

Thermal Effects of Bioimplants

© IMAGE SOURCE, PHOTO DISC, EYEWIRE Power Dissipation Characteristics and Computational Methods

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s biomedical implants increase in complexity and functionality, we are beginning to face an entirely new challenge: Is the thermal increase in the human body caused by operation of the implant significant, and, if so, what are its implications? While this may not have been a significant problem in the past, when implanted devices were characterized by basic functionality, we are now reaching the stage of considering implantable systems that enjoy high data rate wireless communications with external devices, stimulate the human body with thousands of channels at high repetition rates, possess sensor capabilities to inform us of the status of the implant, and are extremely small. Regardless of whether we can technologically fulfill some or all of these desired engineering features of the next generation of implantable systems, there is a growing concern that such complex devices may generate enough heat to limit their actual functionality and characteristics. Implantable devices are well on the way to becoming small, dedicated, and highly complex embedded systems. As such, they are plagued by the same thermal management problems that afflict the computer industry: increased functionality causes increased heat generation. And once the heat is generated, it is not easy to dissipate effectively.

The Problem

An implantable device with very limited electronics and limited communication with the external world is rarely a problem from the thermal point of view. The operations performed by the device are limited enough so that no significant or noticeable heat is generated. Similarly, some devices have complex electronics but are silent most of the time. In such cases, the limited operations performed by the implant do not generally cause enough power dissipation to result in a thermal increase in the surrounding tissue.

When, instead, we are dealing with an implant that constantly stimulates the human body and its neural tissues with a large number of electrodes and is in continuous communication with the external world, heat dissipation could become an important element of the design. Clearly, there are numerous parameters that affect the thermal increase associated with the operation of an implantable device; such parameters include the current requirements of the stimulating electrodes, the number of electrodes, the power requirements of the implanted electronics, and the characteristics of the telemetry.

Herein, we consider the effects of various parameters on the temperature increase in the human body tissue, with a focus on a specific proposed implant: a dual-unit retinal prosthesis to restore partial vision to the blind. This particular example is educative since it includes most of the potential causes of thermal dissipation: a microchip that could dissipate relatively large power, a telemetry system, and a potentially large number of stimulating electrodes.

A Closer Look at the Causes of Temperature Increase

Figure 1 shows a schematic representation of the potential causes of temperature increase in the tissue caused by the operation of an implantable device and, in particular, of a dual-unit retinal prosthesis system. As noted previously, the power dissipated by the implanted electronics (microchip, telemetry coil, and stimulating electrodes) contributes to the potential temperature increase in the surrounding tissues. Electromagnetic fields induced in the human body must be also accounted for in the design of the telemetry system.

In the following, we will consider with somewhat more detail these various origins of temperature increases and possible methods to compute or measure them.

Electromagnetic Fields Caused by the Telemetry Systems

If the implantable device uses a telemetry system to transmit power and data, electromagnetic fields impinging on the human body could lead to dissipated power in the tissue and, in turn, to temperature increase. Once again, this is generally not of concern for a low-power device with minimal functionality. If, however, the implanted electronics consume significant power (where the meaning of the term *significant* depends on a number of factors to be clarified later), substantial electromagnetic fields could be induced in the human body. Most implantable devices are powered by low-frequency (<1 MHz signal) magnetically coupled coils (often modulated to include the data telemetry), where the exact frequency results from the joint design for the optimization of transmit and receive circuits and from geometrical constraints. Below the frequency of 1 MHz, induced conduction currents in the human body should be evaluated to ensure that potentially hazardous field and current levels are not reached, as indicated by some electromagnetic safety standards, such as [1]. At frequencies higher than 100 kHz (which are used in most telemetry applications), most international standards for electromagnetic safety identify limits of power dissipated in the human body in terms of specific absorption rate (SAR), expressed in W/kg or the generally more restrictive maximum permissible exposure (MPE) for current and fields. This is, in essence, the power dissipated per unit mass of tissue. Mathematically, at any point in the human body, the SAR for a sinusoidal excitation is expressed as

SAR(x, y, z) =
$$\frac{\sigma(x, y, z)E^2(x, y, z)}{2\rho(x, y, x)}$$
, (1)

where ρ is the tissue density (in kg/m³), σ is the conductivity (S/m), and *E* is the electric field amplitude (V/m) at point of coordinates *x*, *y*, and *z*.

Excellent reviews have been published summarizing the rationale behind the notion of SAR and limits considered safe for human exposure. Generally, above the frequency of 100 kHz (and below the frequency of 6 GHz), SAR limits are expressed in terms of SAR averaged over any 1 g of tissue in the shape of a cube and whole-body averaged SAR. For example, the IEEE standard prescribes an acceptable maximum 1 g average SAR of 1.6 W/kg for the general population. Medical devices do not necessarily need to comply

with these recommended values of exposure (as clearly indicated in the scope of [1]). However, it should be noted that devices that are expected to function on a continuous basis, such as the retinal prosthesis, are to be used by the patient in a variety of settings and situations. This means, for example, that other individuals in close proximity to the patient are also exposed to the electromagnetic fields radiated by the telemetry devices. It is, therefore, highly desirable that telemetry devices used for implantable devices meant for everyday use by the patient meet the recommended levels of radiation, such as those set by the IEEE.

In the human body, SAR can be determined using numerical techniques or experimental methods using phantoms. One of the most diffused numerical methods for this purpose is the finite-difference time-domain method (FDTD)[2], which has been widely used over the past 15 years for numerous dosimetric problems along with computational models of the human body derived from MRI scans of volunteers. Human body models of resolutions lower than 1 mm have been developed for use in conjunction with the FDTD method.

Figure 2 shows a rendering of a portion of a 0.25-mm resolution model of the human head (derived from a 1-mm resolution model), classified into more than 20 different tissue types, that has been used to determine the field induced by telemetry devices in a dual-unit retinal prosthesis system [3]. Figure 3 shows an example of the SAR results obtained when a 2-mm resolution head model is illuminated by an external coil operating at the frequency of 2 MHz with a current of 2 A flowing through it and positioned in front of the



Fig. 1. A schematic of potential causes of temperature increase in the human body associated with the operation of implantable devices.

eye, ideally on a pair of eyeglasses. In this figure, tissues are transparent while the SAR on the face of the model is opaque, with a color scale ranging from red (highest SAR values) to blue (lowest SAR value). The peak, 1-g SAR in this case, was considerably lower than 1.6 W/kg [3]–[5].

Alternatively, one could use human body phantoms filled with tissue-simulating materials to measure the induced electric fields by means of miniature probes and, therefore, determine the SAR. The advantage of the experimental method to determine the SAR is that the radiating device used for the experiments is the actual device that one intends to use rather than a model. Using the actual device eliminates any uncertainty about the accuracy in the modeling of the radiating system. However, the disadvantages of such an experimental method reside in the fact that only a very simple model of the human body can be used, often homogeneous; of course, calibrated equipment must be maintained (tissue simulant fluids of constant dielectric properties and calibrated probe characteristics).

The complexity of the SAR determination increases with the complexity of the telemetry system to be used. In applications such as the retinal prosthesis, it might be interesting to use a dual-frequency system, with inductive coupling at a frequency lower than 1 MHz for powering the system and a higher frequency (inductive coupling at tens of megahertz or antennas in the gigahertz region) for the data telemetry to satisfy the need of bandwidth to control the stimulating electrodes. In this case, SAR must be assessed at both frequency bands.

Power Dissipated by the Implanted Electronics

In addition to the issue of electromagnetic fields induced in the human body, it is important to consider whether the current induced in the implanted coil can cause a significant power dissipation in the coil itself (because of the finite resistivity of the coil) and whether this, in turn, could cause a temperature increase in the surrounding tissues. This question can be answered, once again, using numerical or experimental methods. As with the electromagnetic simulations, numerical methods involve using an anatomically correct, discretized, human body model with known thermal properties of various tissues along with a numerical code to solve the bioheat equation [3]:

$$\underbrace{C\rho \frac{\partial T}{\partial t}}_{\text{Thermal}} = \underbrace{\nabla \cdot (K\nabla T)}_{\text{Thermal Spatial}} + \underbrace{A_0}_{\text{Tissue}} - \underbrace{B_0 (T - T_B)}_{\text{Blood Perfusion}} + \underbrace{\rho \text{SAR} + P_{\text{Electronics}}^{\text{Density}}}_{\text{External Heat Sources}} \left[\frac{W}{m^3}\right], \quad (2)$$

where T is temperature (°C), C is specific heat [J/ (kg °C)], ρ is tissue density (kg/m³), K is thermal conductivity [J / (m s °C)], A₀ is the basic metabolic rate [J / (m³ s)], B₀ is the blood perfusion coefficient [J / (m³ s °C)], T_B is the temperature of blood (°C), and $P_{\text{Electronics}}^{\text{Density}}$ is the power dissipated by the implanted electronics (W/m³). Note that while the SAR is a source of heat dissipation and is accounted for in (2), it may be neglected if its values are lower than values recommended in international standards. In this case, the thermal impact of the SAR is minimal.

Heat exchange at the skin can be modeled by the following boundary condition:

$$\underbrace{X \frac{\partial T_{(x,y,z)}}{\partial n}}_{\substack{\text{Temperature} \\ \text{Chance}}} = -\underbrace{H_a \left(T_{(x,y,z)} - T_{\text{Environment}} \right)}_{\text{Convective Heat Exchange}}, \quad (3)$$

where H_a is the convective transfer coefficient [J/(m² s °C)] and $T_{\text{Environment}}$ is the temperature of the external environment (°C).



Fig. 2. A human head computational model used for the numerical evaluation of the electromagnetic absorption caused by telemetry devices (from (3)).



Fig. 3. An example of the SAR results for an external telemetry coil positioned in front of the left eye of a computational human head model.

The temperature dependence of metabolic processes in tissues can be modeled as [6]

$$A(\vec{r}, T(\vec{r})) = \underbrace{A_0(\vec{r}, T(\vec{r}))(1.1)^{(T(\vec{r}) - T_0(\vec{r}))}}_{\text{Temperature Dependence of Tissue Metabolism}},$$
(4)

where T_0 is the basal temperature (°C).

The temperature dependence of the blood perfusion in the inner tissues can be represented by

$$\begin{cases} B(\vec{r}, T(\vec{r})) = B_0(\vec{r}) & \text{for } T(\vec{r}) \le 39^{\circ} C \\ B(\vec{r}, T(\vec{r})) = B_0(\vec{r}) & \\ \times [1 + S_B(T(\vec{r}) - 39)] & \text{for } 39^{\circ} C \le T(\vec{r}) \le 44^{\circ} C \\ B(\vec{r}, T(\vec{r})) = B_0(\vec{r})(1 + 5S_B) & \text{for } T(\vec{r}) \ge 44^{\circ} C \end{cases}$$
(5)

while that of the skin can be expressed by

$$B(\vec{r}, T(\vec{r})) = [B_0(\vec{r}) + \underbrace{F_{SB}\overline{\Delta T_S}}_{\text{Weighted Skin}}] \cdot 2^{\frac{\left(T(\vec{r}) - T_0(\vec{r})\right)}{6}}, \quad (6)$$

where F_{SB} is weight coefficient for the skin temperature signals, $\overline{\Delta T_S}$ is the average temperature increase of the skin, and S_B is a coefficient set at a value of 0.8 °C⁻¹.

Several methods can be used to solve the bioheat equation (2) with boundary condition (3) and temperature dependences such as (4)-(6). The simplest method is based on an explicit finite-difference solution that leads to a time-marching algorithm [3], [4]. Alternatively, one can use unconditionally stable methods to reduce the computational time. Numerical methods for solving the bioheat equation are extremely attractive because they do not involve the complexities associated with in vivo experiments that are otherwise needed for an experimental approach. While experiments with phantoms similar to those for the SAR assessment can also be performed, their results must be interpreted with caution. In fact, phantom models lack two critical components that affect the heat distribution and rate of temperature increase: blood perfusion and tissue metabolism. In circumstances such as the retinal prosthesis, the main organ affected is the vitreous humor, which has no blood perfusion. In this case, phantoms could prove useful, even though they provide limited results.

Table 1 provides the dielectric and thermal properties (at the hypothetical frequency of operation of the telemetry system of 2 MHz) as reported in [3] and collected from various sources. Dielectric properties at different frequencies are available from numerous sources [7], [8]. Assumptions made to justify some of the properties not readily available can also be found in [4].

Table 1. Dielectric and thermal properties of the human body model used for computational evaluation of the SAR and temperature increases. Dielectric properties are provided for the frequency of operation of the telemetry system of 2 MHz.

Tissue	Relative Permitivity ε_r	Conduct- ivity σ (S/m)	Mass Density ρ(kg/m³)	Specific Heat C (J/(kg °C))	Thermal Conductivity K (J/(m s °C))	Blood Perfusion B (J/(m ³ s °C))	Metabolic Rate A ₀ (J/(m ³ s))
Muscle	826	0.5476	1,040	3,600	0.498	2,700	690
Deep Fat	22.95	0.0255	920	2,500	0.250	520	180
Bone	106	0.0285	1,810	1,300	0.300	1,000	0
Cartilage	815.5	0.2776	1,100	3,400	0.450	9,100	1,000
Skin	858	0.0371	1,010	3,500	0.420	9,100	1,000
Subcutaneous Fat	22.95	0.0255	920	2,500	0.250	520	180
Gray Matter	656.5	0.1807	1,039	3,680	0.565	35,000	10,000
White Matter	340.6	0.1118	1,043	3,600	0.503	35,000	10,000
Blood	1,681	0.9261	1,060	3,840	0.530		0
Sclera	1,145	0.6889	1,170	4,178	0.580	0	0
Scleral Muscle	826	0.5476	1,040	3,430	0.498	2,700	690
Cornea	1,429	0.7438	1,076	4,178	0.580	0	0
Pupillary Muscle	826	0.5476	1,040	3,430	0.498	2,700	690
Postchamber	76.65	1.5010	1,003	3,997	0.578	0	0
Lens	829.7	0.4170	1,100	3,000	0.400	0	0
Ciliary Muscle	826	0.5476	1,040	3,430	0.498	2,700	690
Vitreous Humor	76.65	1.5010	1,009	3,997	0.594	0	0
Retina	1,145	0.6889	1,039	3,680	0.565	35,000	10,000

Results: The Example of the Dual-Unit Retinal Prosthesis

As mentioned previously, the dual-unit retinal prosthesis is educative since it requires close attention to the possible causes of thermal increase in the human tissue. The electromagnetic analysis can be performed using a full-wave numerical electromagnetic method or an experimental method. One can then determine, based on SAR values of the particular telemetry system to be adopted, whether to compute the thermal increase caused by electromagnetic radiation. Determining the temperature increase caused by implanted electronics requires assessing the impact of power dissipation in the implanted telemetry coil, microchip, and stimulating electrode array.

Using a numerical method based on the finite-difference scheme, we have computed the heat induced in the human eye by a coil of finite resistivity implanted in place of the lens for the dual-unit retinal prosthesis. In this example, the coil is 7 mm in diameter (with a wire of 0.25-mm diameter), has four turns, and is made of gold of resistivity $\rho = 2.44 \ 10^{-8} \ \Omega$ -m, density = 19,300 kg/m³, specific heat $C = 128 \ J/(kg \ K)$, and thermal conductivity $k = 60 \ W/(m \ K)$. The coil is covered with Teflon-like insulator of density = 3,890 kg/m³, specific heat C = 880 J/(kg \ K), and thermal conductivity $k = 30 \ W/(m \ K)$. Three possible current levels flowing in the implanted coil have been considered: 1) $I = 70 \ mA$ (total dissipated power $P_d = 215 \ \mu \ W$), 2) $I = 110 \ mA$ (total dissipated power $P_d = 546 \ \mu \ W$), and 3) $I = 150 \ mA$ (total dissipated power $P_d = 984 \ \mu \ W$).

Figure 4(a) shows a horizontal cross section of the numerical head model through the center of the eyeball, indicating



Fig. 4. (a) The horizontal cross section through the center of the eye of the numerical head model showing the position of the implanted coil and (b) a graphical representation of the temperature increase in the same cross section for a coil dissipating 984μ W.



Fig. 5. (a) The time variation of the temperature increase in the ciliary muscle and the cornea for three implanted coils carrying 70, 110, and 150 mA, respectively, and (b) the maximum temperature increase in the ciliary muscle and the cornea as a function of the current flowing in the coil.

the position of the coil, while Figure 4(b) shows that the maximum temperature rise takes place on the ciliary muscle right next to the coil surface. This rise is lower than 0.5 °C in the worst case of power dissipated in the coil by ohmic losses $P_d = 984 \ \mu$ W. The temperature variation as a function of time for both the ciliary muscle and the cornea is shown in Figure 5(a) for the three considered cases, while the steady-state temperature variation for these three cases as a function of the input current is plotted in Figure 5(b). The temperature increase in other organs for the worst case of dissipated power $P_d = 984 \ \mu$ W has been computed, and results for the maximum temperature increase in each organ are shown in Table 2. These results suggest that the impact of the current flowing through the implanted coil should be considered when assessing the temperature increase in the tissue.

The implanted microchip could be the major cause of a temperature increase in the tissue as a result of implantable devices that continuously stimulate the tissue with many stimulating electrodes. Devices that operate only intermittently are not generally affected by this problem since, to generate noticeable heat dissipation in the surrounding tissue, the microchip needs to operate continuously for at least a few minutes.

We have computed, under different operating conditions, the temperature increase caused by the implanted microchip in the dual-unit retinal prosthesis example; a detailed study of this is presented in [3]. Figure 6(a) shows the temperature increase with respect to the basal temperature in the horizontal cross section of the human body model through the center of the eye, induced by a chip positioned in the center of the eyeball. The power dissipated by the chip is 12.4 mW and the chip is $4 \times 4 \times 0.5$ mm, coated with an insulating material. This chip induces a maximum temperature increase on the surface of the chip of approximately .8 °C with respect to the computed basal temperature. The same chip dissipating 49.6 mW causes a temperature increase on the surface of the chip of approximately 2.9 °C with respect to the basal temperature [Figure 6(b)].



Fig. 6. The temperature increase with respect to the basal temperature in the horizontal cross section of the human body model through the center of the eye, induced by a chip positioned in the center of the eye for a dual-unit retinal prosthesis system. (a) A chip of size $4 \times 4 \times 0.5$ mm dissipating 12.4 mW and (b) a chip of size $4 \times 4 \times 0.5$ mm dissipating 49.6 mW.

Table 2. The temperature rise in various human organs caused by a 7-mm-diameter implanted coil in place of the lens dissipating 984 μ W.

Tissue	Maximum Temperature Rise (° C)		
Retina	0.025		
Skin	0.089		
Fat	0.152		
Bone	0.018		
Brain-Gray	< 0.001		
Brain-White	< 0.001		
Mucous Membrane	0.002		
Ciliary	0.415		
Cornea	0.240		
CSF	<0.001		



Fig. 7. The temperature increase with respect to the basal temperature in the horizontal cross section of the human body model through the center of the eye, induced by a chip positioned in place of the lens for a dual-unit retinal prosthesis system. A chip size of $4 \times 4 \times 0.5$ mm dissipating 12.4 mW.

Numerous parameters affect the actual temperature increase besides the total dissipated power. For example, the location of the chip within the body has a significant impact. Other important factors are the uniformity of power dissipation within the chip, materials used for the insulation, and the size of the chip. Figure 7 shows the temperature increase with respect to the basal temperature in the horizontal cross section of the human body model through the center of the eye, induced by a chip positioned in the anterior chamber of the eye in place of the human lens. As in the previous cases, the chip dimensions are $4 \times 4 \times 0.5$ mm, and the chip is coated with insulating material. The chip dissipates 12.4 mW. As with the chip positioned in the center of the eye [Figure 6(a)], the maximum temperature increase on the surface of the chip is approximately 0.8 °C. However, while the chip implanted in the center of the eye induces a temperature increase on the retina of approximately 0.13 °C, the chip implanted in place of the lens causes a temperature increase of about half of that, or 0.052 °C.

A similar analysis can be performed for electrodes stimulating neural tissues. The power flowing through the electrodes leads to power dissipation that can be included in (2), as in the previous cases. The complete thermal analysis will then include all the effects presented above. This provides an accurate pattern of the temperature increase in the tissue under a variety of conditions and, therefore, improves the design specifications of implantable devices.

Concluding Remarks

Without question, the power dissipation characteristics of implanted electronic systems will have increasing importance for the design of future implantable devices. As shown in this article, in some cases, this power dissipation can lead to temperature increases in the human tissue that are not negligible. In particular, the design of implantable stimulating devices with a large number of stimulating channels must be performed with a clear idea of the potential thermal implications of the device. Fortunately, reliable numerical and experimental methods are available to characterize the temperature increase caused by the implantable device. These methods should be used during the design phase of these devices.

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