result substantiates physically a requirement that had previously been introduced empirically [1]: a thermal adaptation phase of 2–3 min is both necessary and sufficient - earlier readings are systematically overestimated, and further waiting does not improve the accuracy. For an instrument with a registration rate of 2 measurements per second this corresponds to discarding the first 240–360 readings of each channel.

4.4. Limits of detectability

The practically most important question for clinical application is what size of focus, and at what depth, the method is able to detect. A series of 35 two-dimensional computations was performed in which the radius of the pathological focus was varied between 3 and 18 mm and the depth of its upper boundary between 1 and 20 mm, at a fixed focus thickness of 6 mm and an activity corresponding to $\tau_d = 100$ days (Fig. 7).

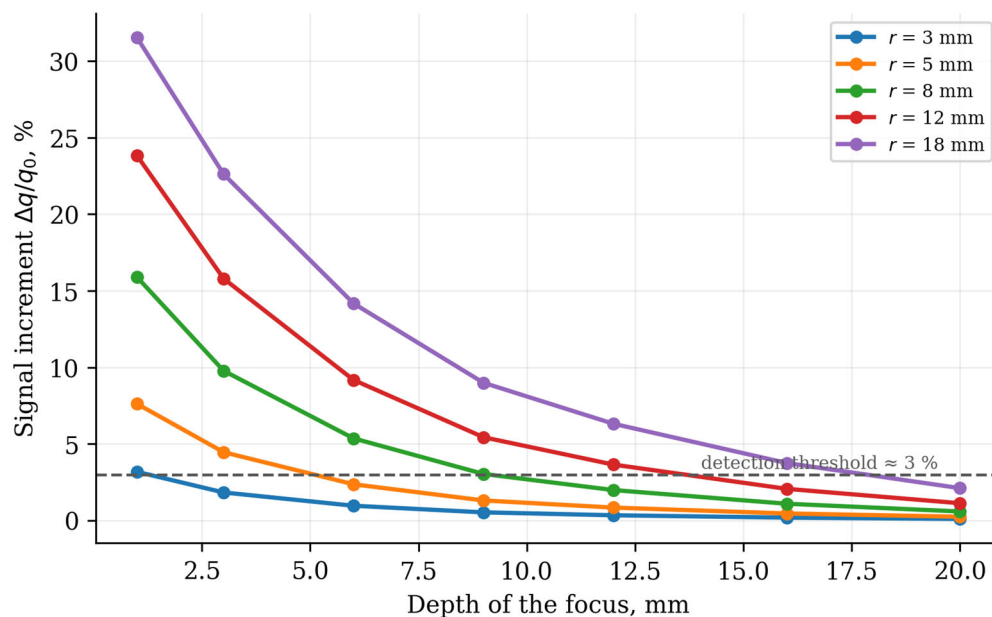


Fig. 7. Detectability of a pathological focus as a function of its size and depth

Taking as the threshold of confident registration an increment of 3 %, which corresponds to the passport error of the instrument with some margin, the following holds. For a focus of radius 8 mm the signal decreases from 15.9 % at a depth of 1 mm to 3.0 % at 9 mm and to 0.6 % at 20 mm; the decay is not a single exponential, its characteristic depth lengthening somewhat with depth (about 4 mm over the shallowest interval, widening to about 7 mm at depth), so the sensitivity falls off a little more gently in the deep tissue than near the surface. Physically this reflects the diffusive spreading of the thermal disturbance and the shunting action of perfusion, which carries the excess heat away with the blood flow and prevents it from reaching the surface. There is thus a clear boundary of detectability in the “size–depth” coordinates: small foci ($r \leq 3$ mm) are detected only in the most superficial layer (down to about 1–2 mm); foci of medium size ($r = 8$ mm) down to a depth of about 9 mm; large ones ($r = 18$ mm) down to about 16 mm.

In practice this means that the method is effective for near-surface processes – inflammations of soft tissues, trophic disorders of the skin and subcutaneous tissue, superficial neoplasms – and is fundamentally limited in its sensitivity to deeply located foci, for which tomographic methods are required. It also permits a correct interpretation of a negative result: the absence of a thermal anomaly does not exclude pathology but only restricts its possible parameters to the region below the detectability boundary, and this qualification should be reflected in the formulation of the conclusion of a heat flux examination.

5. Consequences for measurement practice

The results obtained allow the requirements to the procedure of medical measurements to be formulated in a physically substantiated way, each provision following from a specific quantitative result.

Standardization of conditions: room temperature 22 ± 2 °C with recording of the actual value, relative humidity 40–60 %, absence of draughts (§ 4.2, Fig. 5, c, d).

Patient adaptation: at rest for not less than 15 min before the measurement, to establish thermal equilibrium of the body with the environment.

Sensor placement: symmetric positioning on the region under study and on the contralateral one; elastic fixation with uniform contact and without excessive pressure; contact gel where necessary (§ 4.2, Fig. 5, b); spacing between adjacent sensors not less than about 20 mm (§ 4.1, Fig. 4).

Thermal adaptation phase: 2–3 min after placement, the readings of this phase being excluded from processing (§ 4.3, Fig. 6).

Main measurement phase: 5–10 min of registration at 2 measurements per second per channel, with monitoring of signal stability (drift of the running mean not exceeding 2 % per minute) as an indicator of the quality of the thermal contact.

Interpretation: the primary criterion is the asymmetry of the readings of symmetric regions, $A = 2|q_1 - q_2|/(q_1 + q_2)$, with a diagnostic threshold of 10 %; it is invariant, by construction, with respect to the external conditions that act equally on both channels of a simultaneous measurement. Comparison of absolute values with reference ranges is a secondary criterion and requires allowance for the actual room temperature, since the individual variability of the thickness of the subcutaneous fat layer alone shifts q_0 from +10 % to –5 % (fat thickness 2 to 15 mm against the 8 mm base case) - an asymmetric range, not a symmetric ± 8 %, because q_0 depends on fat thickness nonlinearly. An increase of the flux in the affected region is characteristic of inflammatory processes, a decrease of ischaemic and destructive processes and of hypoperfused neoplasms; for tumours the sign is determined by the balance of metabolic activity and perfusion (§ 1.4), so heat flux data should be interpreted together with the clinical picture. Two further qualifications limit the use of q_0 as an absolute reference value rather than as one side of a within-patient comparison. First, 13.5 mW/cm² is a locally measured quantity, not a whole-body constant: extrapolated over a representative body surface area of about 1.8 m² it would correspond to some 240 W of dry heat loss, several times the resting metabolic rate, which is physiologically consistent only because the model's Dirichlet boundary at $T_b = 37$ °C represents an effectively unlimited internal heat source rather than a closed whole-body energy balance. Second, the model's constant perfusion coefficients cannot represent the vasoconstrictor response, which is the dominant physiological reaction of bare skin at 22 °C and acts in the same direction, and of comparable size, as the ambient-temperature sensitivity already quantified in § 4.2 (about ± 13 % per ± 2 °C); a measured absolute value therefore reflects the patient's autonomic state at the time of measurement as much as it does the local tissue condition. Both qualifications reinforce, rather than undermine, the priority given above to the asymmetry criterion, which is unaffected by either.

Finally, the model also yields recommendations for the design of the instrument. The thermal resistance of the sensor stack should be minimized, since it determines both the methodical error and the settling time; the lateral dimensions in the range 8–20 mm may be chosen from anatomical and technological considerations, as they affect the error only weakly; the contact unit is the critical element and must provide uniform elastic pressure; and the upper surface of the sensor should be protected from draughts and direct radiative heating, because the conditions of heat removal from it enter directly into the effective heat transfer coefficient U_c of (6).

Conclusions

1. A physical and mathematical model of the complete “multilayer biological tissue – thermoelectric sensor – environment” system has been constructed on the basis of the Pennes bioheat equation. Unlike the models used previously, it includes the measuring instrument itself as a set of thermally resistive layers, which for the first time makes it possible to evaluate quantitatively the perturbation that a contact sensor introduces into the quantity it measures. Pathological states are described by local changes of two physiological parameters only – the volumetric blood perfusion rate and the specific metabolic heat generation.
2. The model was implemented in Python with the NumPy and SciPy libraries by the control volume method (the Thomas algorithm for the one-dimensional problem and a direct sparse solver for the two-dimensional one, 16 800 unknowns) and verified at three independent levels: against an analytical solution for a degenerate case (discrepancy below 10^{-4} %), by grid convergence (below 0.07 %) and by cross-comparison of independent one- and two-dimensional formulations (13.52 against 13.53 mW/cm², below 0.1 %).
3. It has been established that under standardized conditions (22 °C, still air) the undisturbed heat flux density from the skin surface is 13.5 mW/cm² at a surface temperature of 31.8 °C, and that blood perfusion is by far the dominant physiological driver of this flux relative to volumetric metabolism: switching off the perfusion term lowers the flux by 26.7 %, whereas switching off the metabolic term lowers it by only 0.8 %, a ratio of about thirty to one (the remaining flux is conduction from the deep boundary, which is itself maintained by the systemic circulation, so this test if anything understates the physiological role of perfusion). It follows that thermoelectric measurement of heat flux is primarily a method of assessing the state of tissue blood supply, and also that a simplified pure-conduction model is inadmissible for this class of problems, the Pennes equation being the minimally sufficient description.
4. It has been shown that for a pathological region occupying the whole tissue layer under the sensor – an upper-bound, one-dimensional estimate – inflammation raises the surface heat flux by 24 %, a proliferating tumour (by the Gautherie relation) by up to 42 %, and ischaemia lowers it by 20 %. For a realistic, laterally localized focus these figures are smaller and depend on depth: a tumour of 8 mm radius, for instance, raises the flux by 15.9 % at 1 mm depth, falling to 3.0 % at 9 mm. Both the whole-layer and the localized estimates for inflammation and ischaemia clear the clinical threshold of physiological asymmetry (10 %); for a deep or small tumour focus this margin narrows, and detectability should be read from

- the size–depth map of point 7 rather than from the whole-layer figure alone. The sign of the anomaly of an oncological focus is determined by the balance between its metabolic activity and its perfusion, which reconciles within a single model the two types of anomaly observed experimentally – increased heat generation of proliferating tumours and reduced heat generation of foci with a necrotic core.
5. Heat flux measurement has been shown to be substantially more sensitive to the pathological changes studied than surface thermometry. The comparison “so many degrees against so many per cent” is not by itself physically meaningful, since it depends on the arbitrary choice of temperature scale (2.3 °C is 0.75 % of the absolute temperature, not the 7 % obtained by using the Celsius reading); the meaningful comparison is each modality’s signal against its own noise floor. On that basis, the temperature changes produced by the pathologies studied (1.9–2.3 °C) are twenty to fifty times the resolution of clinical thermography, whereas the heat flux changes (20–24 %) are five to eight times the sensor’s own passport error (3–5 %) – both are comfortably detectable, and the flux channel’s advantage lies not in a raw multiple but in its insensitivity to the ambient-radiative and surface-emissivity artefacts that dominate the error budget of thermometry, and in the physiological, not instrumental, character of the 10 % asymmetry threshold that ultimately limits both modalities equally.
 6. The methodical error of contact measurement has been determined for the first time and, for a sensor of 14×14×3 mm under ideal thermal contact, amounts to –4.6 %. Decomposing it shows that lateral bypass of the flux around the sensor – the effect usually invoked to explain such an error – is in fact its smallest contributor (about one sixth of the total, –0.8 percentage points out of –4.6, confirmed independently by an energy balance over the whole modelled surface); about half of the error (–2.3 pp) is the series thermal resistance of the sensor’s own layer stack, and roughly a third (–1.5 pp) is the emissivity mismatch between the sensor’s top face (0.90) and skin (0.95) – a coating specification, not a metrological limit, correction of which alone would reduce the headline error to about –2.8 %. The most critical *procedural* factor remains the thermal resistance of the “skin–sensor” contact, which increases the error to –12.5 % for poor contact; the sensor size affects it only weakly (–5.1 to –4.2 % over the range 2–14 mm), so that miniaturization of the sensor encounters no fundamental metrological obstacle. The strongest external factor is the ambient temperature, about 0.9 (mW/cm²)/°C.
 7. The duration of the transient process after the placement of the sensor on the skin has been determined ($t_{95} \approx 1.5\text{--}2$ min), which substantiates physically a thermal adaptation phase of 2–3 min in the measurement procedure, and a map of the detectability of pathological foci in the “size–depth” coordinates has been constructed: at a detection threshold of 3 % a focus of radius 8 mm is detected down to a depth of about 9 mm and one of radius 18 mm down to about 16 mm, the depth sensitivity decaying with a characteristic depth of 4–7 mm (increasing somewhat with depth, rather than as a single exponential), which defines the method as effective for near-surface processes.
 8. On the basis of the results obtained, physically substantiated requirements have been formulated for the design of thermoelectric heat flux sensors and for the procedure of

medical measurements, including the standardization of conditions, the duration of the adaptation and main phases, the spacing between the sensors of a multichannel instrument and the priority of the asymmetry criterion over the comparison of absolute values.

Taken together, the results establish the physical basis of thermoelectric heat flux diagnostics of inflammatory and destructive processes and define the limits of its applicability. The next stage of the work is the quantitative validation of the model against controlled measurements with the developed multichannel heat meter and its extension to three-dimensional geometries of regions of high curvature.

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**Моделювання теплопереносу в системі
«Біологічна тканина – Термоелектричний сенсор – Середовище»
для діагностики запальних та деструктивних процесів**

У роботі представлено фізичну та математичну модель повної вимірювальної системи, утвореної багатошаровою біологічною тканиною, контактним термоелектричним сенсором густини теплового потоку та навколишнім середовищем. Модель ґрунтується на рівнянні біотеплопереносу Пеннеса, яке враховує кровоперфузію та метаболічне тепловиділення, і включає сам сенсор як сукупність термічно резистивних шарів, що вперше дає змогу кількісно оцінити збурення, яке прилад вносить у вимірювану ним величину. Патологічні стани описано локальними змінами лише двох фізіологічних параметрів: об'ємної швидкості перфузії крові та питомого метаболічного тепловиділення. Крайові задачі розв'язано методом контрольних об'ємів, реалізованим мовою Python із застосуванням наукових бібліотек NumPy та SciPy; реалізацію верифіковано за аналітичним розв'язком, збіжністю за сіткою та перехресним порівнянням незалежних одновимірної і двовимірної постановок. Показано, що за стандартизованих умов незбурена густина теплового потоку з поверхні шкіри становить 13.5 мВт/см^2 , і що кровоперфузія є домінантним фізіологічним чинником цього потоку – приблизно у тридцять разів впливовішим за об'ємне метаболічне тепловиділення (26.7 % проти 0.8 % величини потоку, тест вимкнення), тож модель чистої теплопровідності є неприйнятною для задач цього класу. Для патологічної ділянки, що охоплює всю площу ураженого шару під сенсором, потік змінюється на +24 % (запалення), +42 % (проліферуюча пухлина, співвідношення Готьє) і –20 % (ішемія); для реалістичного локалізованого вогнища радіусом 8 мм сигнал пухлини менший і залежить від глибини – від +15.9 % на глибині 1 мм до +3.0 % на глибині 9 мм. Показано, що вимірювання теплового потоку помітно чутливіше до цих патологічних змін, ніж поверхнева термометрія, корисний сигнал якої (1.9–2.3 °C) лише у кілька разів перевищує роздільну здатність клінічної термографії, тоді як наведені зміни потоку – у п'ять–вісім разів більші за паспортну похибку самого приладу. Методична похибка контактного сенсора $14 \times 14 \times 3 \text{ мм}$ – зумовлена порівнянною мірою тепловим опором його власного пакета шарів, розбіжністю ступеня чорноти його верхньої грані й шкіри та, найменшою мірою, обтіканням теплового потоку навколо сенсора – становить –4.6 % за ідеального теплового контакту і зростає до –12.5 % за незадовільного контакту; час встановлення показів сенсора становить 1.5–2 хв. Одержані результати визначають фізичні межі термоелектричної теплотеметричної діагностики та обґрунтовують вимоги до конструкції сенсорів і методики вимірювань.

Ключові слова: термоелектрика, густина теплового потоку, термоелектричний сенсор, телурид вісмуту, рівняння Пеннеса, кровоперфузія, комп'ютерне моделювання, метод контрольних об'ємів, медична діагностика, запалення, пухлина, ішемія.