THERMOREGULATION:

(a) ***To outline the mechanisms for heat transfer between the body and its environment.***

(b) ***To describe the mechanisms by which heat is produced by the body.***

(c) ***To describe the mechanisms by which heat is lost and gained by the body.***

(d) ***To explain the processes used for conserving as well as generating heat under situations of lowered environmental temperature, and the effects of anaesthesia on these processes.***

(e) ***To explain the processes used for losing heat as well as increasing heat loss under situations of raised environmental temperature, and the effects of anaesthesia on these processes.***

(f) ***To define thermoneutral zone, and describe the energy requirements for maintaining normal body temperature.***

(g) ***To explain how the neonate differs in the regulation of body temperature compared with the adult and to explain the physical and physiological reasons for these differences.***

(I) **Temperature and Heat**:

Temperature:

- A measure of a physical property of a substance that determines the tendency for heat to flow from one object to another → heat energy is transferred from a region of higher temperature to a region of lower temperature
- Units for temperature:
    - SI unit → Kelvin (K), which is 1/273.16 of absolute temperature of "triple point of H_2O" (where H_2O exists in equilibrium as a solid, liquid and gas → occurs at 0.01°C)
    - Non-SI unit → Celsius (°C), where 1°C is $1/100^{th}$ the difference between the freezing and boiling point of H_2O

$$K = °C + 273.15$$

Heat:

- A form of kinetic energy → being a state of "thermal agitation" of molecules in a substance
- Units for heat:
    - SI unit → Joules (J)
    - Non-SI units → calorie (where 1 calorie = 4.186 J → raises temperature of 1 g of H_2O from 14.5°C to 15.5°C); Calorie (where 1 Calorie = 1000 calories = 4186 J)

Aside – Heat can be transferred by:

- (i) Conduction (via collision of molecules through a substance)
- (ii) Convection (via bulk flow of fluid (liquid/gas) surrounding the substance)
- (iii) Evaporation (via vaporisation of water from a substance's surface → Nb. Latent heat of vaporisation of H_2O is 580 cal/g or 2.4 MJ/kg at 37 °C)
- (iv) Radiation (via emission of EMR, often within IR band) → Nb. Unlike other heat transfer mechanisms, this can occur through a vacuum and does not require direct contact

Note – Heat transfer is dependent on the ambient environment:

- (i) Heat is transferred down its "temperature gradient" (from ↑ to ↓ temperature)
- (ii) Relative humidity is vital for determining heat transfer via evaporation (as it establishes a "moisture gradient")

Specific Heat:

- Specific heat of substance is the amount of heat energy required to ↑ temperature of 1 g of substance by 1°C
- SI unit → J/kg (Eg. 4.2 kJ/kg per °C for H_2O; 3.6 kJ/kg per °C for body tissue (of which 85% is H_2O))

(II) **Body Temperature**:

Core body temperature:

- Refers to deep body temperature of main internal organs (in head, trunk, abdomen) → sites where metabolic activity occur (Ie. heat production)
- Kept constant at 37 +/- 0.4 °C ("Normothermia") → displays normal variations:
    - Diurnal variation – ↑ in evening (37.3°C) and ↓ in early morning (35.8°C)
    - Menstrual variation – ↑ 0.5 °C in latter half of cycle

Note – Variations in core body temperature:
- Normothermia → core body temperature 37 +/- 0.4 °C
- Hypothermia → core body temperature < 36 °C
- Hyperthermia → core body temperature > 37.5 °C

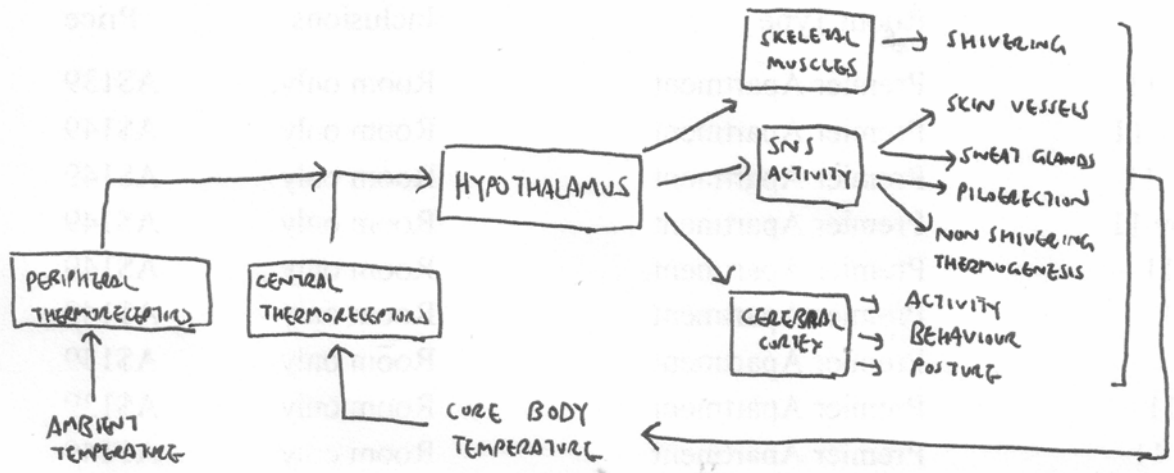
Note – Body maintains a constant core body temperature at "normothermia" (37 +/- 0.4°C) b/c proteins (esp enzymes) function optimally within a narrow temperature range (35-45 °C), and denature at temperatures > 45 °C

Peripheral body temperature:
- Refers to body temperature peripherally (Ie. skin, arms, legs, superficial tissues of core sites) → sites where heat loss occur
- Temperature varies widely → always LESS than core body temperature

Body temperature is determined by the balance of heat loss and heat production:
- (1) Heat loss:
  - To the environment mainly from the skin (95%) and respiratory tract (5%), with small amounts lost via urine/faeces (< 1%) → via following mechanisms:
    - (i) Radiation (40-50%)
    - (ii) Convection (20-30%)
    - (iii) Evaporation (15-20%)
    - (iv) Conduction (5-10%)
- (2) Heat gain:
  - (a) From the environment occurs via the same 4 heat transfer mechanisms also
  - (b) From metabolism (MAIN) → via:
    - (i) Muscle activity (vital) → 25%
    - (ii) Catabolism of fuel sources (CHO, fat, a.a.)
    - (iii) ATPase pumps (esp Na/K ATPase)

(III) **Regulation of Body Temperature**:

Follows a "sensor-integrator-effector model":



Afferent temperature sensors:
- (1) Core thermoreceptors → measure core body temperature
  - (a) Deep tissues/viscera (Eg. in intestinal wall)
  - (b) Brain (anterior hypothalamus and extra-hypothalamic areas)

    - (c) Spinal cord
- (2) Peripheral thermoreceptors → measure ambient/environmental temperature
    - (a) Subcutaneous tissues
        - Two types of receptors located within the dermis:
            - (i) Cold receptors (bulb of Krause) – Silent activity at 40 °C, but ↑ activity with decreasing temperature b/t 10-30 °C → transmits via myelinated Aδ and unmyelinated C sensory fibres
            - (ii) Warm receptors (bulbs of Ruffini) – ↑ activity with increasing temperature b/t 30-45 °C → transmits via unmyelinated C sensory fibres
        - Primary sensory afferents synapse in the dorsal horn of the spinal cord → secondary afferents then decussate and ascend via lateral spinothalamic tracts (anterior spinal cord) to the medulla → then to the anterior and posterior hypothalamus
    - (ii) Corneas

<u>Central integration</u>:

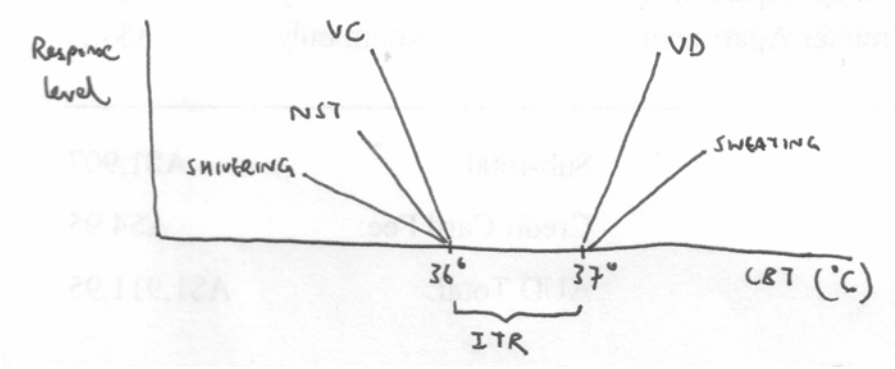
- Hypothalamus is the central integrator responsible for regulating body temperature → its functions include:
    - (1) Generating an optimal set-point temperature ("Interthreshold range" – see below)
    - (2) "Thermostat function" – Maintains core body temperature within the interthreshold range by integrating afferent inputs from thermoreceptors, and invoking effector mechanisms to minimise the difference between measured body temperature and the set-point temperature

> Note – Hypothalamus has two important sites:
> - (1) Anterior hypothalamus
>   - Responds to passage of warm blood locally → triggers heat loss mechanisms (Ie. sweating, vasodilation, Etc.)
> - (2) Posterior hypothalamus
>   - (i) Responds to cold afferent inputs from peripheral thermoreceptors → triggers heat production and heat loss prevention mechanisms (Ie. vasoconstriction, non-shivering thermogenesis, shivering)
>   - (ii) Establishes "interthreshold range"

- "Interthreshold range":
    - Defined as a range of core body temperatures over which no autonomic thermoregulatory responses are triggered → normally 0.2-0.4 °C (36.6-37.0 °C)

> Note – This range is influenced by various factors → (i) Age (↑ in elderly), (ii) Gender (↑ in women, esp during menstrual cycle), (iii) patient condition (↑ in critically ill pts or pts under anaesthesia), (iv) circadian rhythm, (v) food intake, (vi) thyroid function, (v) drugs, (vi) thermal adaptation to warm/cold ambient temperatures, (vii) activity level

    - This range is determined by the ratio of Na^+:Ca^{2+} in the posterior hypothalamus
    - At the thresholds of this range → thermoregulatory responses are triggered:
        - At upper end → vasodilation first, then sweating
        - At lower end → vasoconstriction first, then non-shivering thermogenesis, then finally shivering



Efferent responses:

Temperature deviations from the interthreshold range evoke various thermoregulatory effector responses:

- (1) SNS activity:
  - (a) Vasomotor tone of skin blood vessels
    - Skin consists of a network of capillaries, arterioles and venules (capacitance system of 1.5 L blood) → receives 8% of C.O. (300 mL/min)
    - These vessels receives vasomotor input from SNS → controls heat loss (via radiation, conduction, convection) from the body by altering skin blood flow (via α1-mediated vascular SM contraction)
    - Cutaneous blood flow acts like a "radiator":
      - (i) Vasoactive control of capillaries and arteriovenous shunts within hands/feet, lips, nose and ears
        - Vasodilation due to heat stress → ↑ cutaneous blood flow by 30x (up to 3 L/min) → ↑ heat loss
        - Vasoconstriction due to cold stress → ↓ cutaneous blood flow by 10x → ↓ heat loss
      - (ii) Counter-current system of cutaneous veins within the limbs
        - Cold blood returning from deep veins of a limb acquire heat from the arterial system → minimises heat loss to the environment
  - (b) Sweating
    - Skin sweat glands receive cholinergic SNS input → sweating ↑ with rises in core body temperature (↑ 7x per 0.5 °C ↑ in core body temperature)
    - Evaporation of sweat from skin causes evaporative heat loss only → degree of heat loss dependent on (i) ambient temperature (Ie. ↑ temp. → ↑ evaporative heat loss) and (ii) relative humidity (Ie. ↑ humidity → ↓ moisture gradient → ↓ evaporative heat loss)

    > Note – Sweating allows substantial heat loss from body due to evaporation of H_2O from skin → this is b/c H_2O has a high latent heat of vaporisation (0.58 kcal/g or 2.4 MJ/kg at 37 °C) → so each L of sweat loses 580 kcal of heat!)

  - (c) Non-shivering thermogenesis
    - SNS-mediated (β3 receptor) uncoupling of oxidative phosphorylation in brown fat and skeletal muscle → ↑ metabolic heat production without production of ATP or mechanical work

Important to note – Brown fat:

- Found in newborns only (base of neck, interscapular areas, perinephric fat, around large abdominal vessels) → 2-6 % of total body weight of neonate
- Metabolically active fat tissue → contains many fat globules and mitochondria, highly vascular, and SNS-controlled (via β3 receptor) → responsible for metabolic heat production in neonates during cold stress (can ↑ metabolic rate by 2x)
- Mechanism – Uncouples mitochondrial oxidative phosphorylation so more heat is generated for a given amount of metabolism of metabolic fuels (esp FA oxidation):
    - (i) Metabolism of metabolic fuels → generates a H^+ gradient across the inner mitochondrial membrane → this leads to production of heat
    - (ii) Brown fat uncouples oxidative phosphorylation by inserting channels into this membrane → dissipates the H^+ gradient so no ATP is produced

Aside – White fat:

- Contain large fat globule and minimal # of mitochondria
- Function – Store fat as energy reserve → lipolysis releases glycerol and FFA (latter used for energy in skeletal muscle/myocardium)

    - (d) Piloerection (little importance in humans)
        - Hair acts as an effective insulator → can trap layer of warm air → minimises heat loss
- (2) Behavioural responses from the cerebral cortex
    - Voluntary muscle contraction (Ie. ↑ activity with cold stress; ↓ with hot stress) – Affects heat production
    - Body posturing (Ie. ↓ BSA with cold stress;↑ with hot stress) – Affects heat loss
    - Clothing (Ie. ↑ clothing with cold stress; ↓ with hot stress) – Affects heat loss
- (3) Shivering thermogenesis
    - Involuntary contraction of skeletal muscles mediated by hypothalamus → activated when core body temperature continues to ↓ despite maximal behavioural, NST and skin vasoconstrictive responses → causes heat production
    - ↑ metabolic rate by 100% and global MRO_2 by 2-3x (Nb. can cause hypoxaemia!)
- (4) Appetite
    - Cold stress stimulates food-induced thermogenesis → ↑ metabolic rate and heat production
- (5) Thyroid hormone secretion
    - Cold stress stimulates thyroid hormone secretion → long-term ↑ metabolic rate and heat production

<u>Important to note – Summary of effector responses to hot and cold stress</u>:

Cold stress:

- ↑ metabolic heat production
    - Shivering and ↑ muscle activity
    - Non-shivering thermogenesis
    - ↑ food appetite
- ↓ heat loss
    - Active peripheral vasoconstriction
    - ↓ BSA (curling up)
    - Behavioural response (warm clothes)

Hot stress:

- ↓ metabolic heat production
    - ↓ muscle activity
    - ↓ food appetite
- ↑ heat loss
    - Active peripheral vasodilation
    - Sweating
    - Behavioural changes (take off clothes)
    - ↑ BSA (spreading out)

Aside – Skin is an important organ for thermoregulation because:

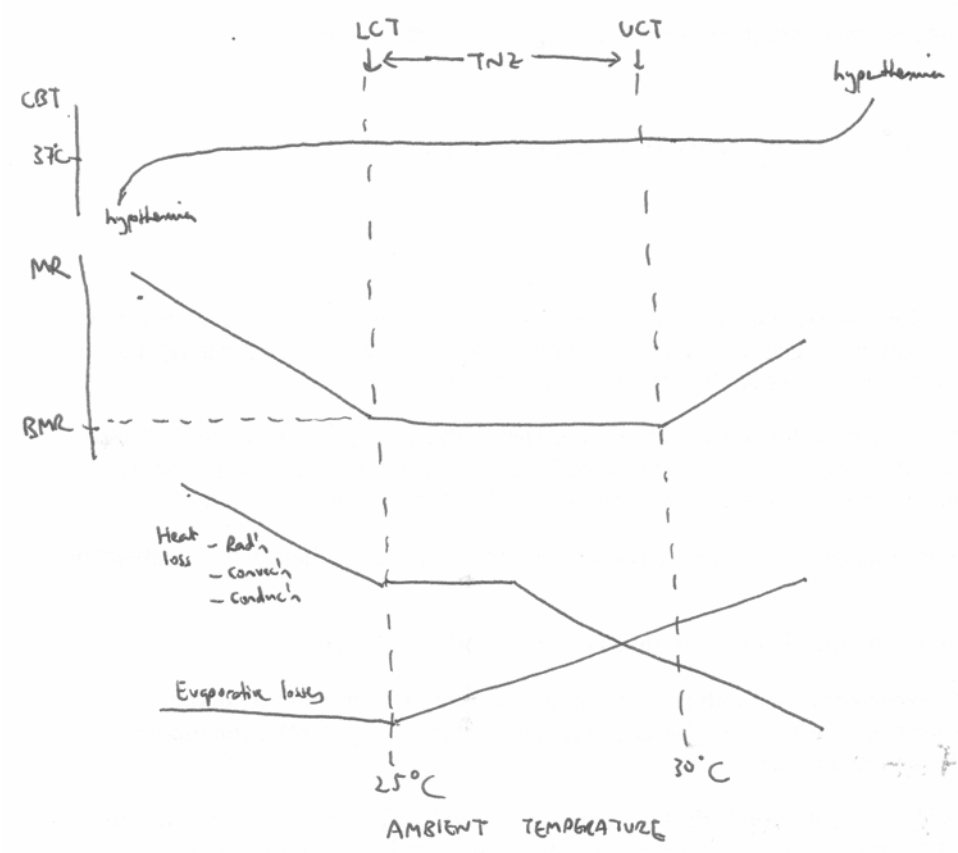
- (1) Provides a large surface area for heat transfer between skin and environment (95% of heat transfer)
- (2) Contains hot/cold thermoreceptors → monitor skin and ambient temperature → provides neural input to temperature regulatory centres in hypothalamus
- (3) Contains effector mechanisms that control heat loss to the environment:
  - (a) Cutaneous blood flow (receives 8% of C.O.) acts as a "radiator" → controls conductive, convective and radiative heat loss from the body (SNS-mediated; α1)
    - (i) Vasoactive control of cutaneous capillaries and arteriovenous shunts within hands/feet, lips, nose and ears
    - (ii) Counter-current system of cutaneous veins within the limbs
  - (b) Sweating (SNS-mediated, M3) → controls evaporative heat loss from the body
  - (c) Piloerection → prevent heat loss

(IV) **<u>Thermoneutral Zone</u>**:

- Defined as the range of ambient temperatures in which core body temperature is maintained without an increase in metabolic rate and O_2 consumption (Ie. body heat production) above a resting level

Neonates:	32-34°C (24-30 °C clothed)
Adults:	25-30 °C (20-22 °C clothed)

- Within this zone → thermoregulation is performed ONLY by mild changes in skin blood flow (requires minimal O_2 consumption/metabolic rate)



Note:

- Below the "lower critical temperature" of this zone → metabolic rate ↑ linearly as ambient temperature decreases to prevent ↓ in core body temperature
- Within the zone → metabolic rate remains constant as ambient temperature increases
- Above the "upper critical temperature" of this zone → metabolic rate ↑ linearly as ambient temperature increases to prevent ↑ in core body temperature

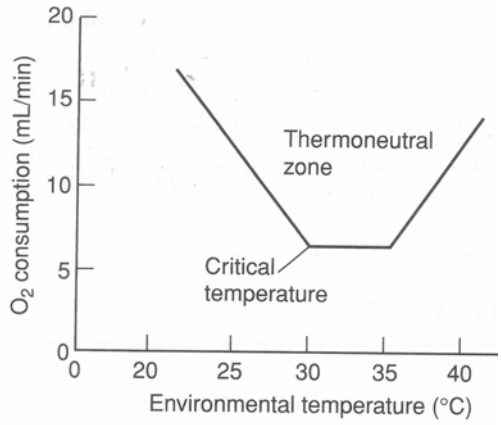
Note – Def'n of BMR occurs within a "comfortable" temperature (Ie. basal energy expended within thermoneutral zone) → 10% of BMR is spent on thermoregulation

(V) **Neonatal Thermoregulation**:

- TNZ of neonate vs adult:
  - (i) Range of neonatal TNZ is higher and narrower cf. adult (see below)→ this is b/c they have ↑ evaporative heat losses requiring higher ambient temperatures to maintain core body temperatures without a significant ↑ in metabolic rate

Pre-term neonate:	35-36 °C
Full-term neonate:	32-34 °C (24-30 °C clothed)
Adult:	25-30 °C (20-22 °C clothed)

  - (ii) "Critical temperature limits" of neonatal TNZ ↓ with ↑ maturity and body side (Ie. lower limit is 35 °C if preterm, 33°C at term, 32 °C after 2/52)



Note:
- Below the "lower critical temperature" of this zone → metabolic rate ↑ linearly as ambient temperature decreases to prevent ↓ in core body temperature
- Within the zone → metabolic rate remains constant as ambient temperature increases
- Above the "upper critical temperature" of this zone → metabolic rate ↑ linearly as ambient temperature increases to prevent ↑ in core body temperature

- Neonates have ↑ difficulty maintaining a stable core body temperature with Δs in ambient temperatures depends due to a ↓ ability to balance heat production and heat loss (due to the reasons below) → thus, ↑ risk of heat/cold stress with Δ in ambient temperatures:
  - (i) Large SA:volume ratio (2-3x cf. adult) → ↑ environmental heat gain and losses
  - (ii) Thin subcutaneous tissues (50% cf. adult)→ ↓ insulating capacity and ↑ evaporative losses (esp when skin is wet) → ↑ environmental heat gain and losses
  - (iii) Higher TNZ (32-34 °C) → neonates have high evaporative heat losses and thus require a higher ambient temperature to maintain core body temperature without a significant rise in metabolic rate → this means they have ↑ environmental heat losses at lower ambient temperatures
  - (iv) Higher BMR (2x cf. adults) → significant heat loss is required to maintain thermal equilibrium (so TNZ is < body temperature) → so ↑ susceptible to heat stress (Ie. keeping a baby at 37 °C is a form of heat stress)
  - (v) Limited sweating capacity (↑ susceptibility to heat stress), shivering response (↑ susceptibility to cold stress), and ability to exert direct control on environment
- Mechanisms of neonatal thermoregulation:
  - Within TNZ → temperature is maintained by changes in skin blood flow only → requires little ↑ in metabolic rate (or O_2 consumption)
  - Cold stress → responses include:
    - (i) Behavioural changes (esp crying to signal attention)
    - (ii) Skin vasoconstriction
    - (iii) Non-shivering thermogenesis (involving brown fat)
    - (iv) ↑ muscular activity and shivering → these are NOT well developed cf. adults, and thus play a minor role
  - Heat stress → responses include:

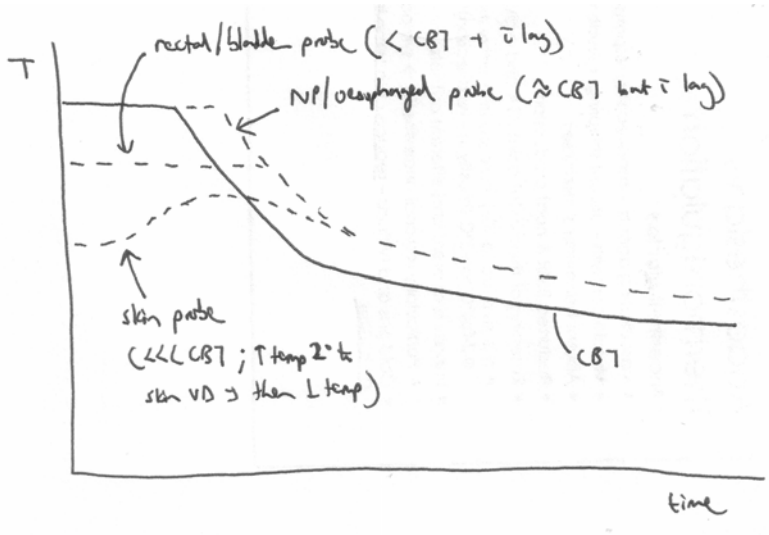
- (i) Behavioural changes (Ie. crying, removing coverings)
- (ii) Skin vasodilation
- (iii) Sweating → limited role in neonates (33% effectiveness cf. adults), BUT sweating results in significant evaporative heat loss (↑ heat loss by 2x)

Important to note:
- Dangers of hypothermia in neonate are similar as in adults – but in neonates there is especially:
  - (1) ↑ hypoxaemia due to (i) ↑ MRO_2/metabolism and (ii) ↑ respiratory distress 2° to ↓ surfactant synthesis
  - (2) ↑ CVS instability (↓ C.O., BP and HR)
  - (3) ↑ hypoglycaemia risk due to ↑ metabolism
  - (4) ↑ delayed recovery from anaesthesia
  - (5) ↑ coagulopathy
  - (6) Significant alteration in PK of drugs
- Dangers of hyperthermia in neonate:
  - (1) ↑ evaporative water loss (due to ↑ SA:vol raito) → ↑ dehydration
  - (2) ↑ apnoeic attacks

(VI) **<u>Effects of Anaesthesia on Thermoregulation</u>**:

<u>Measuring temperature during anaesthesia</u>:
- Core body temperature can be measured using temperature probes placed in various areas:
  - (i) Tympanic membrane → good response time and accuracy
  - (ii) Nasopharynx → less accurate by 0.2-0.5 °C. Also needs to be posterior to soft palate and is affected by gas flows
  - (iii) Oesophageal → accurate if positioned to avoid cooling effects of gas flows
  - (iv) Rectal → accuracy affected by temperature of faecal load and blood returning from lower limbs
  - (v) Bladder → dependent on urine flow (requires > 300 ml/day)
  - (vii) PA catheter → invasive but gold-standard core body temperature measurement
  - (vi) Skin (axilla)
- Temperature is measured several ways (see notes on "Clinical Monitoring"):
  - (i) Non-electrical – Liquid expansion (Hg or EtOH thermometer), Bimetallic strip, Bourdon gauge
  - (ii) Electrical – Infra-red, Thermistor, Thermocouple, Resistance thermometer

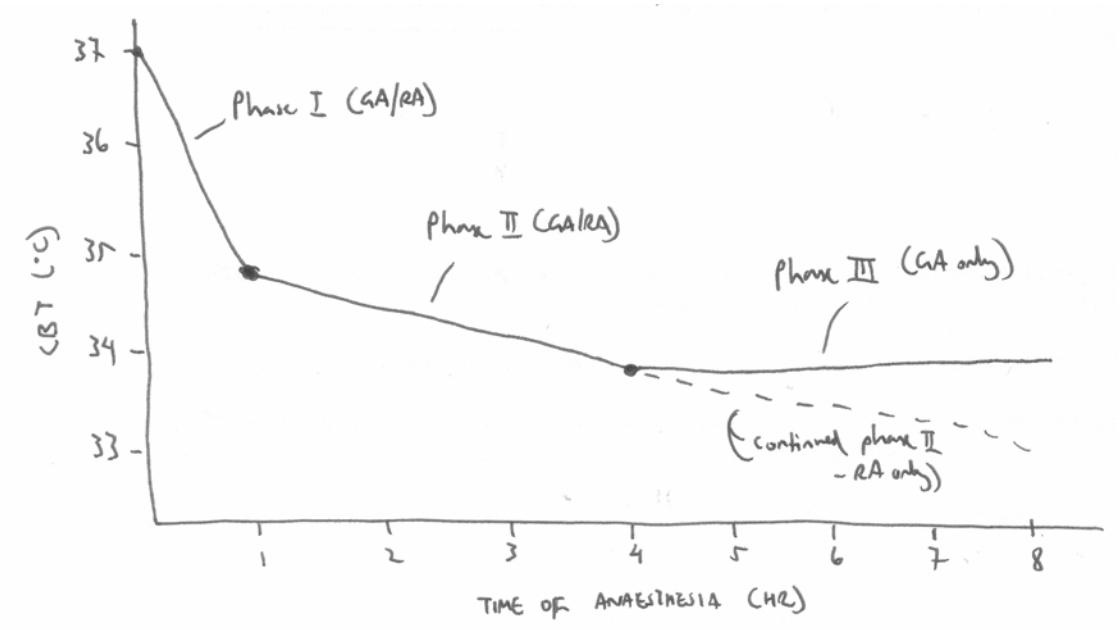
Note – Temperature probe generally uses either a – (i) Thermister (a semiconductor that reduces its resistance predictably with warming), or (ii) Thermocouple (a circuit containing two dissimilar metals that generate a potential difference at different temperatures)



Effect of anaesthesia on core body temperature:

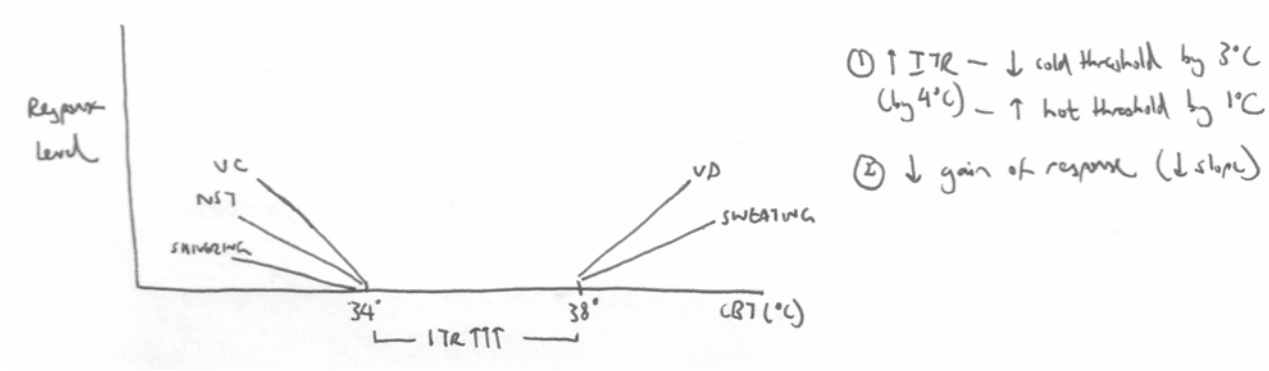
There is a fall in core body temperature during general anaesthesia → occurs in three steps:

- Phase I – During induction of anaesthesia (within first hour), core body temperature ↓ by 1-2 °C due to redistribution of heat from warm central compartments to peripheral compartments due to anaesthesia-induced vasodilation
- Phase II – Following induction, core body temperature continues to ↓ by another 1-2 °C in the next 2-3 hours due to (i) ongoing heat loss to the environment in excess of metabolic heat production, and (ii) resetting of the "interthreshold range"
- Phase III – Core body temperature reaches a steady state after another 2-3 hours when heat loss to the environment equals metabolic heat production. This is caused by thermoregulatory responses triggered to counter the cold stress invoked when core body temperature ↓ below the reset interthreshold range



Fall in core body temperature during spinal/epidural anaesthesia follows a similar pattern EXCEPT:

- Phase III is not seen b/c re-establishment of peripheral vasoconstriction is not possible in the blocked areas due to sympathectomy → so heat loss to the environment continues to be greater than metabolic heat produced
- Phase II thus continues until patient is noted to shiver (first sign of hypothermia)



Mechanisms of ↓ in core body temperature:

- (1) General anaesthesia:
  - (i) Resetting of interthreshold range (phase II)
    - ↑ width of range to 4 °C → ↓ threshold to cold by 3 °C and ↑ threshold to heat by 1° C → ↓ thermoregulatory responses to Δ in body temperature

        - Caused by a central effect → GA agent interferes with normal hypothalamic function to maintain a narrow threshold range
    - (ii) GA agent-induced vasodilation → redistribution of heat from central to peripheral compartments (phase I)
    - (iii) Muscle paralysis → loss of shivering response and muscle activity (phase II)
    - (iv) LOC and paralysis → loss of behavioural responses (phase II)
    - (v) GA-induced ↓ BMR/heat production (phase II)
    - (vi) Cold gases and IVF (phase II) → temp. generally ↓ 0.25-0.5 °C/L of IVF

Aside: To calculate Δ temp. from mass of IVF

$$\Delta T = (T_1\text{-}T_2) \times \frac{(mass_2 \times spec.\ heat_2)}{(mass_1 \times spec.\ heat_1)}$$

Nb. 1L of N/S at room temp → ↓ 0.3 °C; 300 mL of PRBC at 4°C → ↓ 0.2 °C

- (2) Spinal/epidural anaesthesia:
    - (i) Resetting of interthreshold range (phase II) → similar effect to interthreshold range width and thresholds to hot/cold as GA, BUT caused by altered hypothalamic perception of temperature 2° to blocked dermatomes (blunted afferent inputs)
    - (ii) Vasodilation 2° to sympathectomy (blunted efferent outputs) → redistribution of heat from central to peripheral compartments (phase I)
- (3) Surgical factors:
    - (i) Exposure to ambient OT temperature (phase II)
    - (ii) Open moist cavity (Eg. laparotomy) (phase II)

Important to note → Thermoregulatory responses to cold stress during anaesthesia:
- (i) Re-establishment of peripheral vasoconstriction (phase III – ↓ environmental heat loss) → seen during general anaesthesia (but absent during regional anaesthesia)
- (ii) Non-shivering thermogenesis (phase III – ↑ metabolic heat production) → important in infants only
- (iii) Shivering and behavioural changes → this is not seen during GA (but may be seen during regional anaesthesia)

<u>Physiological effects of hypothermia</u>:
- Hypothermia (core body temp. < 36 °C) is common during anaesthesia → especially in:
    - (i) Patients of extreme ages (neonates/elderly)
    - (ii) Procedure of prolonged duration
    - (iii) Abdominal surgery (due to exposure of moist open cavity)
    - (iv) Cold ambient OT temperatures
- Hypothermia has many deleterious physiological effects:

CVS	- Tachycardia initially, then progressive bradycardia with ↑ cold - ↑ cardiac arrhythmias and myocardial ischaemia due to catecholamines released from stress response - ↑ SVR and MAP due to peripheral vasoconstriction - ↓ C.O. due to direct –ve inotropic effect of cold and ↑ afterload/SVR
Respiratory	- Left-shift in Hb O_2 dissociation curve - V/Q mismatching 2° to inhibition of hypoxic pulmonary vasoconstriction - Bronchospasms - ↓ MV (and in severe cases apnoea) - ↑ solubility of gases (incl volatiles)
CNS	- Altered mental state (esp ↑ drowsiness, unconsciousness and delayed awakening from GA) - ↓ CBF
Haematological	- Coagulopathy (due to platelet dysfunction and loss of CF enzyme

and immunological	function) → ↑ transfusion requirements due to bleeding - ↓ WBC activity → ↑ incidence of infections
Hepatic/renal	- Impaired renal function (oliguria) - Impaired hepatic metabolic function (esp ↓ drug metabolism, such as muscle relaxants)
Metabolic and endocrine	- Impaired wound healing (due to catabolic state and wound vasoconstriction) - ↑ protein catabolic state - ↑ stress response (steroids and catecholamine released) - Shivering → 5x ↑ general MRO_2 → causes hypoxaemia (risk of myocardial and cerebral ischaemia) - In absence of shivering → general ↓ MRO_2 (by up to 50%) and ↓ BMR
Others	- ↑ morbidity and mortality rate (due to above reasons) - Hypothermia may be beneficial during cerebral or cardiac ischaemia as it ↓ metabolic O_2 requirements (provided shivering response is blunted) - ↓ anaesthetic requirements (MAC-sparing) - ↓ triggering and severity of MH

<u>Prevention and management of hypothermia during anaesthesia</u>:

- (1) Phase I heat loss → prevented by prewarming the peripheries with FAWD for 30 minutes prior to anaesthesia (MOST important as most heat is lost here)
- (2) Phase II heat loss → prevented by:
    - (i) Intraoperative use of FAWD/warm blankets
    - (ii) Intraoperative use of warmed IVF and wash solutions
    - (iii) Intraoperative use of heated and humidified inspired gases (Ie. HME filter)
    - (iv) Intraoperative use of passive insulators (Ie. wrapping non-operative sites with insulating materials)
    - (v) ↑ ambient temperature of OT
    - (vi) Shortening operating times
    - (vii) Minimising exposure time of wet surfaces (Ie. minimise evaporative heat loss)