Proceeding of the NAVC
North American Veterinary Conference
Jan. 8-12, 2005, Orlando, Florida

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COOLING THE FEBRILE PATIENT – SHOULD YOU DO IT?

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INTRODUCTION

Fever is the result of an increase in the hypothalamic ‘set’ point for temperature control. As a result the body generates and conserves heat in an effort to maintain this higher temperature and any temperature lower than the new set point will be sensed as hypothermia. Technically any elevation in body temperature could be called hyperthermia, generally temperatures > 102.5ºF are considered elevated in dogs and cats. Clinically the term hyperthermia is reserved for all causes of an elevated body temperature other than fever. Hyperthermia results from excessive heat production, decreased heat loss and a loss of normal thermoregulation. Animals with hyperthermia sense they are hot and will usually attempt to maximize heat loss with behaviors like panting, agitation and seeking cool areas.

Thermoregulation is controlled by the anterior hypothalamus. It will stimulate changes in the body’s heat production and heat loss to maintain the ‘set’ temperature point. There is a specialized region of the blood brain barrier of the hypothalamus. The endothelial cells of this area produce prostaglandin E₂ (PGE₂) in response to circulating pyrogens. The prostaglandins diffuse into the anterior hypothalamus where they increase the temperature set point. Pyrogens can be considered endogenous such as tumor necrosis alpha, interferon and interleukin-1 or exogenous such as micro-organisms, micro-organism related products and toxins.

Antipyretic therapy is common practice in both veterinary and human medicine but the benefits of this approach is yet to be shown. Fever tends to be perceived as noxious and the resolution of fever is generally seen as a good sign. These beliefs have never been scientifically proven.

Fever may have many beneficial effects. Studies have found that it can reduce bacterial growth and reproduction and in some cases may cause bacterial death. Fever is believed to facilitate the immune response including increasing leukocyte activity and phagocytosis. It may also impair viral replication in some circumstances. There are both human clinical studies and experimental animal studies that have found increased survival rates in febrile subjects compared with afebrile subjects.

COOLING A FEVER - YES

Proponents of cooling febrile patients cite the benefits of reducing the metabolic demand, reducing myocardial and cerebral oxygen consumption, reducing vasodilation and improving patient comfort. These benefits would be particularly relevant to the patient with hemodynamic, respiratory or neurological compromise. There is some evidence that cooling a febrile patient will reduce oxygen consumption and may be associated with some improvement in outcome in human stroke patients. If cooling leads to a shivering response, most if not all these benefits will be lost. Sedation and possibly neuromuscular blockade may be necessary to prevent shivering. As for improving patient comfort, the few studies available in human medicine suggest reducing fever is does not improve patient welfare.

Extremely high body temperatures can be detrimental and intervention to keep temperatures less than 106ºF is a generally well accepted recommendation.

COOLING A FEVER - NO

Although not proven, there is evidence for the beneficial effects of the febrile response. In critically ill patients there is an argument to take advantage of this natural process. Until the exact role of fever is understood antipyretic therapy may be indicated in patients with cardiopulmonary or neurological compromise.

There are two approaches to antipyretic therapy, active cooling and pharmacological intervention. Active cooling has been found to be as equally efficacious as drug therapy but may not reduce energy expenditure to the same extent. Pharmacological therapy has associated side effects and in many cases these may outweigh the proposed benefits of reducing a fever. Both approaches increase patient care costs, this may be difficult to justify for a treatment choice that lacks a strong scientific basis.

ACTIVE COOLING

Active cooling can be achieved with cool water, ice packs, alcohol and fans. There has been no consistent approach to this therapy. Active cooling has been found to be as effective as any other therapy in lowering body temperature but in one recent study it did not reduce energy expenditure to the same extent as drug therapy did. Patients require constant temperature monitoring and active cooling needs to be stopped at a temperature of ~103ºF to prevent causing hypothermia. If active cooling leads to shivering most of the benefits of this therapy are lost and patient discomfort is inevitable.

PHARMACOLOGICAL THERAPY

Body temperature can also be lowered pharmacologically. The two commonly utilized antipyretic drugs are non-steroidal anti-inflammatory drugs (NSAIDs) and acetaminophen. The NSAIDs block prostaglandin production and have been found to be very effective at reducing fever. As the name indicates they alter the inflammatory response, it is yet to be determined if these effects are desirable. They can have gastrointestinal, renal and thrombocyopathic side effects, the risk of these problems need to be evaluated in each individual patient prior to administration. In many critically ill patients the risk of detrimental effects outweighs any likely benefits.

The exact action of acetaminophen is poorly understood. It is believed to have cyclo-oxygenase inhibitory effects leading to reduced prostaglandin production but with less anti-inflammatory effects than the NSAIDs. It is an effective antipyretic drug but in some studies it was found to be less potent then ibuprofen. Acetominophen can produce hepatotoxicity and should be used with caution in dogs, it is not recommended for cats.

REFERENCES