بسم الله الرحمن الرحيم

Alterations in body temperature

by

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Personally, I have always felt that the best doctor in the world is veterinarian. He cannot ask his patients what is matter…..he is just got to know.

Will Rogers

CONTROL OF BODY TEMPERATURE

- Mammalian species maintain core body temperature within narrow range despite of extremes in environmental conditions.

- Diurnal variation: The normal range of temperature for individuals within a species may vary as much as 1ºC (2ºF).

Neuronal control of body temperature:

- **Warm and cold-sensitive neurons within the hypothalamus; via**

  i- peripheral nerve receptors

  ii- blood bathing the hypothalamus.

- **Integrative structure located in the pre-optic region of the anterior hypothalamus (POAH), that act as a thermostat activate both behavioral and autonomic effector responses to either lose or gain heat.**

Heat production :

1-Muscle activity

2- Digestion of food

3- Heat conservation

i- **Adrenergic autonomic** stimuli to decrease peripheral circulation and cause piloerection.

ii- **Behavioral means** of heat conservation include adopting a "huddled" posture group aggregation, and seeking a sheltered environment.

Heat loss :

1-Conduction

2- Convection

3- Radiation from body surfaces

4- Evaporation.

CONDITIONS OF INCREASED BODY TEMPERATURE

Body temperature disorders in which the core body temperature set point (POAH) is unaltered can occur from:

1- Increased heat production

2- Absorption of heat

3- Impairment of heat loss.

1- Exercise

2- Malignant hyperthermia

3- Ergopeptine alkaloid toxicosis

4- Heat Stroke

5- Anhidrosis

6- Diseases of the nervous system

1- Exercise

- Body temperatures during exercise greater than 2ºC (4ºF) above normal especially if reached early in exercise are usually the result of severe environmental conditions and/or failure in heat loss mechanism.

- Seizure associated with generalized tonic clonic may, like vigorous exercise, cause a rise in body temperature. If central heat regulatory systems are unaffected by the disease process, body temperature return to normal, no longer than 48 hours, after the last seizure. Elevated temperatures that persist for longer periods should prompt investigation into other causes for the increased temperature.

2- Malignant hyperthermia

- It consists of a group of inherited skeletal muscle calcium metabolism disorders in which a hypermetabolic state of muscle is induced by the administration of halogenated inhalation anesthetics, depolarizing skeletal muscle relaxants, or, occasionally, local anesthetics.

- Rapid increase in core body temperature (39ºC-42ºC), skeletal muscle rigidity, tachycardia, metabolic acidosis, and muscle necrosis may lead to death.

3- Ergopeptine alkaloid toxicosis

It causes vasoconstriction and reduced blood flow to the skin of ruminants, bronchoconstriction and pulmonary vasoconstriction, which further compromise ruminants' ability to lose heat especially, during hot environmental conditions. It is recognized as part of the syndrome of

fescue toxicosis of "summer slump".

Heat Stroke

When animals are exposed to high ambient temperatures, in tense solar radiation and/or high humidity.

- Heat stroke is more common in ruminants and Sheep with fleece

- Horses' susceptibility to heat stress (Heat stroke) is enhanced if dehydration and electrolyte imbalances occur because of large losses of sweat.

- Rectal temperature increases above 41.5º C (107º F), deceased blood pressure, and decreased cardiac output occur. The animals are lethargic and have weak, flaccid muscles; prostration and shock occur rapidly. Disseminated intravascular coagulation (DIC), liver damage, renal failure, and myocardial necrosis are frequent complication.

5- Anhidrosis

- **As many as 25% of horses in hot, humid environment lose their ability to sweat and subsequently suffer from hyperthermia as a result of impairment of heat loss.**
- **Horses in training have been reported to have higher frequency of anhidrosis as are horses shipped to hot environments from more temperate regions.**
- **clinical signs are poor performance, total or partial loss of ability to sweat, increased respiratory rate [three to five times normal), and dry, thin hair coats with areas of alopecia.**

6- Diseases of the nervous system

- **Central nervous system disorder that damage areas of the hypothalamus associated with temperature regulation may lead to either decreases or increases in body temperature, although hypothermia is most common.**
- **Hemorrhage, space occupying masses (abscess, tumors), infectious or inflammatory diseases, and degenerative disorders have all been implicated in hyperthermia.**
- **Central hyperthermia is usually characterized by lack of diurnal variation, absence of sweating, resistance to antipyretic drugs, and excessive response to external cooling.**

Certain toxins and drugs may act to increase body temperature by causing an increase in metabolic work. Chlorophenols and nitrophenols, used as herbicides and wood preservatives, cause uncoupling of oxidative phosphorylation within mitochondria and lead to rapid extreme rises in body temperature. Chronic and/or low level exposure to these compounds may manifest clinically as hyperthermia.

FEVER

- True fever differs from other hyperthermic states in that the desired core body temperature or set point (POAH) is elevated.
- This new, higher set point is vigorously defended by same mechanism that maintains body temperature in health.

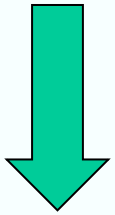
Pathophysiology

Initiation of the febrile state can occur

by a variety of infectious, inflammatory,

immunologic, neoplastic, or injurious

conditions



these stimuli cause the production of pyrogenic cytokines by a wide variety of cells, but primarily by fixed or circulating monocytes and macrophages. Currently at least 11 cytokines have been shown to induce the febrile response

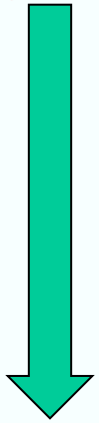
Cytokines: interleukin- I (IL-Iα, IL-Iβ) and tumor necrosis factor alpha (TNF-α)

other pyrogenic cytokines: IL-II,1L-6, interferon (IFN-α, IFN-β, IFN-ɣ), ciliary neurotropic factor (CNF)

act on common receptor (glycoprotein 130).

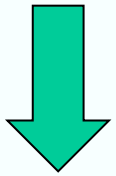
Pyrogenic cytokines reach the POAH via the circulation and attach to receptors on endothelium of the capillaries of circumventricular vascular organs (CVVOs).

induce the production of arachidonic

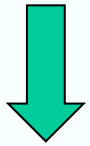


Cyclooxygenase (COX)-2

prostaglandin E_2 (PGE_2)



PGE_2 produced on the brain side of the CVVOs binds to PGE_2 type3 receptors of glial cells and neuronal

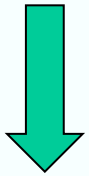


producing a cascade of changes:

- in cyclic nucleotides,

- calcium,

- and neurotransmitters



result in a higher (set point) within the hypothalamic thermoregulatory center.

Beneficial effect of fever

1- Body temperature elevation in pyrogenic mediated fever, in contrast to hyperthermic states, rarely exceeds 2.5º C (5º F) above normal.

2- Although the severity of some viral infection is decreased at these temperatures, most pathogens are not affected by a modest rise in temperature.

3- Bacteria, which require iron for multiplication, are inhibited by the reduction availability of iron during the acute phase reaction.

4- Certain neoplastic cells are inhibited during fever, although it is likely that inhibition of neoplastic cell division results from augmentation of immune responses.

5- Enhancement of host defenses, however, appears to be the primary beneficial effect of fever.

i- Neutrophils and monocytes have increased motility and emigration, enhanced phagocytosis, increased oxygen radical production, and enhanced killing of intracellular bacteria. IFN production increases, and its antiviral, antitumor, antiproliferative, and natural killer (NK) cell-stimulating properties are enhanced.

ii- Increased T-cell proliferative responses to nonspecific mitogens IL- l and IL- 2, and enhanced T-helper cell activation, expression, recruitment, and cytotoxicity have all been correlated with fever, Enhancement of B cells, with a subsequent increase in production of antibodies.

Adverse Effect of Fever

1-The beneficial effects of fever during bacterial infections in rabbits have been shown to reverse at temperatures greater than 3º C (5º F) above normal

2- Cytokine disregulation may result in prolonged or extreme fevers with adverse effects on a variety of body functions in addition to the immune response.

3- Catabolic metabolic processes during fever are markedly different from catabolism of starvation, Protein loss occurs four times as rapidly in individuals with infectious or inflammatory diseases as compared with starvation-adapted individuals.

4- This cytokine-driven catabolism, combined with the decreased feeding behavior that accompanies fever, variable anorexia (even if feed is provided), and increased metabolic rate at higher temperatures, can result in rapid and severe muscle wasting, weakness, and atrophy.

4- In humans high fevers frequently cause seizures, especially in children, but this is rare in animals unless temperatures reach 42ºC (108º F) in neonates.

5-Prolonged high fevers in debilitated animals may lead to failure of the cardiovascular system.

Toxemia and endotoxemia

- The presence of toxins produced by bacteria or injury to tissue cells. Toxemia does not include the diseases caused by toxic substances produced by plants or insects or ingested organic or inorganic poisons.

- The most common form of toxemia in large animals is endotoxemia, caused by the presence of lipopolysaccharide cell wall components of Gram-negative bacteria in the blood, and characterized clinically by abnormalities of many body systems.

ETIOLOGY OF TOXEMIA AND ENDOTOXEMIA

Toxins can be classified as:

- antigenic - metabolic.

Antigenic toxins

- produced by bacteria and to a lesser extent by helminthes.

- Stimulate the development of antibodies.

- Antigenic toxins are divided into exotoxins and endotoxins

Exotoxins

- protein substances produced by bacteria.
- specific in their pharmacological effects and in the antibodies that they induce.

The important bacterial exotoxins are those produced by Clostridium spp.,

- Ingested preformed, as in botulism.
- produced in large quantities by heavy growth in the intestines, such as in enterotoxemia, or from growth in tissue, as in blackleg and black disease.

Endotoxins

- The endotoxins are lipopolysaccharides found in the outer wall of Gram negative bacteria.

- Endotoxins are released into when bacterial cell wall breaks.

- Endotoxin gains access to the blood when there is a severe localized infection, such as a coliform mastitis in dairy cattle, or a disseminated infection, such as coliform septicemia in newborn calves.

Metabolic toxins

accumulate as a result of incomplete elimination of toxic materials normally produced by body metabolism, or by abnormal metabolism.

- When normal mechanisms are disrupted, particularly in hepatic dysfunction.

-In obstruction of the lower alimentary tract there may be increased absorption of toxic phenols, cresols and amines that are normally excreted with the feces, resulting in the development of the syndrome of autointoxication.

Clinical findings of Toxemia and Endotoxemia

Acute toxemia

The clinical findings of acute toxemia in most

nonspecific toxemias are similar.

Depression, anorexia and muscular weakness, and

fever

The clinical findings of severe endotoxemia include:

o Depression

o Hyperthermia followed by hypothermia

o Tachycardia followed by decreased cardiac output

o Decreased systemic blood pressure

o Cool skin and extremities

o Diarrhea

o Congested mucosae with an increased capillary refill time

o Muscular weakness, leading to recumbency.

Renal failure is common and is characterized by anuria

TREATMENT OF TOXEMIA

The principles of treatment of toxemia , endotoxemia or septic shock include:

1) Removal of the foci of infection.

2) Administration of antimicrobial agents with a Gram-negative spectrum e.g. ß-lactam antibiotics

3) Aggressive **fluid and electrolyte therapy** to combat the relative hypovolemia, hypoglycemia, and electrolyte and acid-base disturbances;

4) **NSAIDs** e.g. Flunixin meglumine

5) **glucocorticoids** e.g. dexamethasone

for the inhibition of cytokines and the products of the cyclooxygenase pathway. These four treatments. are routinely applied.

Septicemia

It is the acute invasion of the systemic circulation by pathogenic bacteria accompanied by sepsis or septic shock.

ETIOLOGY

All species

Anthrax, pasteurellosis and salmonellosis

are found in all species of food animal.

CLINICAL FINDINGS

The major clinical findings in septicemia are:

- fever
-cardiovascular dysfunction and shock
- submucosal and subepidermal hemorrhages that are usually petechial and occasionally ecchymotic.

The hemorrhages are best seen under the conjunctiva and in the mucosae of the mouth and vulva.