born to a mother infected with Zika virus. In other tropical countries in Southeast Asia, the situation is similar.2

This observation is consistent with the finding by Adhikari et al.1 Ronchetti and Bianco3 noted that there might be some unknown reasons for the high prevalence of abnormal infants born to Zika virus–infected mothers in South America.

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REPLY

We appreciate the interest in our recent report on infant outcomes associated with maternal Zika virus infection. We emphasize an important distinction in the epidemiological terms used in characterizing Zika virus spread: epidemic versus endemic. The current Zika epidemic has been primarily confined to the Americas and Pacific Islands, whereas endemic (a constant, low-level) infection has been established in Southeast Asia, including Thailand.3

In this context, endemic countries may have occasional clusters of cases but generally not in numbers large enough to be considered an outbreak. Thus, rates of new infections in endemic countries may be relatively low in a predominantly immune population. This differs from our report of Zika infections associated with travel to or from epidemic regions.

We agree, however, that many unanswered questions about the processes that lead to fetal infections in different populations remain. Although both French Polynesian and Brazilian strains of Zika virus have been shown to infect and replicate within placental and fetal tissues, an understanding of the specific factors that make this likely to occur in an individual patient are still elusive.2,3 We look forward to future research into understanding these factors influencing the burden of disease in different populations.

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Limits of current cardiotocography interpretation call for a major rethink

TO THE EDITORS: Clark et al1 should be commended for a robust retrospective case-control study in a very difficult area with important contributions to the current knowledge base, which should encourage debate and stimulate progress.

But their results were somewhat predictable.2,3 The study design description in the abstract seems confusing and needs clarification. Is not the condition (1) blind review/interpretation by experts and condition (2) secondary unblinded review of acrdemic babies by experts; both using the same published algorithm?3

Second, what system of cardiotocography (CTG) interpretation was used by the clinicians in actual practice in the authors’ institutes during the study period? Was it the same expert algorithm,3 especially in 2013? During the blind case-control review, the experts’ awareness of the high chance of CTG being abnormal (up to 50%) could increase detection to 45.8% (even without increasing the false-positive rate).

The paper acknowledges that under actual practice conditions, the detection rate of acrdemia would be closer to 30% even with the expert algorithm; thus little improvement. The main pessimistic conclusion about the “limits of CTG”
presumes that no improvement over the expert algorithm is possible in the identification of pathological fetal heart rate (FHR) patterns.

Justifiably, the singular focus of the expert algorithm is a more nuanced elucidation of FHR decelerations, under-scoring their maximal influence on CTG pattern recognition. This study showed that the abnormal decelerations were absent in 18% and present for less than 60 minutes in a further 18% of acidemic babies. But obviously there would have been different/milder decelerations that could be potentially recognized as pathological for longer than 60 minutes in many more cases. Moreover, fetal stimulation tests or fetal scalp blood sampling may not be perfect tests but could be useful adjuncts after more liberal identification of pathological decelerations.

Importantly, the experts need to be transparent that the current American categorization of FHR decelerations has distinctly deviated from that by Hon and in many ways contradictory. The current categorization essentially is rapid and gradual decelerations, the latter further divided into gradual early (not found) and gradual late. The majority of decelerations are classified as variable, supposedly because of cord compression underpinned by false incompatible hypotheses and there are almost no benign decelerations due to head-compression (or other nonhypoxic mechanisms). This may not be a benign digression/distortion because the most common cause of fetal hypoxemia in labor is contraction-induced reduction in uteroplacental perfusion, not cord compression at all. This “post-truth” framework fundamentally distorts the cognition/pattern recognition by clinicians setting oneself up for failure, may simply not be science, and will blight ensuing computerized interpretation as well. A “back-fire response” impedes progress. Lastly, alternative new technologies practically applicable on mass scale seem extremely remote.

This study calls for a major rethink (improved scientific pattern recognition of FHR decelerations and use of adjunctive test) rather than resignation.

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REPLY

We appreciate the author’s interest in our study but fail to understand the relevance of most of his comments to our data. We agree that a major rethink is necessary, but we believe the type of rethink the author appears to favor to be too timid. In reference to outmoded systems of care, the Institute of Medicine noted that “trying harder will not work. Changing systems of care will.”

We believe this observation is germane to the principal conclusion of our study, namely that we have wrung from Dr Hon’s visual fetal heart rate pattern recognition schemes all that we are likely to ever get. No matter how cleverly we tweak the terminology, this fact is unlikely to change.

The incorporation of these patterns into standard algorithms may improve outcomes. However, closing the remaining gap between observed and desired outcomes will require either computer-assisted identification of new patterns, perhaps not visually discernable, or the development of entirely new technological approaches to assess fetal brain oxygenation during labor.

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