Fever in the new born infants

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The human body is in a continuous heat production through its metabolic processes against wide range of environmental variations in the temperature. The normal body temperature is about 37°C or 98°F with a very narrow range. The body heat is produced mainly in the skeletal muscles and liver during resting state and in the skeletal muscles during exercise and fever by contraction or frank shivering. On the other hand neonates primarily respond to cold stress by a non-shivering thermogenesis that takes place in the brown adipose tissue.Fever in neonates is defined as rectal or axillary temperature equals or more than 37°C, and it depends upon:1.Heat created by the metabolic processes of the body acting upon food and fluid ingestion.2.Physical activity which can quickly raise the metabolic rate and therefore body temperature.3.Heat absorbed by the body from the environment.4.Heat loss from the body via lungs and skin.The control of heat exchange is governed by complex physiological mechanisms. Ultimately from a centre situated in the hypothalamus, which also controls water balance and vasomotor and humeral activities aimed at maintaining the temperature level.Fever is a well recognised sign of disease. However, only the past three decades begun to be clarified. Studies in animals and in human beings have expanded knowledge of the pathogenesis of fever and more recently information has been obtained at the molecular level. It is unlikely that exogenous pyrogens, including infections agents or their products, exert a direct effect on these thermosensitive neurons, because the size and the molecular complexity of most exogenous pyrogens, such as bacterial endotoxins, would perclude their gaining access to the hypothalamus. However, a sufficiently small mediator substances could circulate during fever and affect these neurons. This mediator is called endogenous pyrogen. All the studies, along with the original observations in the rabbit, provide the corner stone for the current hypothesis of fever; that regardless of the stimulus fever is mediated by a substance of leukocytic origin. The preoptic region of the anterior hypothalamus is rich in thermosensitive neurons, which appear to control the consistency of the -body temperature, as well as the initiation of fever. Corticosteroids are potent anti inflammatory agents act on several levels; decreasing leukocyte motility, function and ability to produce leukocytic pyrogen as well as decreasing the hypothalamic responses to leukocytic pyrogen. The same degree of fever may be associated with a serious, life-threatening disease or a benign, self-limited illness. By far the most common cause of febrile illness in children is acute infection.The great majority of these are viral. The important and frequently difficult responsibility of the physian is to determine which febrile diseases are amenable to specific treatment. The

presence of fever is not in itself an indication for treatment. There is some evidence, although conflicting, to suggest that fever may play a role in natural body defenses. Moreover, the temperature course may be a valuable diagnostic and prognostic guide and should not be artificially masked with antipyretic drugs. Many components of the nonspecific host defense responses to infection such as leukocyte motility, lymphocyte transformation, and effects of interferon appear to be enhanced by elevations in temperature that simulate moderate fevers. In addition, some evidence indicates that a fever in conjunction with the changes in plasma iron levels known to occur during infection in synergistic host defense response. Only recently has the concept of using differential thermosensitivity been reintroduced as useful therapy, particularly for neoplastic cells and several viruses.Treatment of fever should be directed toward the cause. As fever in a greater part almost associated with infections, medication for fever is mostly medication for fever and infection, so an antibiotic is recommended in fever due to infection. Non-pharmacologic antipyretic measures should be directed toward maintaining adequate hydration, facilitating heat loss from the body, and combating heat retention caused by peripheral vasoconstriction and central pooling of the blood. Parents must be educated not to overdress or wrap in blankets a child with fever. In addition sponging with tepid water is a safe and effective way to increase heat loss by evaporation. When cautiously performed with tepid water the procedure is probably the safest one available for reducing febrile temperatures. On the other hand, excessively rapid reduction of fever can be dangerous, and has led to peripheral vascular collapse and death, espcially in critically ill infants.The recommended treating the child’s condition with antipyretic medications only if the temperature is over 39°C, and recommended sponging the child with tepid water only if the temperature is higher than 40°C as it has not respond to treatment with antipyretics and the patient is uncomfortable. Sponging must be done one hour after the administration of antipyretic to enable the hypothalamic set point to be lowered. Aspirin is a safe and effective antipyretic when used in appropriate doses. It has the drawback of not being available in liquid form, but it is supplied in the form of suppositories for younger children. The risk of accumulation of salicylate to toxic concentrations is increased in marginally hydrated or dehydrated febrile children who have impaired ability to excrete the drug. Controlled studies have shown no difference in antipyretic efficacy between aspirin and acetaminophen. However, acetaminophen has no anti-inflammatory activity and therefore is of -limited benefit in conditions in which a combination of antipyretic and anti inflammatory effect is desired.Acetaminophen poisoning is much less amenable to treatment than salicylate poisoning. The toxicity of acetaminophen is related to formation of highly reactive minor metabolites that convalently bind to cellular macromolecules and produce the cellular death. Oral administration of acetylcysteine (Mucomyst) appears to offer some protection against acetaminophen toxicity.