Simulation of late decelerations in labor with a mathematical model

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Introduction
Late decelerations (LD) in the fetal heart rate are often seen during labor on the cardiotocogram (CTG, i.e. continuous registration of fetal heart rate (FHR) and uterine contractions). They are caused by uteroplacental insufficiency (UPI): the lack of placental capacity to fulfill oxygen requirements of the fetus, evokes a reflex cascade that eventually lowers FHR temporarily. UPI can be caused from e.g. blood volume reduction, resistance increase or diffusion capacity reduction. To obtain a tool for training insight into physiology and to increase realism of obstetric team training, we included a physiology-based scenario of LD in our cardiovascular simulation model [1].

Methods
The mechanism of LD was studied and cardiovascular regulation [2] was implemented in the existing model (figure 1). FHR response was simulated for:
• normal labor (reference contraction $p_{ut,ref}$),
• UPI ($p_{ut,ref}$ with 50% $V_{d,ut}$ or with 200% $R_{ut,0}$ or with 50% $D$),
• blocked pathways ($\alpha$, $\beta$-sympathetic and vagal nerve).

Results
Late decelerations follow from the model after uterine contractions are applied. Results (figure 2) are in accordance with reported literature response [3-5] for normal labor, UPI and the blocked pathways.

Discussion
The model thus links utero-placental insufficiency in labor to late decelerations in the CTG trace. The fetus uses flow redistribution to maintain oxygen delivery to the brain in expense of the other organs. In addition, the brainsparing mechanism also reduces FHR until new oxygen is available. The model reveals a fragile balance between cardioaccelerator and -decelerator pathways during the blockade studies. After validation by clinical experts, the model can be used in an educational simulator.

Figure 1: Model interactions for late decelerations scenario.
Figure 2: Late decelerations cascade calculated by the model. The reference contraction $p_{ut,ref}$ (dotted line) evokes a late deceleration. UPI leads to lower $p_{ut}$ and thus to altered FHR signals: lower baseline $pO_2$ leads to lower baseline FHR. Small changes in $pO_2$ lead to small changes in FHR (due to saturation of the system). MAP is almost not affected. In the blocking studies, the late deceleration is altered (reduced, increased or changed into a late acceleration) due to the altered balance between cardioaccelerator and -decelerator input to heart rate. MAP cannot be maintained if either one of the pathways is blocked. For color legend: see figure 1.