Maternal fever in labor: etiologies, consequences, and clinical management

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Intrapartum fever is common and presents diagnostic and treatment dilemmas for the clinician. True maternal sepsis is rare; only an estimated 1.4% of women with clinical chorioamnionitis at term develop severe sepsis. However, the combination of inflammation and hyperthermia adversely impacts uterine contractility and, in turn, increases the risk for cesarean delivery and postpartum hemorrhage by 2- to 3-fold. For the neonate, the rates of encephalopathy or the need for therapeutic hypothermia have been reported to be higher with a maternal fever >39°C when compared with a temperature of 38°C to 39°C (1.1 vs 4.4%; $P<.01$). In a large cohort study, the combination of intrapartum fever and fetal acidosis was particularly detrimental. This suggests that intrapartum fever may lower the threshold for fetal hypoxic brain injury. Because fetal hypoxia is often difficult to predict or prevent, every effort should be made to reduce the risk for intrapartum fever. The duration of exposure to epidural analgesia and the length of labor in unmedicated women remain significant risk factors for intrapartum fever. Therefore, paying careful attention to maintaining labor progress can potentially reduce the rates of intrapartum fever and the risk for cesarean delivery if fever does occur. A recent, double-blind randomized trial of nulliparas at >36 weeks' gestation demonstrated that a high-dose oxytocin regimen (6×6 mU/min) when compared with a low-dose oxytocin regimen (2×2 mU/min) led to clinically meaningful reductions in the rate of intrapartum fever (10.4% vs 15.6%; risk rate, 0.67; 95% confidence interval, 0.48–0.92). When fever does occur, antibiotic treatment should be initiated promptly; acetaminophen may not be effective in reducing the maternal temperature. There is no evidence that reducing the duration of fetal exposure to intrapartum fever prevents known adverse neonatal outcomes. Therefore, intrapartum fever is not an indication for cesarean delivery to interrupt labor with the purpose of improving neonatal outcome. Finally, clinicians should be ready for the increased risk for postpartum hemorrhage and have uterotonic agents on hand at delivery to prevent delays in treatment.

Key words: chorioamnionitis, epidural-associated fever, fetal inflammatory response syndrome, hyperthermia, inflammation, interleukin 6, intrapartum fever

Introduction

Intrapartum fever is common and presents diagnostic and treatment dilemmas for the clinician. This review covers assessment of maternal temperature, normal temperature curves in labor, and the diagnosis of intrapartum fever. This report also examines the evaluation and management of cases with intrapartum fever, as well as the associated maternal and neonatal morbidities. The focus of the data and recommendations presented here is specific to cases with intrapartum fever at term. This is because the causes of maternal fever are different in preterm than in term labor with infection being in important contributor to fever occurring in preterm birth. Further, the effects of intrapartum fever on neonatal outcomes are different among term than among preterm populations, and it can be difficult to tease out the effects of gestational age drivers from those of intrapartum fever.

Assessment of temperature in labor

Maternal temperature is generally assessed every 4 hours in uncomplicated term labor with intact membranes and every 1 to 2 hours following rupture of membranes.[1] There are 2 purposes for maternal temperature assessments, namely (1) to identify patients at risk for increased maternal or neonatal morbidity and (2) to assess the degree of fetal exposure to hyperthermia. In the United States, oral and tympanic temperature assessments are the most common, and axial and rectal assessments are less common. Systematic comparisons of the different methods of temperature assessment suggested that oral maternal temperature assessment had the best correlation with direct measurements of intrauterine temperature.[2–6] Other studies suggested that oral and tympanic temperatures are equally accurate and relatively well correlated (Figure 1). In all reports, maternal temperature consistently underestimated the intrauterine temperature or fetal skin temperature by approximately 0.6°C to 0.8°C. In turn, fetal skin temperature may underestimate the fetal core temperature by up to 0.75°C. Therefore, a maternal temperature of 38°C may represent a fetal core temperature of up to 39.6°C, whereas a temperature of 39°C likely indicates a fetal core temperature of >40°C.

Temperature regulation in normal labor

Maternal temperature curves have been assessed in the absence of epidural analgesia or opioids to describe normal temperature regulation. In one cohort study, maternal temperature curves were described by the following formula:

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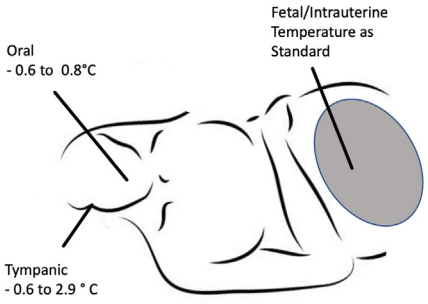
temperature = 36.6°C + (duration of labor in hours × 0.0064).[7] In nulliparous women, the mean axillary temperature increased from a mean of 37.08°C to a mean of 37.86°C over the course of labor.[8] In multiparous women, the temperature curves were similar; the mean axillary temperature increased from 36.94°C to 37.33°C. Fever does occur in the absence of epidural analgesia but the rates are low, even in nulliparous patients, at between 0.6% and 7%.[9–15] Some have hypothesized that the rates of intrapartum fever are reduced in women without epidural analgesia because of the antipyretic effect of maternal opioids. However, the evidence for this in laboring women is slim, and exposure to opioids does not seem to modulate fever risk among women with or without epidural analgesia.[16]

FIGURE 1
Concordance between maternal and intrauterine temperatures[2,5]



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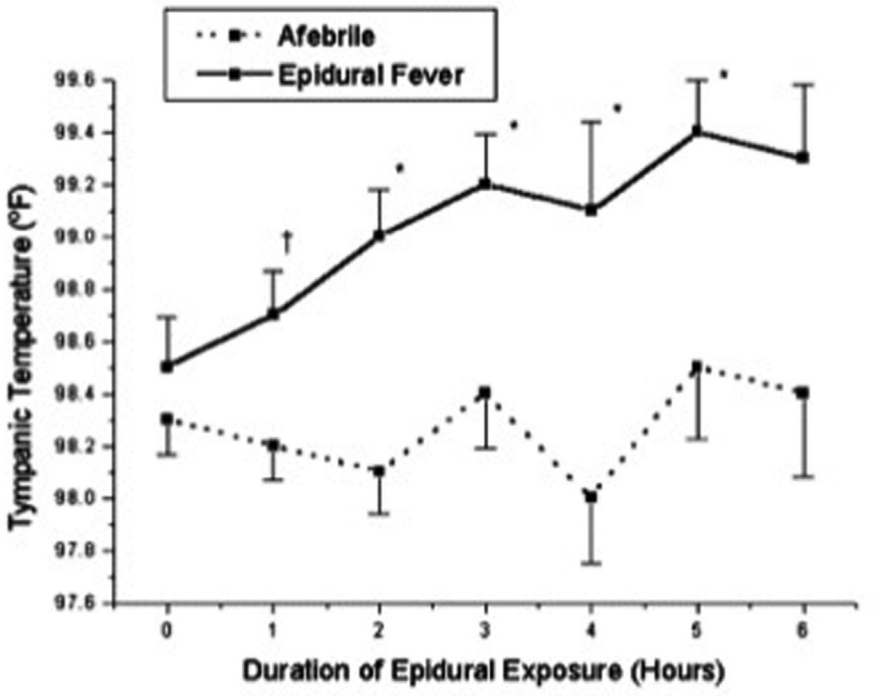
Definition of maternal fever

Given that the maternal temperature rises minimally during normal labor, all significant temperature increases likely represent some pathologic process—whether infectious or noninfectious. However, for clinical and research purposes, the definition of intrapartum fever can vary depending on which of the following is the ultimate goal: (1) identifying increased risks for adverse maternal outcomes; (2) identifying increased risks for adverse neonatal outcomes; or (3) setting a threshold for clinical intervention. Several publications have examined the differences in maternal and neonatal outcomes in 3 temperature groups, namely afebrile (<38°C), mild fever (38–39 °C) and severe fever (>39°C).[17,18] Although we will review the specific morbidities in detail hereafter (in the sections Effects of fever on maternal morbidity and Effects of fever on the fetus), it is reasonable to generalize that there is a dose-dependent increase in both maternal and neonatal morbidity. A mild fever is associated with a 3-fold increase in composite neonatal morbidity (adjusted odds ratio [aOR], 3.6; 95% confidence interval [CI], 2.26–4.42), and severe fever is associated with a 6-fold increase in composite neonatal morbidity (aOR, 6.24; 95% CI, 3.7–10.55). Composite maternal morbidity was increased to a similar degree with both mild and severe fever—approximately 3-fold (aOR, 2.96 and 3.16, respectively). Therefore, any temperature ≥38°C should raise concern for clinicians. The threshold for intervention should depend on whether a specific intervention improves maternal or neonatal outcomes coupled with a risk-benefit analysis of treatment based on the risk for potential maternal or neonatal harm. Based on a definition of fever as a temperature ≥38°C, the rates of intrapartum fever vary widely with the highest rates reported for nulliparas receiving epidural analgesia (11%–33%) and lower rates reported among multiparous patients with epidural analgesia (4%).[14] Key factors that influence a study population's fever risk include parity mix (increased with nulliparity), rates of epidural analgesia (increased with exposure to epidural analgesia[13,19–23] and with increasing duration of exposure to epidural analgesia[10,11,24]), racial and ethnic mix (increased with Hispanic ethnicity and among Asian or Pacific Islanders[25]), and practice patterns including oxytocin protocols (increased with standard-dose when compared with high-dose oxytocin regimens[26]).

Mechanisms of fever in labor

The majority of intrapartum fever has one of two mechanisms: infectious or epidural associated.[27] Infrequent etiologies include prostaglandin E2 or other drug exposure, dehydration, hyperthyroidism, or excess ambient heat. Both infectious and epidural-associated fevers have a primary inflammatory mechanism. In the setting of infection, the maternal immune response to infectious elements triggers the release of inflammatory cytokines including interleukins (IL) (1α, 1β, 6), tumor necrosis factor (α, β), and interferons.[28] The risk for clinical chorioamnionitis may be mediated by polymorphisms in the promotors of inflammatory cytokines.[29] One common finding in both infectious and noninfectious fever during labor is neutrophilic infiltration or inflammation of the placenta.[12,30,31] In addition, cytokine-mediated shivering may contribute to thermogenesis.[32,33] With epidural-associated fever, the stimulus for the inflammatory response is not clear. Epidural-associated fever is not commonly reported in clinical contexts (including women who are pregnant undergoing cesarean delivery) other than in labor; therefore, there is some combination of circumstances unique to labor the underlies the mechanism. Women who ultimately become febrile have distinct temperature responses to epidural placement, responding briskly and immediately (Figure 2).[24] In contrast, women who remain afebrile have little change in temperature; differences in temperature response are statistically different between groups as early as 1 hour following catheter placement, and clinical fever can appear within 4 hours of epidural placement.[10] If maternal temperature increases only in a small subset

FIGURE 2
Maternal temperature curves after epidural analgesia



Dagger indicates $P=.09$; *asterisk* indicates $P<.05$.[24]

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of women immediately following epidural placement, then potential biomarkers may be able to identify this subset. In this study, pre-epidural maternal IL-6 levels were significantly higher in women who later developed a fever (26.1±4.4 vs 88.9±21.8 pg/mL; $P=.01$). Other investigations have also reported an association between increased levels of inflammatory cytokines before epidural placement and subsequent fever risk or intrapartum fever following epidural placement; this suggests that risk may be linked to a preexisting inflammatory state.[12,34,35] Finally, epidural analgesia–associated fever can be almost completely suppressed by high-dose prophylactic parenteral methylprednisolone at the time of epidural placement but is not prevented by prophylactic antibiotics.[36,37] Despite this compelling evidence, not all cases of intrapartum fever in the setting of epidural analgesia are triggered by epidural placement. In one prospective cohort study, microbiologically proven chorioamnionitis, assessed using a combination of membrane culture and polymerase chain reaction for bacterial products, was present in 4.5% of cases overall and was not more common following epidural analgesia (4.7% vs 4.0%; $P>.99$). Similarly, most intrapartum fever cases seems to be noninfectious with only 5.4% demonstrating microbiologically proven chorioamnionitis. Others have reported a composite rate of 10.4% for either bacteremia or a positive placental culture following intrapartum fever.[38] These findings are consistent with the observed attributable risk of epidural analgesia to intrapartum fever of 94.5% to 96.2%.[9,10] One study of transabdominal amniocentesis in women with clinical chorioamnionitis at term suggested that 63% had a combination of detectable microorganisms (most commonly ureaplasma, *Gardnerella vaginalis*, roseolovirus, or herpes simplex virus) and elevated IL-6 levels.[39] However, a significant proportion had their amniocentesis before epidural analgesia, suggesting enrichment for infectious chorioamnionitis.

Effects of fever on maternal morbidity

There are 3 major adverse maternal effects linked to the processes associated with intrapartum fever, namely (1) infectious morbidity or sepsis, (2) excess antibiotic treatment because of the difficulties in discerning between infectious and noninfectious fever (detailed in Management of fever in labor hereafter), and (3) deleterious effects of inflammation and hyperthermia on uterine contractility that, in turn, increases the risk for cesarean delivery and postpartum hemorrhage. True maternal sepsis is rare; only an estimated 1.4% of women with clinical chorioamnionitis at term develop severe sepsis. Unfortunately, predicting which women will fall into this subset using clinical criteria is not particularly effective.[40] The rates of cesarean delivery and operative vaginal delivery rise in the setting of intrapartum fever; the OR is estimated to be between 1.9 and 3.3 for cesarean delivery and between 1.6 and 2.1 for operative vaginal delivery.[17,38,41–44] For cesarean delivery, there is no difference in the risk following a fever of >38°C to 39°C and one of >39°C (aOR, 0.56; 95% CI, 0.56–2.09).[17] Increased rates of cesarean delivery may be a consequence of changes in the fetal heart rate tracing secondary to fetal exposure to hyperthermia, physician anxiety, or direct effects on uterine contractility. Higher maternal serum levels of inflammatory cytokines are associated with slower active labor.[45] Arrest of dilation is associated with an increased expression of genes involved in inflammation and response to inflammatory cytokines and heat.[46] Inflammation is associated with myometrial apoptosis and down-regulation of the oxytocin receptor on the surface of myometrial cells.[47,48] In one report, uterine contractility was maintained for 2 hours after the onset of maternal fever but declined significantly and steadily thereafter by an average of 6.9±3.2 Montevideo units (MVU)/h ($P=.03$), despite the absence of a parallel decline in oxytocin exposure.[49] Multiparas and nulliparas showed a similar pattern of waning uterine contractility. Patients who

delivered vaginally maintained contractility as assessed by MVUs, whereas those who delivered via cesarean delivery had diminishing contractility. Similarly, rates of postpartum atony, hemorrhage, and blood transfusion are increased 2- to 3-fold in women with intrapartum fever.[17,41,50,51]

TABLE 1
Risk of neonatal encephalopathy based on intrapartum factors at term

	Afebrile	Intrapartum fever
Fetal acidosis (cord pH <7.05)	1.58%	12.50%
No fetal acidosis	0.12 %	1.58%

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Effects of fever on the fetus

In the setting of intrapartum fever, the fetus is often exposed to a combination of hyperthermia and inflammation and can also be exposed in some cases to infection.[52–54] The true incidence of infection can be difficult to determine because most cases of suspected or treated neonatal sepsis have been exposed to maternal antibiotics *in utero*. Only 0.7% of infants born in the setting of clinical chorioamnionitis have culture-proven, early-onset sepsis.[55] Regardless of whether an infection is present, hyperthermia and inflammation can both be deleterious to the term fetus or neonate either independently or in synergy. For the purposes of this review, the focus will be on long-term neonatal outcomes rather than on shorter-term neonatal outcomes such as antibiotic treatment, neonatal sepsis evaluation, admission to the neonatal intensive care unit, etc. This is because short-term outcomes may vary significantly because of local practice patterns and, although economically and socially significant, are unlikely to have as much long-term clinical significance as neurologic outcomes or perinatal mortality.

Neurologic outcomes

It is not unexpected that hyperthermia might be detrimental in terms of neurologic outcomes, especially if coupled with inflammation. Whole-body hypothermia is an effective treatment for neonatal ischemic encephalopathy.[56,57] The specific mechanisms of neuroprotection have not been elucidated fully but may include decreased metabolism or energy demand, reduced free radicals, reduced inflammation, and inhibition of excitotoxicity and cellular apoptosis.[58] Overall, maternal fever has been reported to be associated with an adjusted odds of neonatal encephalopathy of 3.8 to 4.7 in multiple reports.[59,60] Rates of encephalopathy or need for therapeutic hypothermia have been reported to be higher with a fever >39°C than with one of 38°C to 39°C (1.1 vs 4.4%; $P<.01$).[17] In one large cohort study, the combination of intrapartum fever and fetal acidosis was particularly detrimental.[61,62] This suggests that intrapartum fever may lower the threshold for fetal hypoxic brain injury (Table 1). Further, it is striking that the risk for neonatal encephalopathy is similar for either isolated acidosis or isolated intrapartum fever. The mechanism for this increased susceptibility is not known but is not mediated by an increase in fetal oxidative stress.[63] Placental neutrophilic infiltration may also increase the risk for subsequent acidosis.[64] Maternal fever is also associated with a subsequent increased risk for cerebral palsy (CP).[65] A maternal temperature >38°C in labor was associated with an increased risk for unexplained CP (OR, 9.3; 95% CI, 2.7–31) in a large population-based study of children born in 4 Californian counties in the 1980s. Clinical chorioamnionitis was also associated with an increased risk for CP at term in infants born within the Kaiser Permanente system in the 1990s (aOR, 3.8; 95% CI, 1.5–10.1).[66] The population-attributable fraction of chorioamnionitis or intrapartum fever to CP was 11%. A recent population-based study from Sweden linked chorioamnionitis or intrapartum fever to increased risks for CP (ajudsted hazard ratio [aHR], 7.43; 95% CI, 5.90–9.37).[67] Weak associations were also observed between chorioamnionitis or intrapartum fever and autism (aHR, 1.43; 95% CI, 1.21–1.68), attention-deficit/hyperactivity disorder (aHR, 1.17; 95% CI, 1.03–1.33), and intellectual disability (aHR, 1.99; 95% CI, 1.53–2.58).

Perinatal mortality

Intrapartum fever has been associated with an increased risk for perinatal mortality in several reports.[68,69] Intrapartum fever has also been reported to be associated with an increased risk for all-cause early neonatal mortality at term (OR, 1.7; 95% CI, 1.2–2.45). When stratified by parity, intrapartum fever was associated with an increased risk for asphyxia-related early neonatal death (OR, 2.81; 95% CI, 1.48–5.34) and infant death (OR, 2.42; 95% CI, 1.35–4.35). Intrapartum fever was also associated with infection-related neonatal death; this association may have been amplified by the low rates of intrapartum fever in this database (1.6%), suggesting a significant underreporting of isolated intrapartum fever.

Prophylaxis to prevent intrapartum fever

Prophylactic steroids

One randomized trial compared placebo treatment with low-dose (25 mg) and high-dose (100 mg) methylprednisolone treatment in a population at high risk for intrapartum fever (term nulliparas).[36] Intravenous (IV) steroids were administered at the time of epidural placement and then again at 8-hour intervals. Intrapartum fever occurred in 21.8% of women in the placebo group, in 34% of women in the low-dose steroid group, and in 2% of women in the high-dose steroid group. Although the high-dose steroid treatment decreased the incidence of epidural fever by 90% ($P<.001$) and significantly reduced the elevated umbilical cord blood inflammatory cytokines, it was associated with an unacceptably high rate of asymptomatic neonatal bacteremia (9.3%) detected using screening blood cultures performed as a safety endpoint. Subsequent

trials sought to replicate the positive effects of steroids while avoiding neonatal side effects. A small Chinese trial of epidural steroid administration (dexamethasone 0.2 mg/mL, continuous infusion) seemed to be effective in partially suppressing the maternal hyperthermia response.[70] Although there was no difference in the incidence of intrapartum fever, maternal temperature curves were somewhat dampened and maternal inflammation was blunted. A subsequent trial hypothesized that epidural steroids might be effective if the subclinical doses of epidural steroids were replaced with clinical doses (40−80 mg).[71] However, a US Food and Drug Administration black box warning[72] led to premature ending of the trial and no statistically significant reductions in maternal fever or inflammation were noted. Currently, steroids (either epidural or IV) are not recommended for the prevention of intrapartum fever.

Epidural technique

The technique and pharmacologic cocktail used at the time of epidural analgesia do not seem to alter the rates of subsequent fever.[6] Epidural fever occurs in the presence or absence of epidural opioids and with various local anesthetic agents at various concentrations.[73−77] One randomized trial reported lower maternal temperatures 4 hours after epidural placement with 0.075% ropivacaine when compared with 0.1% ropivacaine, but these results have not been replicated. There is no evidence that the rates of fever or exposure to inflammatory cytokines are altered with traditional epidural when compared with combined spinal epidural nor with patient-administered epidural boluses. No specific epidural technique has been recommended to reduce maternal fever or fetal inflammation.

Prophylactic antibiotics

Prophylactic antibiotic treatment does not alter the subsequent rate of intrapartum fever to a degree that is statistically or clinically significant, nor does it reduce the rates of placental inflammation.[37] Prophylactic antibiotic treatment is not recommended except in the setting of prevention of group B streptococcal infection.

Active labor management

Duration of exposure to epidural analgesia or length of labor in unmedicated women remains a significant risk factor for intrapartum fever.[10,11,24] Therefore, paying careful attention to maintaining labor progress can potentially reduce the rates of intrapartum fever. A recent double-blind randomized trial of nulliparas at >36 weeks' gestation demonstrated that a high-dose oxytocin regimen (6×6 milliunits/min) when compared with a low-dose oxytocin regimen (2×2 milliunits/min) led to clinically meaningful reductions in the rate of intrapartum fever (10.4% vs 15.6%; risk ratio, 0.67; 95% CI, 0.48−0.92)[26]; this may have been mediated by the significant reduction in the length of labor in the higher dose group. Of note, umbilical artery acidemia occurred significantly less frequently in the high-dose group although this benefit did not persist after adjustment. Further, there is no evidence to demonstrate that cervical examinations are an independent risk factor or causal factor for intrapartum fever after controlling for duration of labor.[26,78] Therefore, serial cervical examinations should not be avoided because they allow early detection of abnormal progression of labor and adoption of corrective measurements such as oxytocin administration.

Clinical evaluation in the setting of maternal fever

When maternal fever is diagnosed in labor, it is important to exclude rare but more dangerous etiologies that require intervention and specific therapies, such as meningitis, appendicitis, urosepsis, influenza, COVID-19, etc. Once nonobstetrical etiologies are excluded through a targeted physical examination and workup, little mental energy should be spent attempting to determine if the observed intrapartum fever is infectious or noninfectious. The standard clinical diagnosis of intraamniotic infection requires a maternal intrapartum temperature of ≥39°C or a maternal intrapartum temperature of 38°C to 38.9°C and 1 additional clinical risk factor, including maternal leukocytosis, purulent cervical drainage, or fetal tachycardia.[79] Practically, however, purulent cervical drainage is rarely seen and both maternal leukocytosis and fetal tachycardia are poor discriminators between infectious fever and noninfectious inflammatory fever. White blood cell counts are most strongly correlated with duration of labor.[80] Fetal tachycardia is commonly seen with fetal hyperthermia secondary to increased metabolic demands.[4] Further, neither fetal tachycardia nor maternal leukocytosis was associated with culture-proven infection at term (Table 2).[38] Even if small differences in these factors might have become statistically significant in a larger study, the positive and negative predictive values are clearly too low to be clinically

TABLE 2
Potential clinical indicators of chorioamnionitis

Indicator	Positive maternal blood or placental culture (n = 43)	Negative culture (n = 265)	*P* value
PPROM	32.6%	30.8%	.86
Maternal tachycardia >120 bpm	58.1%	46.2%	.19
Fetal tachycardia >160 bpm	69.8%	60.5%	.31
Maternal leukocytosis >15,000 cells/mm^3	60.5%	62.4%	.87

PPROM, preterm premature rupture of membranes.

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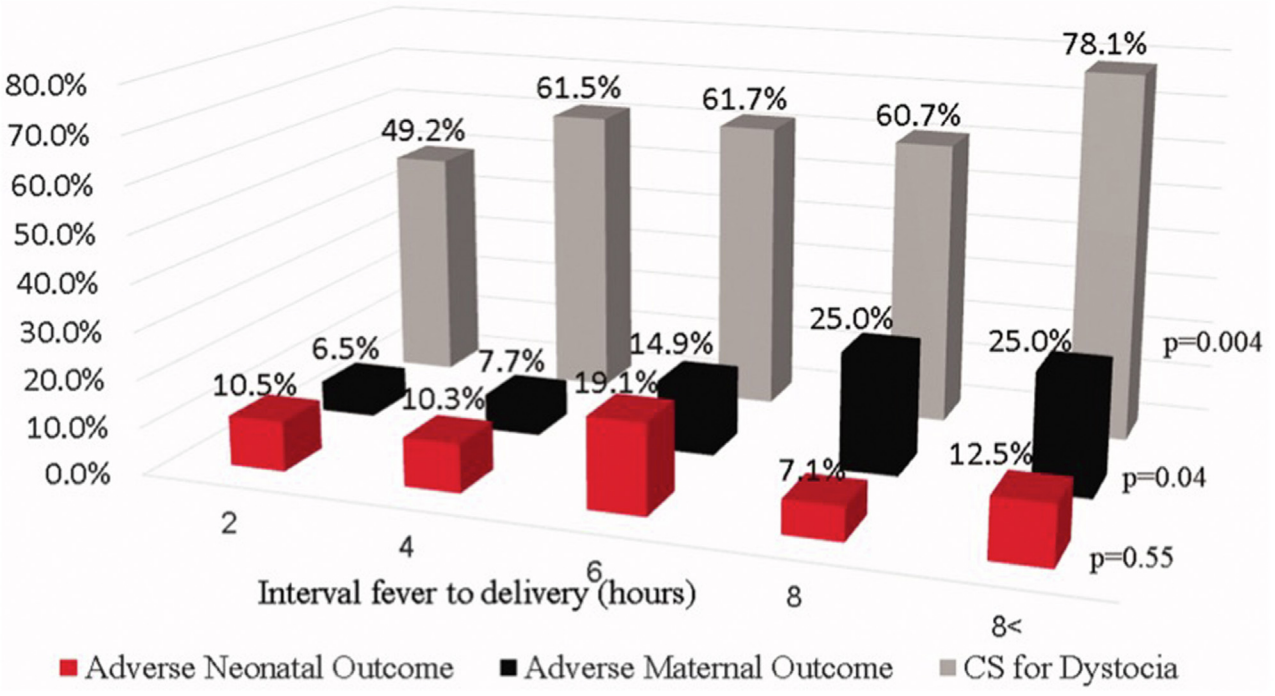
FIGURE 3
Adverse perinatal outcomes stratified by fever duration[38]



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useful. Similarly, poor discrimination was noted in a study with amniocentesis-based diagnosis of infection.[81] Although some studies have showed promise that biomarkers in amniotic fluid samples may be useful for discrimination, noninvasive tests would be more likely to be widely accepted clinically.[82,83]

Given these limitations, it is not surprising that the guidelines end with the following recommendation: isolated maternal temperature of 38°C to 38.9°C without apparent cause is not considered to be intraamniotic infection. However, given the potential benefits for both mother and fetus, if no obvious source for maternal temperature elevation is apparent, antibiotics are also recommended in the setting of isolated maternal fever.[79,84] Therefore, for all intents and purposes, the only criterion for treatment is intrapartum fever >38°C on one occasion (persists for 30 minutes). Treatment of isolated fever is more likely to signal a diagnosis of chorioamnionitis to the neonatology team and lead to neonatal treatment but may also reduce postpartum maternal fever.[85] Until better biomarkers are available to accurately sort intrapartum fever into infectious and noninfectious categories, treatment should be standardized and universal. Antibiotic treatment decreases the rate of neonatal bacteremia, pneumonia, and sepsis, albeit in the pre-epidural analgesia era when intrapartum fever was much more likely to be infectious.[86]

Management of fever in labor

Antibiotic protocols may vary by institution but typically should include ampicillin (2 g IV every 6 hours) and gentamycin (2 mg/kg loading dose, followed by 1.5 mg/kg every 8 hours).[84] In the setting of mild penicillin allergy, cefazolin (2 g IV every 8 hours) can be substituted for the ampicillin. In the setting of severe penicillin allergy, either clindamycin (900 mg IV every 6 hours) or vancomycin (1 g IV every 12 hours) can be substituted for ampicillin. Blood cultures should be obtained from women with clinical sepsis to help tailor antibiotic treatment. Prophylactic acetaminophen is ineffective in preventing frank intrapartum fever.[77,87] Acetaminophen treatment following the development of fever is of limited effectiveness and does not alter maternal or neonatal morbidity but is low risk.[2,17,88] IV acetaminophen is not superior to oral acetaminophen.[89] Intrapartum fever is associated with reduced uterine contractility and the majority of women with intrapartum fever will require oxytocin.[90] Uterine contractility may degrade over time and effects are seen within 2 hours following intrapartum fever.[49] Therefore, close attention should be paid to the uterine contraction pattern and consideration should be given to the placement of an intrauterine pressure catheter if clinically indicated to adequately address any diminution in uterine expulsive forces. Longer fever duration is associated with an increased risk for cesarean delivery (Figure 3).[38] Given the known increased risk for

postpartum hemorrhage and need for transfusion, it is prudent to have uterotonic agents at hand. Finally, there is no evidence that reducing the duration of fetal exposure to intrapartum fever prevents known adverse neonatal outcomes.[38,91] Therefore, intrapartum fever is not an indication for cesarean delivery to truncate labor with the purpose of improving neonatal outcome.[84]

Summary and recommendations

Intrapartum fever is a common occurrence, especially among nulliparous patients receiving epidural analgesia. Intrapartum fever generally has a noninfectious inflammatory origin, however, infection cannot be excluded with any available clinical or biochemical markers. Therefore, antibiotic treatment should be considered even with an isolated intrapartum fever of >38°C. Care should be taken to manage the immediate side effects of intrapartum fever, especially decreased uterine contractility. Paying close attention to contractility may ameliorate the known increased risk for cesarean delivery and/or operative vaginal delivery. Similarly, clinical awareness of the increased risk for postpartum hemorrhage may improve timely treatment. Finally, communication of intrapartum fever status to the neonatal team ensures appropriate neonatal management postpartum. ■

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