



General Physiology Spring 2024



Lecture 35

Body temperature regulation

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Objectives

- List and define the four mechanisms of heat transfer from the skin to the environment.
- Explain the feedback control of internal body temperature.
- Understand the short-term response to cold (to increase heat production and minimize heat loss) and heat (to decrease heat production and maximize heat loss).
- Understand the mechanisms of fever.

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Normal body temperature

- **Core temperature:** temperature of the **deep tissues** of the body, **constant**, measured either orally (affected by ingestion of hot or cold drinks & mouth breathing, normally **36.3- 37.1**) or rectally (0.5 higher)
- **Skin temperature:** rises & falls according to temperature of the surroundings

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Physiological variations

- 1- circadian fluctuation: 0.6 lower in the morning
- 2- monthly cycle: 0.5 C higher at the time of ovulation & second $\frac{1}{2}$ of the cycle & in the first trimester
- 3- exercise: rectal temperature can rise up to 40 C
- 4- emotional: due to unconscious tensing of muscles

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The Balance Between Heat Production & Heat Loss

- | | | |
|--|---|--|
| <ul style="list-style-type: none"> • heat production • Basal rate of metabolism • Food ingestion • Extra metabolism:
Slow prolonged by thyroxin
Rapid short by epinephrine,
norepinephrine, sympathetic | = | <ul style="list-style-type: none"> heat loss 2steps:
conduction of heat to skin
transfer to the surrounding |
|--|---|--|

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Heat loss

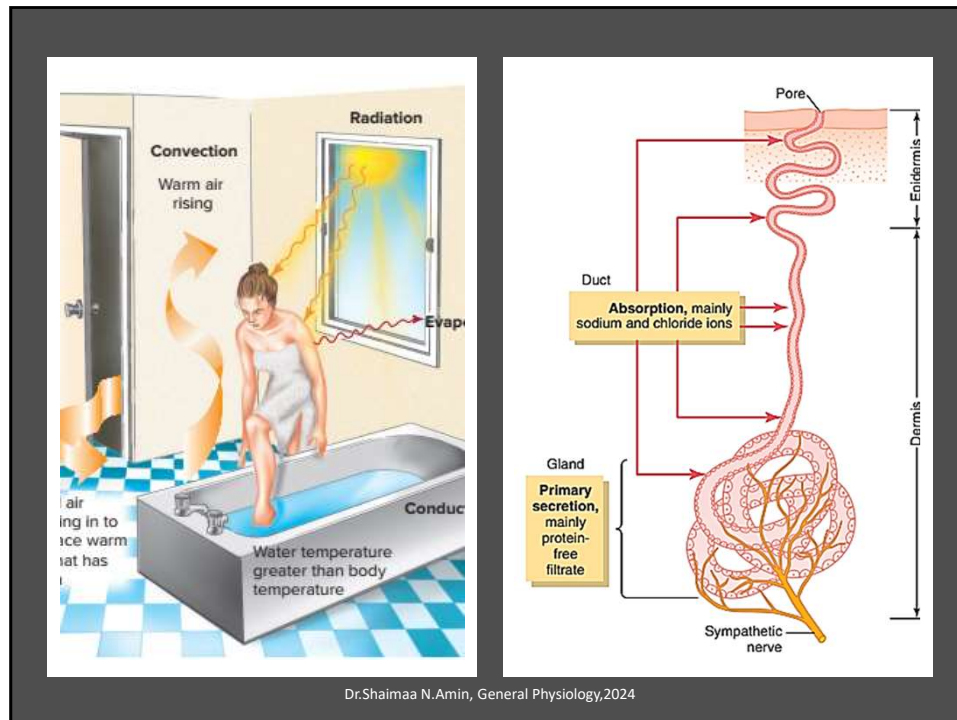
a)Conduction of heat to the skin:

The skin & subcutaneous tissue act as insulator, but blood vessels penetrate the fat to the skin

The rate of heat conduction to the skin (tissue conductance) depends on the blood flow to the skin regulated by vasoconstriction by sympathetic

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Heat loss

b) Transfer to the surroundings: by

1- heat radiation: as infrared electromagnetic rays, not in contact, the surroundings should be less than body temperature, normally 60% of heat loss is by radiation

2-conduction: the objects must be in contact with objects like chair & bed, larger amounts are lost to air

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Heat loss

b) Transfer to the surroundings: by

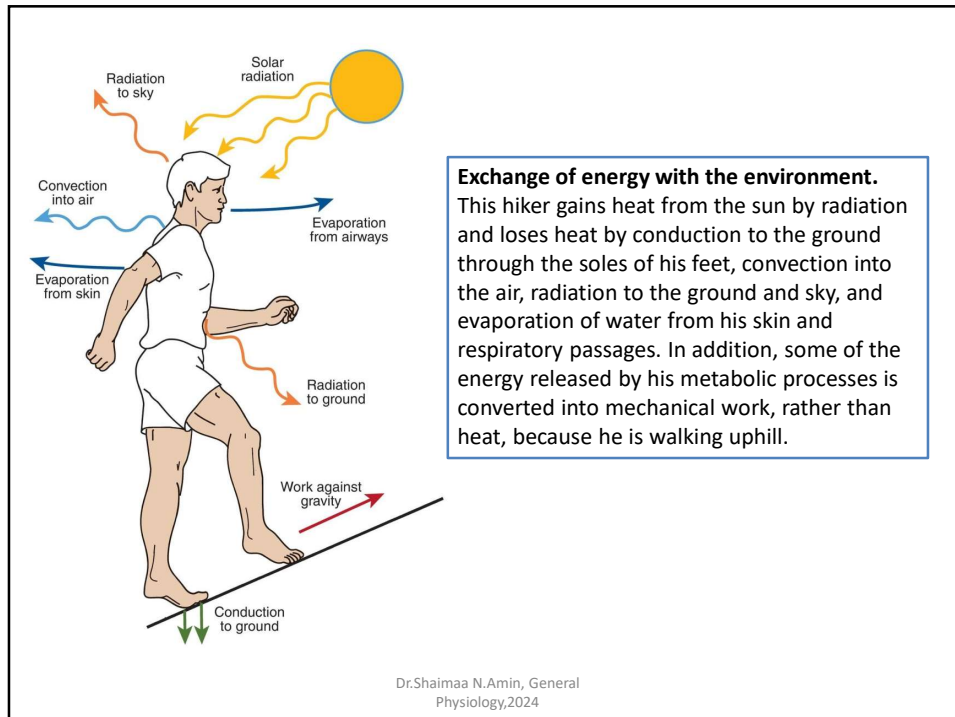
3- convection: air currents: 12% of heat is lost to air & removed by convection, wind increases heat loss

4- evaporation: 1gm H₂O removes 0.6 Kcal, when the person is not sweating, *certain amount of water still evaporates from skin & lungs at rate of 600ml/day (insensible water loss), the only way of heat loss if the surrounding is greater than the skin*, decreased by humidity of the environment,

5- small amount are removed by urine & feces

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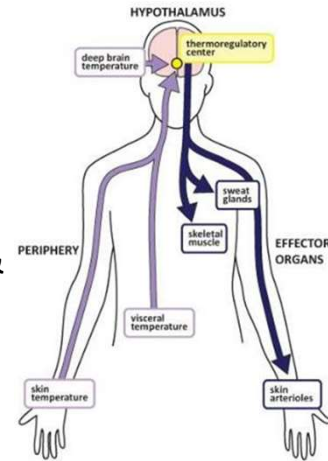
Temperature regulating centers

1-Anterior hypothalamus: *receptors*

Heat sensitive neurons

Cold sensitive neurons

2-Posterior hypothalamus: receives signal from anterior hypothalamus & from peripheral receptors, mainly *responsible for heat producing & heat conserving reactions*



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- **Peripheral receptors, present in:**

a) skin: more cold receptors, mainly for cold detection

b) deep tissues: in the spinal cord & abdominal viscera & around great veins, mainly cold receptors, detect core temperature

Increased temperature: stimulates heat sensitive neurons in the anterior hypothalamus, increase sweating

Decreased temperature: peripheral & central receptors activate posterior hypothalamus



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Temperature regulating mechanism

- Set point in the control system at 37C
- Regulation mechanisms include autonomic, somatic, endocrine & behavioral changes

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Temperature decreasing mechanisms are (heat exposure):

a) increase heat loss:

1- cutaneous vasodilatation: by inhibition of posterior hypothalamus, inhibition of sympathetic

2- sweating: stimulation of anterior hypothalamus
Autonomic pathways through spinal cord....sympathetic cholinergic fibres to sweat glands....increase sweat secretion

3- behavioural responses

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Temperature decreasing mechanisms are (heat exposure):

b) decrease heat production:

- 1- strong inhibition of heat production mechanisms
- 2- anorxia
- 3- apathy & inertia

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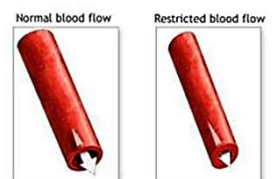
Temperature increasing mechanisms (cold exposure):

a) decrease heat loss:

1- cutaneous vasoconstriction: by stimulation of posterior hypothalamus sympathetic centers

2- pilo erection: sympathetic stimulation, not important in human (goose skin), to entrap air

3- behavioral responses



*ADAM



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Temperature increasing mechanisms (cold exposure):

b) increase heat production:

1- shivering: increase tone then shivering can increase heat production 5 times the normal

2- semiconscious increase in motor activity

3- increase secretion of epinephrine & norepinephrine (chemical thermogenesis)

4-increased thyroxin secretion



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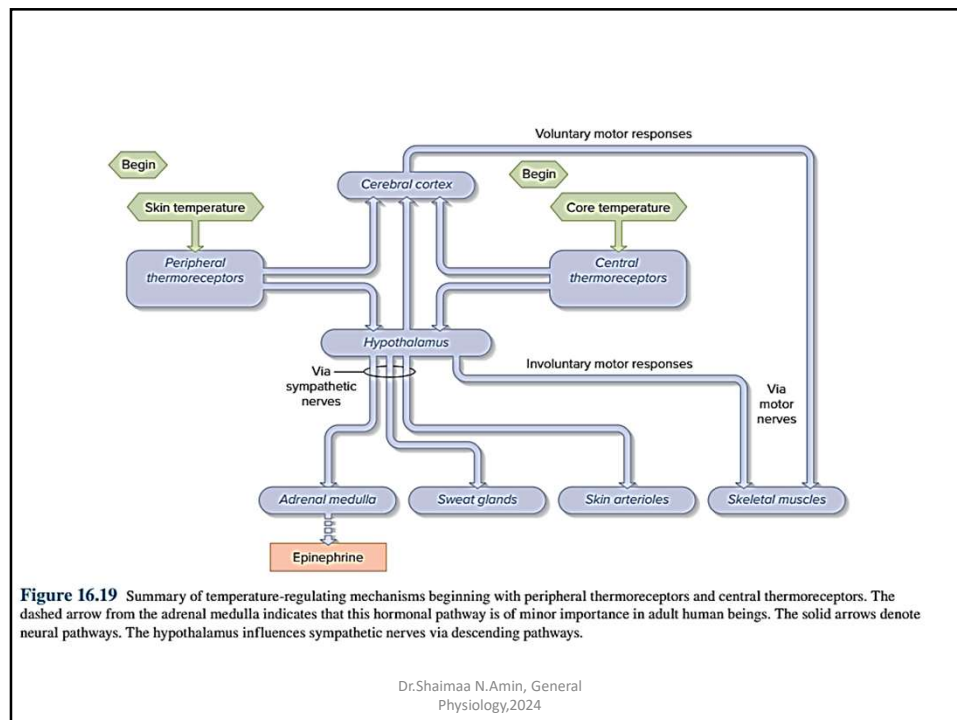


Figure 16.19 Summary of temperature-regulating mechanisms beginning with peripheral thermoreceptors and central thermoreceptors. The dashed arrow from the adrenal medulla indicates that this hormonal pathway is of minor importance in adult human beings. The solid arrows denote neural pathways. The hypothalamus influences sympathetic nerves via descending pathways.

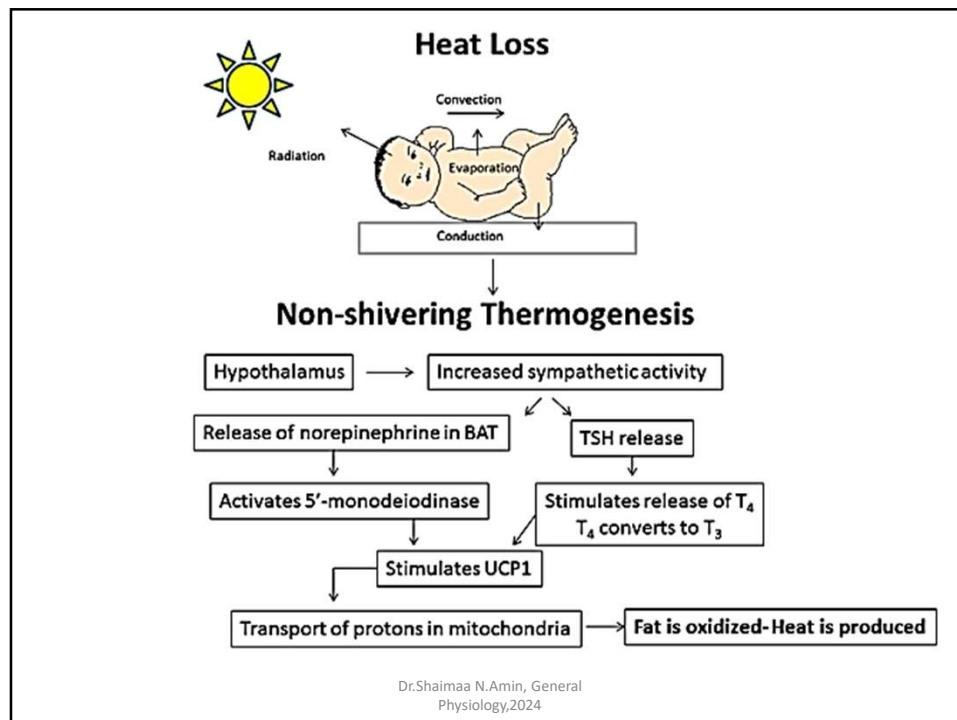
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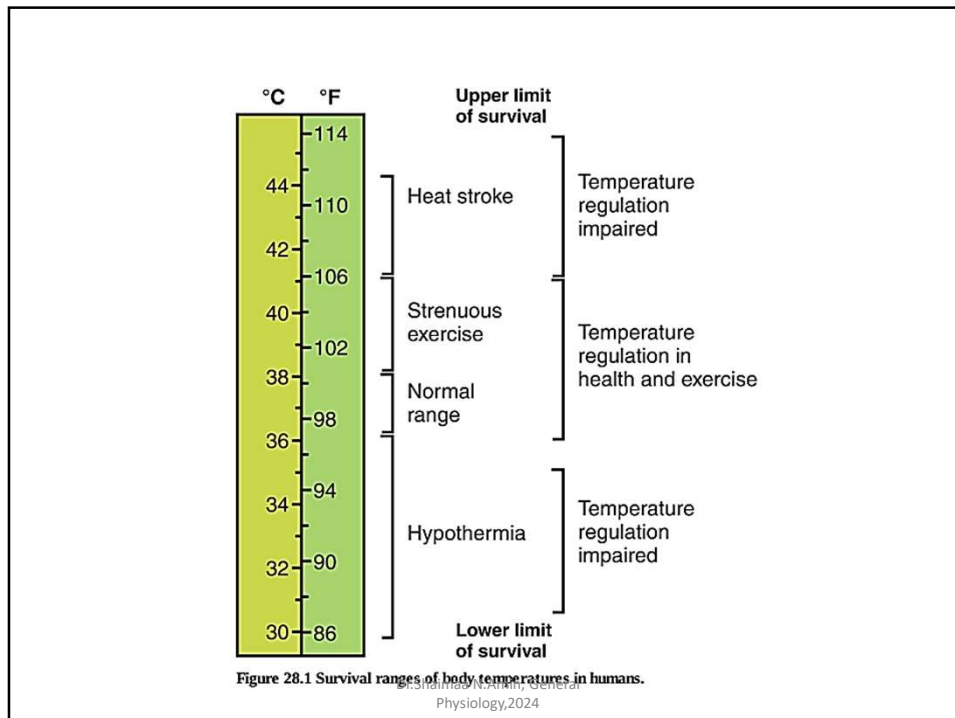
nonshivering thermogenesis. Its causes include an increase in the activity of a special type of adipose tissue called brown fat, or brown adipose tissue. This type of adipose tissue is stimulated by thyroid hormone, epinephrine, and the sympathetic nervous system; it contains large amounts of a class of proteins called uncoupling proteins. These proteins uncouple oxidation from phosphorylation and, in effect, make metabolism less efficient (less ATP is generated). The major product of this inefficient metabolism is heat, which then contributes to maintaining body temperature. Brown adipose tissue is present in infant humans (and to a smaller extent in adults). Nonshivering thermogenesis does occur in infants, therefore, whose shivering mechanism is not yet fully developed.

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Abnormalities of body temperature regulation

Fever:

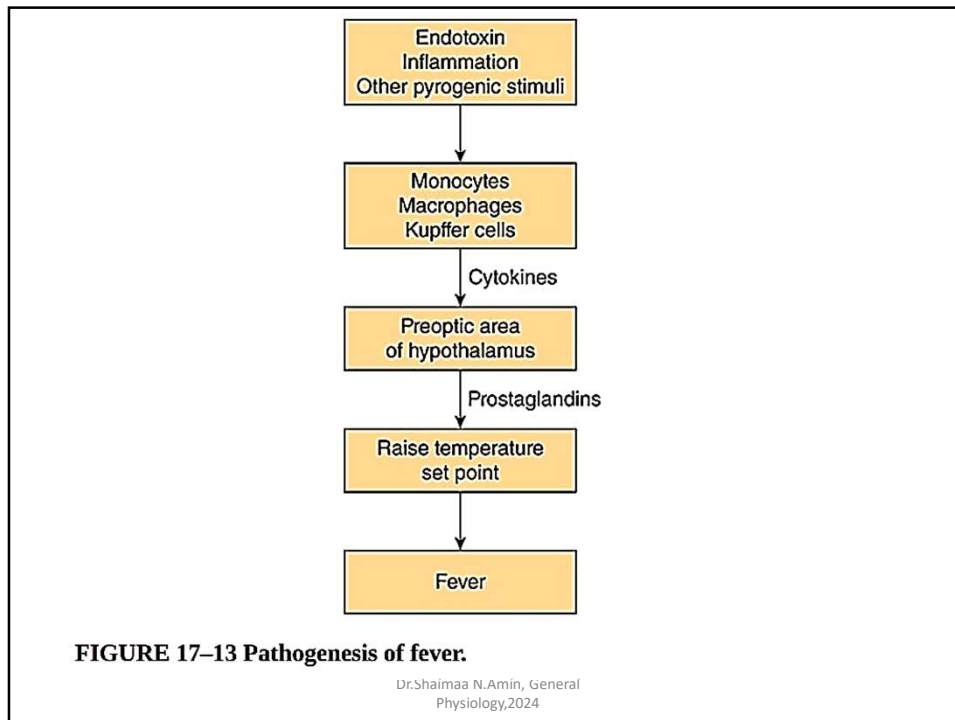
Def: elevation of core body temperature above level normally maintained by the individual, due to elevation of the hypothalamic set point, with activation of body temperature raising mechanisms. It rarely exceeds 41.1C

The pathogenesis of fever is summarized in **Figure 17–13**. Toxins from bacteria, such as endotoxin, act on monocytes, macrophages, and Kupffer cells to produce cytokines that act as **endogenous pyrogens (EPs)**. There is good evidence that IL-1 β , IL-6, IFN- β , IFN- γ , and TNF- α (see Chapter 3) can act independently to produce fever. These circulating cytokines are polypeptides and

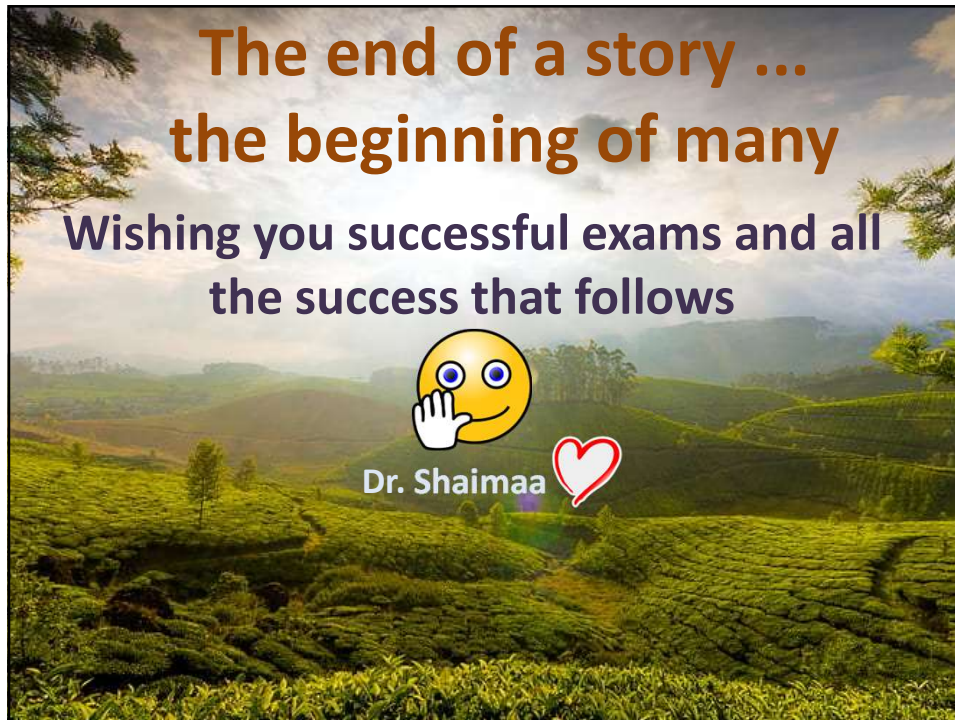
The fever produced by cytokines is probably due to local release of prostaglandins in the hypothalamus. Intrahypothalamic injection of prostaglandins produces fever. In addition, the antipyretic effect of aspirin is exerted directly on the hypothalamus, and aspirin inhibits prostaglandin synthesis. PGE₂ is one of the prostaglandins that causes fever. It acts on four

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