

**“10 TIPS FOR OVERCOMING COMMON CHALLENGES OF INTRAPARTUM FETAL MONITORING”**  
 M. SEAN ESPLIN, MD, AND  
 ALEXANDRA G. ELLER, MD, MPH (MAY 2016)

**Determining fetal demise**

I appreciate and thank Drs. Esplin and Eller for their discussion of fetal monitoring pitfalls. I agree with their sentiment that this is an inexact science. After 40 years of looking at these strips, I am convinced there must be a better way. I look forward to some innovative approach to better determine fetal well-being in labor. This article raises a question I have asked, and sought the answer to, for years.

On occasion, I have diagnosed intrauterine fetal demise by detecting the maternal heart rate with an internal fetal scalp electrode. On one particular occasion, somewhere between the time of admission, spontaneous rupture of membranes, and applying the fetal scalp electrode, the fetus died. This case was similar to the one you describe in which early efforts with the external Doppler were unsatisfactory and fetal status was suspect. My question: “What is the time interval from the moment of fetal death and loss of fetal electrical activity until the fetus becomes an effective conduit for the conduction of the maternal cardiac signal? Is it minutes, hours, days? Clearly, this would be difficult to evaluate other than on animal models, but I have yet to find an answer.

**Edward Hall, MD**  
 Edgewood, Kentucky

**» Drs. Esplin and Eller respond**

*We are grateful for your interest in our article. Unfortunately the answer to your question about the timing between fetal demise and the appearance of maternal electrocardiac activity detected by a fetal scalp electrode*



MAY 2016

*after transmission through the fetal body is not clear. We are not aware of any data that would conclusively prove the time required for this to occur. It is likely that this type of information would require an animal model to elucidate. However, we are aware of at least 2 clinical cases in which fetal cardiac activity was convincingly documented at admission and for several hours intrapartum with subsequent episodic loss of signal and then delivery of a dead fetus wherein retrospective review confirmed that for a period of time the maternal heart rate was recorded and interpreted to be the fetal heart rate. From these experiences we conclude that this is possible shortly after the fetal demise, likely within minutes to hours.*

*Despite this uncertainty, we are confident that the information in our article will help clinicians identify and correct those instances when the maternal heart rate is being recorded instead of the fetal heart rate. Fortunately, this rarely involves a situation in which there has been an undiagnosed intrauterine fetal demise.*

**“SERMS IN MENOPAUSE: MATCHING AGENTS TO PATIENTS’ SYMPTOMS AND ATTRIBUTES”**  
 JAMES H. LIU, MD, AND GRETCHEN  
 COLLINS, MD (MAY 2016 SPECIAL ISSUE)

**“SERMs” definition inaccurate**

I disagree with Drs. Liu and Collins’ description of selective estrogen receptor modulators (SERMs) on page S18, in which they state, “Estrogens and SERMs are lipid-soluble steroid hormones that bind to 2 specific hormone receptors, estrogen receptor  $\alpha$  and estrogen receptor  $\beta$ ...” SERMs are not hormones, and they are defined improperly as such.

**Gideon G. Panter, MD**  
 New York, New York

**» Drs. Liu and Collins respond**

*Thank you for your interest in our article. SERMs are typically synthetic organic compounds that can activate estrogen receptors or modify activity of the estrogen receptor and, thus, can be considered hormones.*

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## Comment & Controversy

CONTINUED FROM PAGE 14

### “START OFFERING ASPIRIN TO PREGNANT WOMEN AT HIGH RISK FOR PREECLAMPSIA”

ROBERT L. BARBIERI, MD  
(EDITORIAL; MAY 2016)

#### Stop aspirin in pregnancy?

Like many colleagues, I had been stopping low-dose aspirin prior to planned or expected delivery. Evidence suggests a bigger risk of rebound hypercoagulability than bleeding after stopping low-dose aspirin, according to an article on aspirin use in the perioperative period.<sup>1</sup> Because of lack of benefit and increased risks of stopping aspirin, it may be time to change our practice and continue aspirin to minimize peridelivery thromboembolic risk.

**Mark Jacobs, MD**  
Mill Valley, CA

#### Reference

1. Gerstein NS, Schulman PM, Gerstein WH, Petersen TR, Tawil I. Should more patients continue aspirin therapy perioperatively?: clinical impact of aspirin withdrawal syndrome. *Ann Surg.* 2012;255:811-819.

#### » Dr. Barbieri responds

*I thank Dr. Jacobs for his advice to continue low-dose aspirin throughout pregnancy in women taking aspirin for prevention of preeclampsia. The review he references is focused on elderly patients taking aspirin for existing heart disease, which is a very different population than pregnant women. There are no high-quality data from clinical trials on whether to continue or stop low-dose aspirin in pregnant women as they approach their due date. I think obstetricians can use their best judgment in making the decision*

*of whether to stop low-dose aspirin at 36 or 37 weeks or continue aspirin throughout the pregnancy.*

### “CESAREAN SCAR DEFECT: WHAT IS IT AND HOW SHOULD IT BE TREATED?”

CAMRAN NEZHAT, MD; LINDSEY GRACE, MD;  
ROSE SOLIEMANNJAD, BS;  
GITY MESHKAT RAZAVI, MD; AND  
AZADEH NEZHAT, MD (APRIL 2016)

#### Technique for preventing cesarean scar defect

I read with interest the proposed treatment options that Dr. Nezhat and colleagues suggested for cesarean scar defect. However, nowhere did I see mention of preventing this defect.

For 30 years I have been closing

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## Comment & Controversy

CONTINUED FROM PAGE 17

the hysterotomy in a fashion that I believe leaves no presence of an isthmocele and is a superior closure. I overlap the upper flap with the lower flap and, most importantly, close with chromic catgut. A cesarean scar “niche” occurs with involution of the uterus causing the suture line to bunch up. Chromic catgut has a shorter half-life and will “give;” a suture made of polypropylene will not stretch. I use a running interlocking line with sutures about 0.5 inches apart.

**Donald M. Werner, MD**  
Binghamton, New York

**» Dr. Nezhat and colleagues respond**  
*We thank Dr. Werner for his inquiry regarding the prevention of cesarean scar defects; as we all agree, the best treatment is prevention. As mentioned in our article, there are no definitive results from the studies published to date that show superiority of one surgical technique over another in regard to hysterotomy closure and prevention of cesarean scar defects. Possible risk factors for developing cesarean scar defects include low (cervical) hysterotomy, single-layer uterine wall closure, use of locking*

*sutures, closure of hysterotomy with endometrial-sparing technique, and multiple cesarean deliveries. Although these factors may be associated with increased risk of cesarean scar defects, additional randomized controlled trials need to be performed prior to being able to offer a recommendation on a conclusive preventative measure. For additional information, I would direct you to references 3 and 4 in our article. We thank you for sharing your positive experience and eagerly await additional studies on the topic.*