CHAPTER 7

POSTOPERATIVE FEVER IN THE PODIATRIC SURGICAL PATIENT

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The purpose of this pilot study is to investigate the significance of postoperative fever in patients undergoing elective foot surgery, and to determine whether or not a post-anesthetic core body temperature correction is noted in patients undergoing foot and ankle surgery. Moreover, we are interested in exploring the relationship between postoperative temperature elevation and the presence of clinical complication such as infection, pulmonary or urinary tract dysfunction, and other causes of fever. Often, the presence of a postoperative fever is alarming to the podiatric physician, and expensive diagnostic testing usually follows. This study examines the mechanisms behind fever, and hopes to establish preliminary guidelines for what is normal and abnormal. Data collection will determine the presence and magnitude of fever in a selected group of patients. Patient demographics, details of the surgical procedure, and the postoperative course will be studied in an attempt to seek correlation with postoperative fever.

REVIEW OF THE LITERATURE

Fever is defined as an increase in body temperature above the normal circadian range as a result of changes in the thermoregulatory center in the anterior hypothalamus.[1] Generally speaking, the hypothalamus maintains homeostasis by balancing heat production and heat dissipation. Any signal that affects the anterior hypothalamus may interfere with this balance. Fever has also been defined as an increase in the core temperature of an individual, best measured in the urinary bladder or lower esophagus.[2]

Body temperature is affected by many factors. The circadian rhythm refers to normal temperature fluctuations during a 24-hour period. Temperatures are generally lowest between 4-6 am, and highest between 4-6 PM. Exercise and environmental causes raise body temperature by insufficient heat dissipation (hyperthermia). In women, the AM temperature is usually lower during the two weeks before ovulation and rises one degree at ovulation through menses.[2] Many systemic diseases may also cause variations in body temperature. Diabetes causes immunosuppresion, which may not allow the body to mount an adequate febrile response.

The way that body temperature is recorded also causes variation. The mercury glass thermometer is the most commonly employed method of temperature monitoring, and has inherent sources of error including bore size and calibration. Disposable plastic thermometers, commonly employed in the office, have thermal sensitive color indicators. Inadvertent exposure to heat or cold may decrease the accuracy of temperature readings. Problems with the newer electronic thermometers involve their calibration and interference from disposable covers. Rectal temperatures are notoriously inaccurate due to their distance from arterial centers as well as the presence of heat producing microorganisms.[3]

Any substance that acts on the anterior hypothalamus and causes fever is broadly termed a pyrogen. Exogenous pyrogens are mainly due to microorganisms and their byproducts. For example, gram negative bacteria produce an endotoxin referred to as *LPS* that induces the production of other *endogenous* pyrogens. Some gram-positive organisms such as *Staph* and *Strept* produce pyrogenic exotoxins. Endogenous pyrogens are produced either systemically or locally by cells like macrophages and endothelial cells in response to infection or inflammation. Examples of endogenous pyrogens include interleukins 1 and 6, tumor necrosis factor alpha, and interferon gamma. When these substances are released into the blood stream they cause the release of arachidonic acid metabolites like PGE2 and cAMP from endothelial cells near the anterior hypothalamus. Signals are sent to the hypothalamus and the set point for body temperature is raised.

The preoptic anterior hypothalamus is responsible for temperature regulation and receives input from both peripheral nerves and from the temperature of the blood in the region. Once the set point is raised, the hypothalamus sends signals via the efferent nerves to conserve heat. Heat conserving mechanisms include sympathetic vasoconstriction, shivering, and behavioral changes such as posturing and moving to warmer environments. This

heat production continues until the blood bathing the anterior hypothalamic neurons matches the new setting.[1]

When the fever " breaks", the hypothalamus is in effect reset. This is due to either a decrease in production of pyrogenic cytokines or by inhibition of local prostaglandin synthesis by COX inhibitors or acetaminophen. The hypothalamus will then send signals to release heat causing vasodilatation, sweating, and behavioral changes. A balance is restored between heat production and heat dissipation.

POSTOPERATIVE FEVER

The first attempt to classify postoperative fever was Roe in 1968.[4] He described a transient drop in body temperature of 2 degrees in the immediate postoperative period as normal. This phenomenon is due to a continuation of intra-operative hypothermia. General anesthesia affects the anterior hypothalamus and interferes with the thermoregulatory mechanism. Postoperative hypothermia within the first twelve hours should simply be considered a continuation of intra-operative hypothermia. Sometimes this is manifested as shivering immediately postoperatively as the body tries to reach the altered set point.[5,6] The postoperative overshoot (see Fig I) refers to a temperature rise of less than 2 degrees within the first 24-29 hours, referred to as benign postoperative fever by Roe.[4] The set point was raised during anesthesia, and the body responds by trying to reach this new set point by mechanisms described earlier. Classically, atelectasis is described as causing fever within the first 24 hours following surgery wherein general anesthesia was used. In 1992, Engoren studied the association between atelectasis and fever. 100 postoperative cardiac patients were evaluated with chest radiographs for 2 days. They found the incidence of atelectasis increased by 79%, while the rate of fever decreased by 17%. No correlation was found between postoperative fevers and atelectasis.[7]

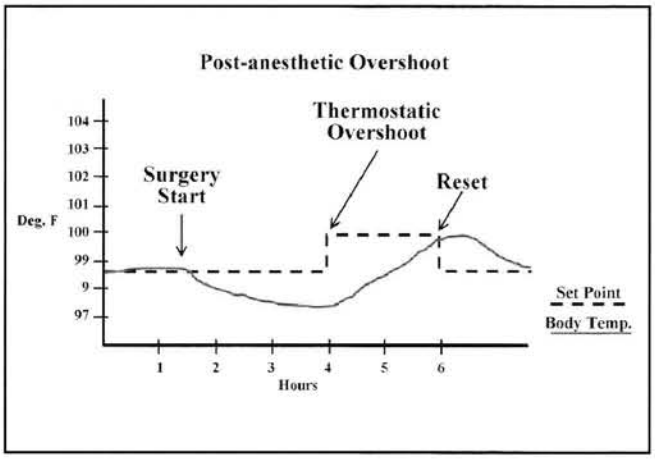

Figure 1. Post-anesthetic Temperature Correction.

Fevers that are caused by postoperative infection tend to occur later in the postoperative period. According to Perlino,[2] wound infections are the most common after any given procedure. The probability of getting a wound infection depends upon the type and length of surgery, whether the procedure is classified as clean or contaminated, and host status. Urinary tract infections are the second most common cause of postoperative infections and are highly associated with bladder catheter placement. Pneumonia is another likely source of infection, with risk factors including endotracheal intubation, inhibition of the cough reflex, and decreased motion of cilia.

Less common causes of postoperative fever include deep vein thrombosis, pulmonary embolism, medications, pancreatitis, and alcohol withdrawal.[2] When one is confronted with postoperative fever, it is imperative that a thorough examination be performed and all possible etiologies be considered.

STRESS-INDUCED FEVER

Recent studies suggest that the length and type of surgery may influence the development of a postoperative fever. A study by Kenan in 1986 analyzed 129 children undergoing 153 orthopedic operations for the significance of postoperative fever. 41% of these children spiked temperatures of greater than 38 degrees Celsius. They found an increased incidence of postoperative fever with surgeries that were greater than 1 hour in length, clubfoot releases, ORIF, and spine fusion.[8]

Guinn (1999) studied 100 patients undergoing knee replacements and 100 patients having hip replacements. They found that the greater the surgical trauma, the more likely postoperative fever will occur without an underlying infection. No significant difference was found between patients undergoing general or spinal anesthesia. They concluded that an immediate postoperative fever is part of the normal inflammatory response, and that a sepsis work-up is not necessary.[9]

Surgical trauma causes the release of inflammatory cytokines, most notably interleukin 6. Interleukin 6 acts as a pyrogen in post-surgical patients and the magnitude of the response is proportional to the amount of tissue injury. This cytokine then induces prostaglandin release at the anterior hypothalamus, causing an elevation of the thermostatic set point.[2] A study by Nishimoto in 1989 demonstrated that the degree of tissue injury and severity of procedure are proportional to increased levels of interleukin 6.[10]

Other potential mediators of the febrile response include postoperative pain, psychological stress, and the catecholamine response. In 1991, Briese experimented with rats and confirmed his hypothesis that a rise in the core temperature induced by stress is regulated, and due to a shift in the set point.[11]

Marraziti, in 1992, investigated the effect of psychological stress on the development of fever. He studied the body temperatures of 22 residents sitting for exams and again 3 weeks later in a calm situation. They found that healthy subjects undergoing stressful situations showed a rise in body temperature. They determined that these were true fevers and not due to physical activity. This study purports a possible relationship between the GABA (anxiety) system and the temperature regulatory mechanism.[12]

Several studies have shown that the mere presence of a postoperative fever is not indicative of infection. Guinn investigated the incidence and clinical significance of postoperative fever in 118 patients undergoing 141 total knee arthroplasties. Postoperative fever was found in 66% of 95 unilateral and 74% of 23 bilateral patients. Of these cases, only 14 developed clinical or laboratory findings to explain their pyrexia. They concluded that the presence of mild fever in the immediate postoperative phase should not be a contraindication to discharge.[9]

Than, et. al., investigated the incidence of postoperative fever in 177 patients who underwent total hip replacement. 39% of patients developed a fever greater than 38 degrees Celsius in the immediate postoperative period. 87% of these fevers were of unknown etiology. Only 4 of 177 patients had culture positive wound infections during the hospital stay, 3 of which had fever. They concluded that postoperative fever is only a minor predictor of wound infections. They discourage the use of antibiotics in the absence of clinical symptoms. Scwandt et al. concluded that an algorithm for the treatment of postoperative fever is safe and cost effective.[14]

PILOT STUDY

A retrospective cohort review of 66 randomized patients undergoing reconstructive foot and ankle surgery was performed. The maximum core temperature was recorded in each patient via infrared tympanic thermometry. Data was collected preoperatively, the day of surgery, and over the 3 postoperative days. Exclusion criteria included those patients with a preoperative temperature greater than 100.4 degrees Fahrenheit as well as those patients with a known source of infection.

The average age of the patients was 45.5 +- 17.8 years and were all graded as having class 1 "clean" wounds. Thirty-four patients were classified as ASA (American Society of Anesthesiologists) class I and thirty-two were ASA class II. The mean duration of surgery was 1.71 +- .99 hours. Anesthesia consisted of general, MAC/local, or spinal (58, 6, and 2 respectively). Twenty-nine patients spent 1 day in the hospital postoperatively, whereas 32 patients spent 2 postoperative days. Only five patients spent 3 or more postoperative days as an inpatient. There were no observed complications in any of the patients, and all used antipyretic medication during the postoperative course (acetaminophen, COX inhibitors).

Table 1 illustrates the type of procedures performed on the study cohort. Most of the patients underwent open reduction and internal fixation or major rearfoot/midfoot arthrodeses. Table 2 and Table 3 illustrate the results of data collection and statistical analysis. Defining fever as a core temperature greater than 100.4 degrees Fahrenheit, 14/66 patients developed pyresis the day of surgery. By postoperative day one, only 6/66 patients met the qualifications for fever.

DISCUSSION AND CONCLUSIONS

Several points are demonstrated by the results of this retrospective pilot study. First, post-anesthetic overshoot was observed (average 0.76 degrees) and normalized within four hours of emergence from anesthesia. This is lower than overshoots observed in abdominal and hip surgery. This most likely represents less tissue injury and the shorter duration of foot and ankle surgery.

Table 1

OPERATIVE PROCEDURES

Surgical Procedure	Number of Subjects
ORIF	26
Ankle/pantalar/triple or tarsal fusion	25
Forefoot reconstruction	4
Tarsal tunnel neuroplasty	1
Removal of deep metal fixation devices	3
Exostectomy	1
Neuroplasty (excluding tarsal tunnel)	2
Achilles tendon repair	3
Fasciotomy	1

Table 2

STUDY RESULTS: TMAX (OF)

Time period of temperature measurement	Mean and standard deviation of Tmax (°F)
Preoperative	98.27 +- 0 .90° F
Day of surgery	98.93 +- 1.65° F
First postoperative day	99.04 +- 0.97°F
Second postoperative day	98.8 +- 0.84°F
Third postoperative day	98.98 +- 0.53° F

Table 3

STUDY RESULTS: "FEVER" OR TEMPERATURE > 100.4°F

Time period of temperature measurement	Number (and %) of subjects displaying fever
Preoperative	2/66 (3.03%)
Day of surgery	14/66 (21.21%)
First postoperative day	6/66 (9.09%)
Second postoperative day	3/37 (8.11%)
Third postoperative day	1/5 (20%)

Subsequently, less inflammatory cytokines are released into the blood stream that interfere with hypothalamic thermoregulatory centers.

There was no observed correlation between fever and postoperative complications of any sort. Therefore, postoperative fever is neither sensitive nor specific for infection. The post-anesthetic overshoot occurs early and is relatively brief, and is limited by avoiding intra-operative hypothermia.

In conclusion, postoperative fever is defined as a regulated increase in Tcore associated with elevated body temperature and vasoconstriction. Causes of post-operative fever include the trauma of the surgery itself, the post-anesthetic overshoot, or the presence of infection. It is imperative that each patient be evaluated when a postoperative fever develops. However, having an understanding of the mechanisms behind postoperative fever will aid in the decision making process. If physical examination reveals signs and symptoms of infection, then necessary diagnostic testing must be initiated.

We are currently in the process of expanding our study to include both inpatients and outpatients on a prospective basis. We will also be investigating the influence of postoperative pain perception on the development of fever. The influence of different anesthetic agents on the development of post-anesthetic overshoot is another interesting area of study consideration.

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