8. Workshop Automatisierungstechnische Verfahren für die Medizin vom 20.-21. März 2009 in Berlin

„Model Based Analysis of the Fetal Heart Rate Traces During Birth“

Mario Šikić, Sabina Eipe, Peter Wolf, Robert Riener
Sensory-Motor Systems Lab, Institute of Robotics and Intelligent Systems (IRIS), ETH Zurich, Switzerland
E-Mail: sikic@mavt.ethz.ch

Robert Riener
Spinal Cord Injury Center, University Hospital Balgrist, Zurich, Switzerland

Band: Fortschritt-Bericht VDI Reihe 17 Nr. 274 „Automatisierungstechnische Verfahren für die Medizin, 8. Workshop, Tagungsband“
Editors: Thomas Schauer, Henning Schmidt, Marc Kraft
ISBN: 978-3-18-327417-8
Pages: 11-12

Model Based Analysis of the Fetal Heart Rate Traces During Birth

Mario Šikić[1], Sabina Eipe[1], Peter Wolf[1], Robert Riener[1,2]

[1] Sensory-Motor Systems Lab, Institute of Robotics and Intelligent Systems (IRIS), ETH Zurich, Switzerland
[2] Spinal Cord Injury Center, University Hospital Balgrist, University of Zurich, Switzerland
Contact: sikic@mavt.ethz.ch

Introduction

Childbirth is often accompanied by more or less pronounced hypoxia of the fetus which bears the risk of cerebral damage. The fetus physiology has mechanisms to defend against the consequences of this hypoxia. Our knowledge about these mechanisms is mainly based on animal studies, where responses to normal as well as pathological conditions of birth were measured. We, therefore, know that the goal of the fetal physiological responses is to maintain the oxygen and nutrient supply to vital organs, especially the brain, heart, and adrenal glands. Thus, hypoxia of the fetus should be observable from the physiological signals during birth. In the clinical setting these measurements are usually limited to fetal heart rate. These give high sensitivity, but relatively low specificity on the fetal condition. This results in many false positive diagnoses for fetal distress. The search for an optimal estimator of fetal well being is still in progress.

The available range of measurements in human fetuses is limited, and the full range of measurements done on animals for research purposes cannot be performed in real human births due to their invasive nature. Today, the only remaining diagnostic device regularly used during labor is the cardiotocogram. It comprises of a Doppler ultrasound probe, used to measure fetal heart rate, and a pressure sensor for observing the contraction intensity, i.e. uterus muscle firmness, which correlates to the intrauterine pressure. The basic hypothesis behind those measurements is that signs of fetal distress would be observable on the heart rate traces. Other measuring devices used for diagnostics during labor, like electrocardiography and oxymetry, were applied with less success [1].

The starting point of this work is the question: can one infer on the fetal state from the measurements done routinely during the birth? Experience with the cardiotocography has shown that this is possible to a certain extent, and better results could probably be achieved using model-based estimation. To devise models of sufficient complexity that are at the same time applicable to clinical settings several problems exist:

- Models of sufficient complexity involve a significant number of parameters,
- A restricted number of non-invasively measurable quantities,
- Intrauterine conditions cannot be controlled, and
- Individuals vary widely in the relative responses to stimuli.

Considering these limitations, it is unreasonable to ask for this estimator of fetal state, which would give us quantitative results from the little information that is available. We can, however, use mathematical modeling to answer more immediate questions about fetal physiology during birth.

In this paper we take theoretical heart rate traces of the fetus (accelerations, decelerations) during uterine contractions, and investigate their consequences on the fetus. More precisely, we investigate both the influence of frequency of uterine contractions and the response of the fetal heart rate to those on minimal oxygen partial pressure in the mixed arterial blood [2]. Minimal oxygen concentration is a reasonable measure of fetal well–being, since it indicates the amount of available oxygen in the fetal blood.

Materials and methods

The model

For our investigation, a model of the fetal cardiovascular system including oxygen transfer and metabolism was developed, based on other models [3,4].

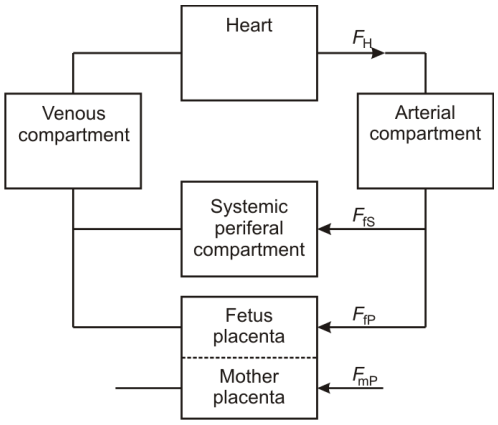

Fig. 1: Block diagram of the cardiovascular system used. *F* refers to flow, where the indexes H, fS, fP, and mP stand for heart, fetal system, fetal side placenta, and mother side placenta respectively.

The fetal cardiovascular system is presented as one systemic circuit comprising of one arterial and one venous compartment. The compartments are considered to be compliant vessels, exhibiting no resistance to blood flow and characterized by the pressure – volume relation, here assumed to be linear. We use three resistance

compartments: one for the fetal muscle tissue and other organs, one for fetus side placenta and umbilical cord, and one for the mother side placenta. These compartments are modeled as linear elements, defined by its vascular resistance, i.e. flow depends linearly on the pressure drop on the compartment.

The heart model has to account for dynamic changes in heart pumping capacity, since the heart rate is not constant. Dependence on heart filling pressure is included according to literature [3]. Pulsatile flow is not included since the time frame we are interested in, i.e. time of one contraction, is much longer than the heart cycle. Thus, only mean values of quantities over one heart cycle are presented.

The influence of the amniotic fluid pressure on the blood pressure in the fetal vessels is neglected. The uterine blood flow is assumed to be dependent on contraction intensity, with zero placenta perfusion at 40 mmHg of amniotic fluid pressure [5]. The contractions are standardized to duration of 2 minutes and to a peak pressure of 60 mmHg.

Oxygen transfer in the placenta is modeled according to existing models [9]. Oxygen expenditure is modeled as first order diffusion compartments with fixed oxygen removal rate, i.e. metabolic rate [4]. So, in our model, we set the metabolic rate to the maximal value in the fetus.

Heart rate response was modeled as a first order system of contractions. We assume that heart rate response is not dependent on oxygen availability. The accelerations and decelerations were chosen to be 20 beats per minute above, i.e. below, the baseline.

Model verification

The model parameter data set for the human fetus was compiled from various sources in the literature [4,5,6,7]. Model verification has been performed as follows:

- Without contractions, the model converges to the steady state values given in literature [4].
- The model output compares well to the measured data of various experiments, e.g. [8].

Results

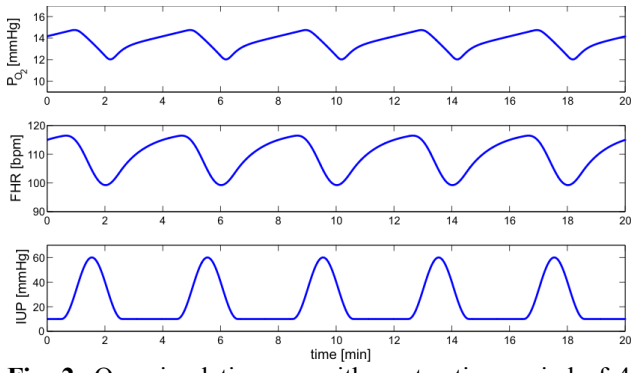

Fig. 2: One simulation run with contraction period of 4 minutes and heart rate decelerations. Displayed are oxygen partial pressure of the arterial blood (P_{O_2}), fetal heart rate (FHR), and contraction intensity (IUP).

The model was applied with different responses of the heart rate (Fig. 2). The minimum oxygen saturation in the fetus arterial blood depends on the heart rate response (Fig. 3), two main causes being high oxygen consumption of the placenta, and lower cardiac output. Note a steep decrease of the minimal arterial oxygen partial pressure as the period of contractions is low. Therefore, fetal oxygen availability is sensitive to period of contractions if that is below 3 min, in agreement with the measurements [2].

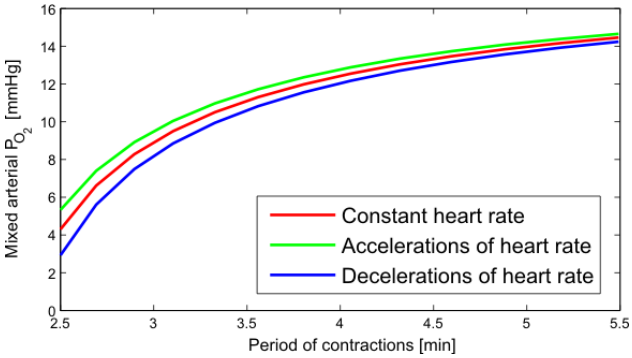

Fig. 3: The dependence of the minimum of oxygen partial pressure in the fetal arterial blood as a function of contraction period. Steady state values are presented.

Discussion and Conclusion

The minimal partial pressure of oxygen in the arterial blood depends on the pause between the contractions and the response of the heart rate. So, given a response of the heart rate, we can define the contractions to be "safe" if the oxygen partial pressure does not fall below a certain threshold (Fig. 3). Our assumption of fixed oxygen expenditure gives us quite a conservative estimate of safe contraction frequency.

Literature

[1] STEER, P.J. : Has electronic fetal heart rate monitoring made a difference? *Semin Fetal Neonat Med* **13** (2008), S. 2 – 7

[2] PEEBLES, D.M. ; O'BRIEN, P.: Fetal Cerebral Oxygenation and Hemodynamics During Labour Measured by Near-infrared Spectroscopy, MRDD Research Reviews 3:59–68, 1997

[3] KAPPEL, F. ; PEER, R.O.: A mathematical model for fundamental regulation processes in the cardiovascular system. *J Math Biol* (1993) **31**, S. 611–631

[4] HUIKESHOVEN, F.J. ; HOPE, I.D. ; POWER, G.G. ; GILBERT, R.D. ; LONGO, L.D.: Mathematical model of fetal circulation and oxygen delivery. *Am J Physiol* **249** (1985) 18, S. R192–R202

[5] DATTA, S.: Obstetric Anesthesia Handbook, 4th ed. Springer, 2006

[6] GUYTON, A.C. ; HALL, J.E.: Textbook of Medical Physiology, 10th ed., W.B. Saunders Company, 2000

[7] KISERUD, T. : Physiology of fetal circulation. *Semin Fetal Neonat Med* **10** (2005), S. 493 – 503

[8] ITSKOVITZ, J. ; LAGAMMA, E.F. ; RUDOLPH, A.M.: Effects of cord compression on fetal blood flow distribution and O_2 delivery. *Am J Physiol* **252** (1987), S. H100–H109

[9] HILL, E.P. ; POWER, G.G. ; LONGO, L.D.: A mathematical model of carbon dioxide transfer in the placenta and its interaction with oxygen. *Am J Physiol* **242** (1973) 2, S. 283–299