CHAPTER-II

A THEORETICAL MODEL FOR STUDYING THE HEAT TRANSFER BETWEEN CORE AND SKIN.

2.1. INTRODUCTION

The temperature distribution in working muscles has been studied experimentally by many authors [Menshih (1969), Saltin, Jagge and Stolwijk (1968), Stow and Shieve (1970), Zavyalov (1969)] Hill (1963) found that during the action of isolated muscles of warm and cold blood animals their temperature dynamics showed a formal resemblance with that observed for a number of internal organs. The study and modelling of bio-heat transfer process cover diverse aspects and have received extensive attention [Shitzer (1975), Stalwizk (1971) Chato (1969) Shitzer and Chato (1970)] due to the fact that the maintenance of a homeothermic state is of primary importance to homeotherms and is also one of the most complicated problems. The important role which the skin of the homeotherm plays in thermal regulation has long been recognized. Zhitomirskii and Kagna (1978) studied temperature distribution in a single layered skeletal muscle undergoing rhythmic contractions. They showed that a smoother muscular motion can not cause a substantial
temperature rise as the parameters adjust in such a way as
as to maintain the internal temperature of the muscle in the
permissible range. The thermal energy balance for a muscle
of an animal comprised thermal energy supplied by blood
flow, energy generated by metabolic process, the transport
of heat through the muscle by temperature gradient and energy
transfer to the environment by conduction, radiation and
evaporation. Due to the complex non homogeneous,
non-isotopic nature of tissues a number of simplifying
assumptions have been made to facilitate analysis. The
rate of heat transfer across the skin will depend upon the
tissue composition and thickness of the skin and
subcutaneous tissue. Many studies have been undertaken to
clucidate the thermal behaviour of biological tissues and
the relationship which exist among the various mechanisms,
viz, convection conduction, generation, evaporation and
radiation, governing the transport of heat in the tissue.
Keller and seller (1971) has developed a very simple model
for heat transfer between an isothermal core and ambient
atmosphere. The outermost thickness of skin was considered
to be very small and calculation were made in the absence of
metabolic heat transfer between either arteries or veins and tissue. One of the controls available to the body in the maintenance of a homoisothermic state is its ability to vary resistance of its subcutaneous regions to heat flow. [Torell and Nilson (1980), Steketee & Vander Heek (1979).]

In earlier models an attempt was made to account for the combination of conduction vasomotor activity by a simple addition effect] [Hardy and Soderstrom (1938) and Barton (1934)] Krogh (1957) had identified, definite vascular structure called rates which in certain species appear to function primarily to conserve heat. In these structure arteries and veins are arranged closely packed for counter current flow Mitchell and Myers (1968). It has been observed that the rate of heat loss from an extremity is related to local blood perfusion rate [Burton and Edhalm (1955), Hardy and Soderstrom (1938), Lefever (1911)]. Pennes (1957) improved these models significantly by treating tissue as a continuum of finite thickness and developing the appropriate heat conservation differential equations to describe the tissue temperature profile. Wessler (1966) solved much more complicated problem of the overall heat
FIG. 2.1 SCHEMATIC DIAGRAM OF SUBCUTANEOUS TISSUE REGION EMPHASIZING ITS VASCULARIZATION AND TEMPERATURE VARIATION
balance by such dividing the body into a number of cylindrical regions thermally connected by the circulatory flow. His model also taken conductive and convective effects but he assumes that arterial and venous blood temperature are uniform in each region so that the effect of local counter current heat exchange is lost. Recently Gupta and Tandon (1984) has developed a model for heat transfer between core and skin. They incorporated the effects of locally variable perfusion rate in addition to convective heat transfer and metabolic heat generation depending on temperature. In earlier models, an attempt was made for either no metabolic heat generation rate or constant arterial and venous temperature. The present chapter is a further generalisation of the earlier work by incorporating the effect of temperature on arterial and venous beds, in addition to metabolic heat generation depending on temperature and different atmospheric conditions.

2.2. FORMULATION OF THE PROBLEM

The Schematic diagram of the model is given by Fig 2.1. The peripheral Layer thickness is very small compared to the radius of curvature
of the Local Surface. Since the region is very small therefore we can consider the temperature distribution only in the direction normal to the surface. Hence the system can be considered to be one dimensional. The steady state heat balance equation can be written as follows:

\[ K \frac{d^2 T}{dx^2} + (ha + Cp \, g)(Ta - T) + ha (Tv - T) + M = 0 \]  \hfill (2.2.1)

\[
\left[ m^0_a - \int_0^x g \, dx \right] \left[ Cp \frac{d}{dx} Ta + ha (Ta - T) \right] = 0 \]  \hfill (2.2.2)

\[
Cp \left[ m^0_v + \int_0^x g \, dx \right] \frac{d}{dx} Tv + (ha + Cp \, g) (Tv - T) = 0 \]  \hfill (2.2.3)

Where \( M \) is the metabolic heat generation rate (Cal/cc-sec), \( Tc \) and \( Ts \) are the core and surface temperatures (°C) respectively, \( Ta \) and \( Tv \) are temperature of artery and vein (°C) respectively. \( L \) is the thickness of peripheral Layer and \( ha \) the heat transfer coefficient times area, \( g \) is the tissue perfusion rate in (g/cc-sec), \( m^0_v \) and \( m^0 a \) are the venous and arterial flow rate g/cm²sec, \( cp \) is the specific heat capacity in (Cal/g-°C)

Assuming \( M \) to vary linearly with \( T \)

\[ M = Mo (1 + \alpha T) \]  \hfill (1.2.4)
\[ T = C_1 \cos \frac{\lambda_1}{\lambda_2} x + C_2 \sin \frac{\lambda_1}{\lambda_2} x - \frac{\lambda_1}{\lambda_2} \]  

(2.4.4)

where

\[ C_1 = T_c + \frac{\lambda_1}{\lambda_2} \]

\[ C_2 = \frac{T_s - \left(T_c + \frac{\lambda_2}{\lambda_1}\right) \cos \left(\frac{\lambda_1}{\lambda_2} L\right) + \frac{\lambda_1}{\lambda_2}}{\sin \left(\frac{\lambda_1}{\lambda_2} L\right)} \]

\[ \lambda_1 = \frac{M_o \alpha - C_P g}{K}, \quad \lambda_2 = \frac{C_P g T_a + M_o}{K} \]

substituting the above value of \( T \) from equation (2.4.4) into the equation (2.4.3) and Integrating we get.

\[ T_v = \frac{g}{(m v + g x)} \left[ C_1 \frac{\sin \frac{\lambda_1}{\lambda_2} x}{\frac{\lambda_1}{\lambda_2}} - C_2 \cos \frac{\lambda_1}{\lambda_2} x - \frac{\lambda_1}{\lambda_2} x \right] + C_3 \]  

(2.4.5)

where

\[ C_3 = T_s - \frac{g}{(m v + g L)} \left[ C_1 \frac{\sin \frac{\lambda_1}{\lambda_2} L}{\frac{\lambda_1}{\lambda_2}} - C_2 \cos \frac{\lambda_1}{\lambda_2} L - \frac{\lambda_2 L}{\lambda_1} \right] \]

substituting the value of \( C_3 \) in equation (2.4.5) we get.
\[
Tv = \frac{g}{(m\dot{V} + gx)} \left[ \frac{C_1}{\lambda_1} \left\{ \sin \frac{1}{\lambda_1} x - \sin \frac{1}{\lambda_1} L \right\} - \frac{C_2}{\lambda_2} \left\{ \cos \frac{1}{\lambda_1} x - \cos \frac{1}{\lambda_1} L \right\} - \frac{\lambda_2}{\lambda_1} (x - L) \right] + Ts
\] (2.4.6)

Using the non dimensional scheme

\[
\theta_1 = \frac{T - T_c}{T_s - T_c}, \quad \theta_2 = \frac{Tv - T_c}{T_s - T_c}, \quad X = \frac{x}{L}
\]

we get

\[
\theta_1 = \left( \frac{C_1}{T_s - T_c} \right) \cos \left( \frac{1}{\lambda_1} L X \right) + \frac{C_2}{T_s - T_c} \sin \left( \frac{1}{\lambda_1} L X \right)
\]

\[- \frac{\lambda_2}{\lambda_1 (T_s - T_c)} - \frac{T_c}{T_s - T_c}
\] (2.4.7)

\[
\theta_2 = \frac{g}{(m\dot{V} + gxL)(T_s - T_c)} \left[ \frac{C_1}{\lambda_1} \left\{ \sin \left( \frac{1}{\lambda_1} LX \right) - \sin \frac{1}{\lambda_1} L \right\} - \frac{C_2}{\lambda_1} \left\{ \cos \left( \frac{1}{\lambda_1} LX \right) - \cos \left( \frac{1}{\lambda_1} L \right) \right\} - \frac{\lambda_2}{\lambda_1} (X-1) \right] + 1
\] (2.4.8)
2.5 RESULTS AND DISCUSSIONS:

The following values of physical and Physiological parameters along with the table (2.1) are used for obtaining temperature profile in the region under study.

\[ K = 0.499 \text{ J/mSec K} \]

\[ C_p = 3799 \text{ J/Kg K} \]

\[ T_c = 310 \text{ K} \]

\[ T_e = 302 \text{ K} \]

The blood perfusion rates are obtained from the expression

\[ \lambda = \sqrt{\frac{C_p \cdot g}{K}} \]

**TABLE 2.1**

Blood perfusion rates for various types of tissue.

<table>
<thead>
<tr>
<th>( \lambda )</th>
<th>Blood perfusion rate ( \frac{\text{Kg}}{\text{m} \cdot \text{Sec}} )</th>
<th>Type of tissue</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.1</td>
<td>( g(1) = 1.314 \times 10^{-6} )</td>
<td>Negligible blood perfused tissue.</td>
</tr>
<tr>
<td>0.1</td>
<td>( g(2) = 1.314 \times 10^{-4} )</td>
<td>Poorly perfused tissue</td>
</tr>
<tr>
<td>5.0</td>
<td>( g(3) = 3.28595 \times 10^{-3} )</td>
<td>Well perfused tissue</td>
</tr>
<tr>
<td>10.0</td>
<td>( g(4) = 1.314 \times 10^{-2} )</td>
<td>Highly perfused tissue</td>
</tr>
</tbody>
</table>

The temperature distributions in tissue and veins are
presented in the fig (2.2) to (2.5). The values of \( \text{Mo} \) considered are 0, 0.17, 0.25. The values of \( \text{g} \) are given in the form of the table 2.1. No exchange between tissue and arterial or venous blood occurs. The values of \( \alpha \) considered are 0, 0.02, 0.4. The values of \( \text{Te} \) considered are 278K, 302K, 303K, 313K. For \( \text{Mo}=0 \) and \( \text{ha}=0 \), the results are same as those of Keller and Seiller (1971).

Figure 2.2 depicts the variation of tissue temperature distribution for various value of axial distance. For negligible blood perfusion rate the graph is almost linear. This may be happened because most of the heat is transferred by conduction process. In this case the heat perfusion in the capillary is very small. The temperature profiles deviates from the linearly as the blood perfusion rates increases. For highly perfused tissue the temperature profile becomes parabolic. Considering the effect of \( \text{Mo} \), it is clear from the figure that for the fixed perfusion rate [i.e poorly perfused tissue of \( g(2) \)] the temperature near the core region decrease with increase in \( \text{Mo} \) and temperature in the outer region increases with increase in \( \text{Mo} \).

Figure 2.3 describes the various of Veneous temperature distribution for various value of axial distance. For
negligible and low blood perfusion rates the results are similar to those of tissue temperature with small variation in quantity. It is clear from the graph that the qualitative behaviour of temperature distribution is quite similar for all types of blood perfusion rates. Considering the effect of Mo it is clear from the graph that for poorly perfused tissue the venous temperature increases with increase in the characteristic parameter.

Figure 2.4 depicts the variation of tissue temperature distribution with axial distance for different atmosphere temperature and co-efficient of metabolism. For poorly perfusion rate the temperature is increases with decreasing value of co-efficient of metabolism. In the case of well perfused tissue i.e. g(3), the temperature in the inner region decreases with the environmental temperature when the atmospheric temperature is lower than that of the core. But in the outer region reverse behaviour has been observed. This is due to the fact that the heat loss from the surface to the environment decreases with increase in the atmospheric temperature.
In figure 2.5 the effect of co-efficient of metabolism and atmospheric temperature on venous temperature distribution have been studied. The venous temperature decreases with increasing the co-efficient of metabolism. The same qualitative behaviour has been observed with atmospheric temperature as tissue temperature distribution. Hence we can conclude that the tissue temperature effects the venous temperature distribution. Therefore we cannot take the temperature distribution of venous beds is constant.

Thus the above study shows that the blood perfusion rate effects the temperature distribution of tissue and venous beds much more in comparison to other parameters.
Fig. 2.2 Tissue temperature distribution for various perfusion rates and metabolism.
Fig. 2.3 Various temperature distribution for various perfusion rates and metabolism.
Fig. 2.4 Tissue temperature profile for various coefficient metabolism and environmental temperature.
Fig. 2.5 Various temperature profile for various coefficient of metabolism and environmental temperature.