Heat production in the body is the result of metabolic processes like oxidative phosphorylation.

Regulation of body temperature relies on balancing heat production with heat dissipation.

I. Mechanisms to Increase Body Temperature
- largely organized by the posterior hypothalamus
  
  a. increase heat production
    - shivering
    - increased metabolic rate
    - in brown fat metabolism (↑ sympathetic)
    - via ↑ thyroid hormone secretion
    - behavioral actions
  
  b. decrease heat loss
    - piloerector muscle contraction = “goose bumps” (↑ sympathetic)
    - vasoconstriction of vasculature in skin (↑ sympathetic)
    - behavioral actions

II. Mechanisms to Decrease Body Temperature
- largely organized by the anterior hypothalamus

  a. increase heat loss
    - vasodilation of vasculature in skin (↓ sympathetic)
    - sweating (evaporation)
    - radiation & convection
  
  b. decrease heat production
    - inhibit thyroid hormone
    - behavioral actions

III. “Normal” Body Temperature

  a) “Core” temperature

  Far more variable than the accepted 98.6 °F (37 °C)

  Changes due to:
    - circadian rhythm
    - hormones
    - age
    - activity

  The “set point” for core body temperature is maintained by the hypothalamus.
III. “Normal” Body Temperature (cont.)

b) Skin temperature

IV. Regulation of Body Temperature

V. Clinical Applications of Hypothalamic Temperature Control

a) Hyperthermia

Heat Exhaustion
- characterized by elevated core temperature and excessive sweating
- fainting may occur as a result of decreased blood volume and pressure

Heat Stroke
- characterized by dangerously high core temperature and the absence of sweating
- usually causes some level of heat-induced tissue damage (denaturing proteins)
b) Hypothermia

Decreased core temperature beyond the capacity of a thermoregulatory response.

Due to the concurrent decrease in metabolic needs during hypothermia, as long as ice crystals don’t form in the tissues, complete recovery is possible.

Induced hypothermia is used in surgery to reduce ischemic injury due to loss of tissue perfusion by blood (ex. heart attack and stroke).

c) Post-menopausal Hot Flashes

Explain the symptoms experienced during a hot flash:
d) Fever

1) pathogens trigger white blood cells to release **endogenous pyrogens**
2) endogenous pyrogens cause release of **prostaglandins**
3) prostaglandins raise the “set point” for core body temperature

**Potential endogenous pyrogens**
- interleukins (1α, 1β, 6 and 8)
- tumor necrosis factors (α and β)
- interferons (α, β and γ)
- macrophage inflammatory proteins (α and β)

★ Destruction of pathogens can release **exogenous pyrogens** like lipopolysaccharides (LPS) which can stimulate further release of endogenous pyrogens

VI. Summary of Hypothalamic Temperature Control Examples
VIII. Antipyretic medications:

- Steroidal anti-inflammatory medications block the release of arachidonic acid which is the precursor molecule for prostaglandin production.

- Aspirin and other non-steroidal anti-inflammatory drugs (NSAIDs) inhibit the production of prostaglandins by inhibiting cyclooxygenase.