The Hemodynamics of Labor in Women Undergoing Vaginal and Cesarean Deliveries as Determined by Whole Body Bioimpedance

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Abstract

Objective The objective of this study was to assess the hemodynamics of labor, delivery, and 48 hours postpartum in women undergoing vaginal and cesarean deliveries by utilizing a whole body bioimpedance-based device.

Methods A prospective longitudinal single-center observational study was performed between September 2014 and September 2015. The hemodynamics of low-risk women undergoing spontaneous vaginal delivery were compared with those undergoing elective cesarean sections. Cardiac index (CI), stroke index, total peripheral resistance index (TPRI), and mean arterial pressure (MAP) were assessed at different time points during delivery and in the immediate postpartum period (1, 24, and 48 hours postpartum).

Results Eighty-seven women were evaluated, 63 parturients in the vaginal delivery group and 24 in the cesarean delivery group. Normal vaginal delivery was characterized by a reduction in MAP and CI after epidural anesthesia, whereas elective cesarean sections were characterized by a rise in MAP and CI after spinal anesthesia. As labor progressed, CI increased reaching its peak during the second stage. Immediately following delivery, TPRI declined to its nadir with no significant change in CI. As opposed to vaginal delivery, in cesarean delivery, TPRI peaked within 1-hour postpartum resulting in a significant decline in CI.

Conclusion Whole body bioimpedance can be used effectively to assess the hemodynamics of vaginal and cesarean deliveries.

Keywords
- bioimpedance
- cesarean
- delivery
- NICaS
- vaginal
- whole body

The physiologic demands of pregnancy cause adaptive changes in the cardiovascular system, resulting in an increase in cardiac output (CO), peaking during labor, and the immediate puerperium.[1,2] In healthy women, these changes are well tolerated. However, in certain situations, such as preexisting cardiac diseases, intrapartum and postpartum hemorrhage (PPH), and preeclampsia, maternal morbidity may rise.

Studies examining the hemodynamics of labor have reported conflicting results. Some showed an increase in basal CO during the first stage of labor[2,3] and in between

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contractions,[4] while others showed such an increase primarily during contractions.[5] In addition, inconsistencies were found among different reports regarding the degree of rise in CO during contractions[6,7] and the effect of epidural anesthesia on the decrease in CO varies as well in different studies.[8,9] Finally, while there is an agreement that CO decreases in the puerperium, the exact time of decline remains controversial.[10–12] These discrepancies are most likely a result of different investigative techniques, invasive (dye dilution[13,14] and thermodilution[15]) and noninvasive (echocardiography[16] and tissue Doppler imaging[17]), and differences in study designs and small sample sizes.[2]

The gold standard for measurements of CO and associated hemodynamic variables remains the invasive thermodilution method,[18] despite its obvious shortcomings. Noninvasive methods, including echocardiography and tissue Doppler imaging, though valid in pregnancy, are operator dependent and their use in the laboring woman is impractical. Thoracic bioimpedance-based devices, though validated in pregnancy and the postpartum period,[19–24] are inconvenient to use during labor, due to their attachment to the abdomen, neck, and thorax. In contrast to this inconvenient configuration, the whole body bioimpedance-based device is a validated device in the different patient populations, such as cardiac patients[25] and patients with pulmonary hypertension.[26] Recently, it has been used successfully in healthy patients with low-risk pregnancies undergoing elective cesarean sections, demonstrating feasibility of this technique.[27] It is relatively easy to use, and its wrist-to-wrist configuration is practical for the laboring woman, enabling continuous monitoring.

In this study, we sought to assess the hemodynamics of labor, delivery, and the first 48 hours postpartum in low-risk women both in normal vaginal and elective cesarean deliveries as determined by whole body bioimpedance.

Methods

Study Population

Women were eligible to participate in the study if they were 18 years or older, healthy, nonsmokers, with a low-risk, singleton term pregnancy (37–41 weeks of gestation). Exclusion criteria were any of the following: multiple gestation, hypertensive disorders (pregnancy-induced hypertension, chronic hypertension, or preeclampsia), diabetes mellitus, small for gestational age fetuses (estimated fetal weight $<10^{th}$ percentile according to local growth curves),[28] hypermetabolic states (e.g., thyroid disease), or known cardiovascular diseases.

Study Design

A prospective longitudinal single-center observational study was performed between September 2014 and September 2015. Two populations of parturients were studied and compared: women undergoing a spontaneous vaginal delivery with epidural anesthesia and women undergoing an elective cesarean section with spinal anesthesia. All women meeting inclusion criteria and willing to participate in the study were connected by two electrodes (one on each wrist) while supine for a time period of 6 minutes at predetermined points in time, as follows:

1. Group A—women undergoing vaginal delivery first stage latent phase of labor (defined as up to 3 cm dilatation and up to 80% effacement—first connection), before (second connection) and after (third connection) epidural anesthesia, first stage active phase of labor (defined as up to 10 cm dilatation—fourth connection), second stage of labor (from full dilatation to fetal expulsion—fifth connection) and postpartum—1 hour (sixth connection), 24 hours (seventh connection), and 48 hours after delivery (eighth connection).
2. Group B—women undergoing elective cesarean delivery—immediately before entering the operating theater (first connection), after administration of spinal anesthesia (second connection), during the operation immediately after fetal and placental removal (third connection) and postpartum—1 hour (fourth connection), 24 hours (fifth connection), and 48 hours after delivery (sixth connection).

The followings parameters were registered by the attending physician who performed the measurements: maternal age, pregestational body mass index (BMI), current weight, blood pressure (measured with standard sphygmomanometers used routinely in the delivery rooms and operating theaters), body position, hematocrit, Na concentration, and oxygen saturation as determined by pulse oximetry. Fetal heart rate recordings were obtained simultaneously, if needed.

All participants were recruited before the onset of active labor and gave informed consent. The study was approved by the Institutional Review Board.

NICaS Device Description

The noninvasive CO monitoring system (NICaS, New NI Medical, Petah Tikva, Israel) evaluates hemodynamic parameters according to whole body bioimpedance. This portable, easy-to-use device, which is unaffected by BMI, requires no operator skill and is nonoperator dependent.[29] It connects to the patient in a simple wrist-to-wrist or wrist-to-ankle configuration and delivers a minor electrical current unsensed by the patient. With each heartbeat, the volume of blood in the arterial system changes resulting in a change in the body's impedance. These changes are measured by the NICaS. CO, as well as additional hemodynamic and respiratory parameters (stroke volume, stroke index [SI], heart rate, cardiac index [CI], cardiac power index, respiratory rate, total peripheral resistance, and total body water), are calculated by proprietary algorithms. In addition, NICaS registers a one channel electrocardiogram.

Data Collection

The following data were collected for each participant: demographic and obstetric characteristics pertaining to previous pregnancies and the index gestation and hemodynamic parameters retrieved—CI (CO relative to body surface area of the patient), mean arterial pressure (MAP), stroke index (SI), and total peripheral resistance index (TPRI).

Table 1 Baseline characteristics of parturients participating in the study

	Vaginal deliver $n = 63$	Cesarean section $n = 24$
Age (y)	28.6 ± 4.6	30.2 ± 5.1
Nulliparity	42 (66.6)	14 (58.3)
Smoking	0 (0)	0 (0)
Gestational age at delivery	39.2 ± 1.3	38.8 ± 1.7
Pregestational BMI (kg/m^2)	27.7 ± 2.3	29.1 ± 3.1

Abbreviation BMI, body mass index.
Note: Data are presented as mean ± standard deviation or *n* (%).

Statistical Analysis

Data analysis was performed with the SPSS v19.0 package (Chicago, IL). Student's *t*-test and Mann–Whitney's *U* test were used to compare continuous variables between the groups with and without normal distribution, respectively. The chi-square and Fisher's exact tests were used for categorical variables. Differences were considered significant when *p*-value was less than 0.05.

Results

During the study period, 87 women met the inclusion criteria and participated in the trial; 63 in the vaginal delivery group (group A) and 24 in the cesarean delivery group (group B). There was no crossover between the groups during labor. No significant differences were found in demographic or pregnancy characteristics in both patient groups (►**Table 1**). Of note, the mean age of the patients in both groups was 29.5 ± 5.2 years and the mean pre-pregnancy BMI was 28.2 ± 1.9. The percentage of primiparous and multiparous women was equivocal in both groups.

The Hemodynamics of Normal Vaginal Delivery

►**Fig. 1** describes the changes in CI measured at different points in time as described earlier. During the latent phase of the first stage of labor, epidural anesthesia resulted in a significant decrease in CI and a significant reduction in MAP, in comparison to the elevated values characteristic of labor before epidural. As labor progresses and the patient enters the active phase of labor, the CI rises significantly, peaking during the second stage to a mean value of 4.6 ± 0.8 L/min/m^2. One hour after the third stage of labor CI decreases significantly and continues its decline, reaching a nadir of a mean of 3.7 ± 0.8 L/min/m^2 48 hours postdelivery.

►**Fig. 2** demonstrates the hemodynamic changes in MAP and CI during labor, delivery, and postpartum in group A. After receiving epidural anesthesia during the latent phase, the MAP and CI significantly decrease, rising again and reaching maximal values during the active phase (CI = 4.4 ± 0.9 L/min/m^2 and MAP = 87.7 ± 10.3 mm Hg) and second stage of labor (CI = 4.6 ± 0.8 L/min/m^2 and MAP = 86.7 ± 9.8 mm Hg), similar to those reached in the latent pre-epidural phase. Within 1 hour postpartum, CI begins to decline while the MAP, that decreased significantly during the first 24 hours postpartum, begins to gradually rise 24 to 48 hours postpartum.

The Hemodynamic of Women Undergoing Elective Cesarean Delivery

In contrast to normal vaginal delivery, characterized by a reduction in MAP and CI after epidural anesthesia, parturients undergoing elective cesarean sections exhibit a rise in CI and MAP after spinal anesthesia (►**Fig. 3**). Their CI immediately prior to operation is nearly as high as the peak reached after spinal anesthesia (CI = 4.3 ± 1.00 L/min/m^2 and MAP 95.8 ± 13.4 mm Hg). At the time of fetal expulsion, CI and MAP decrease significantly, with CI reaching its nadir within 1 hour postpartum (CI = 3.07 ± 0.65 L/min/m^2). In contrast to normal vaginal delivery, after cesarean section, CI rises gradually from its nadir at 1 hour postpartum for the

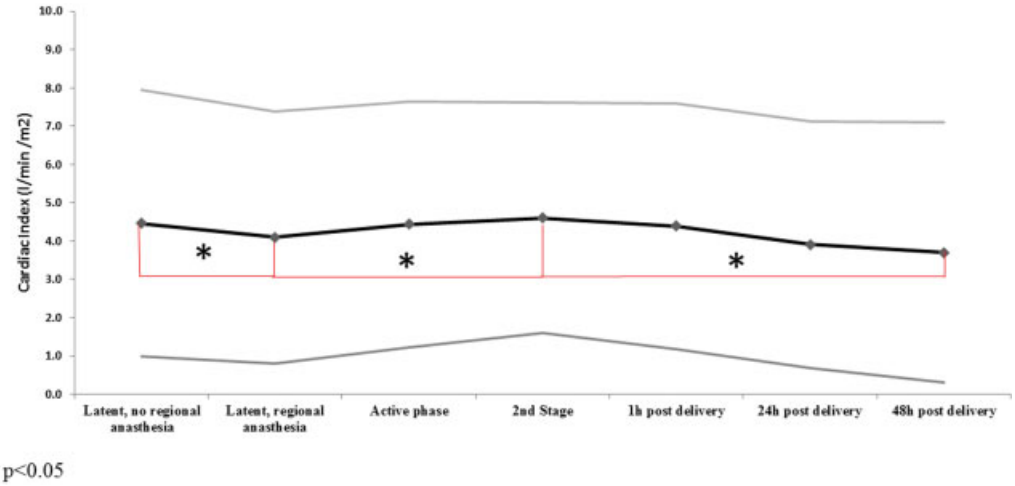

Fig. 1 Cardiac index and during labor, delivery, and early postpartum in normal vaginal delivery. Mean cardiac index and 95% confidence interval during the various points in time of vaginal delivery—latent prior to regional anesthesia, latent with regional anesthesia, during the active phase, during the second stage of labor, 1, 24, and 48 hours postpartum. Mean and standard deviations are represented by the bold and gray lines, respectively.

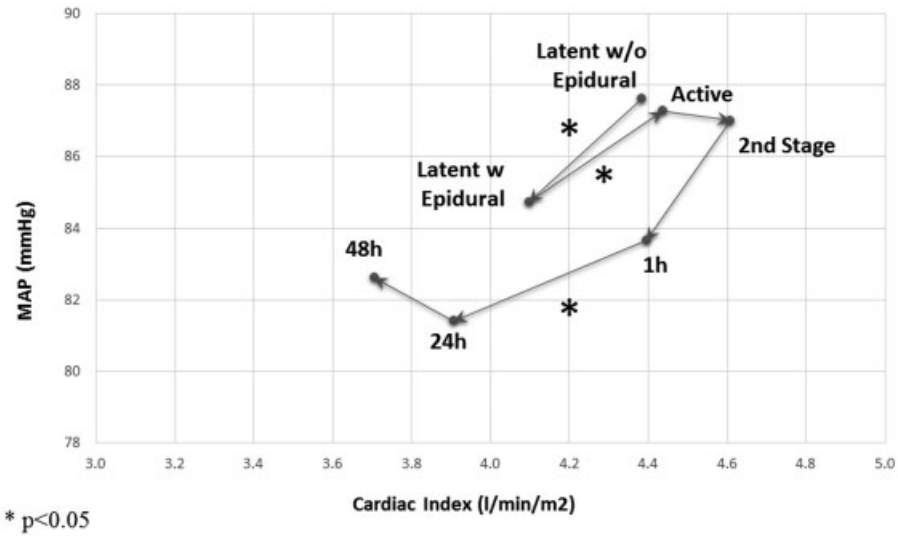

Fig. 2 The hemodynamics of normal vaginal delivery with epidural anesthesia. Mean cardiac index and MAP during the various points in time of vaginal delivery—latent prior to epidural anesthesia, latent with epidural anesthesia, during the active phase, during the second stage of labor, 1, 24, and 48 hours postpartum. MAP, mean arterial pressure.

Fig. 3 The hemodynamics of cesarean delivery with spinal anesthesia. Mean cardiac index and mean arterial pressure (MAP) during the various points in time of cesarean delivery—preoperative, at spinal anesthesia, at delivery, 1, 24, and 48 hours postpartum in elective cesarean delivery with spinal anesthesia.

following 48 hours, with little to no change in MAP (MAP 79.6 ± 9.3 mm Hg) (►**Fig. 3**).

SI changes very little during a normal vaginal delivery, is unaffected by epidural anesthesia, and peaks 1 hour postpartum (56.6 ± 9.0 mL/m^2; ►**Fig. 4A1**), as TPRI reaches its nadir (1.603.1 ± 431.6 dyne × s/cm^5 × m^2; ►**Fig. 4A2**). After this time, SI gradually declines accompanied by the rising TPRI.

In contrast to normal vaginal deliveries, during an elective cesarean section at the time of spinal anesthesia, SI declines (46.5 ± 9.2 mL/m^2; ►**Fig. 4B1**), with little change in TPRI, reaching its nadir 1 hour postpartum (46.4 ± 8.8 mL/m^2; ►**Fig. 4B2**) in parallel with the maximal TPRI achieved 1 hour postpartum (1,275.9 ± 356.0 dyne × s/cm^5 × m^2). From then on and until 48 hours postpartum, SI remains quite constant and TPRI decreases (47.4 ± 7.2 mL/m^2 and 1,043.4 ± 327.6 dyne × s/cm^5 × m^2), both reaching similar values to those measured preoperatively.

Of the 24 women in group B, two women did not exhibit the expected increase in TPRI 1 hour postpartum, but instead, demonstrated a decrease in this value within the first hour after delivery. Both women suffered from PPH. The first patient, exhibiting a 25% reduction in TPRI 1 hour postpartum, had an uneventful cesarean section, but exhibited profuse vaginal bleeding after delivery, with a drop in hemoglobin values from 12.9 g/dL preoperatively to 9.8 g/dL during the first 6 hours postoperatively. The second patient, with a 13% reduction in TPRI 1 hour postpartum, bled significantly during the operation due to adherent placenta. Tension sutures were placed on the placental bed and

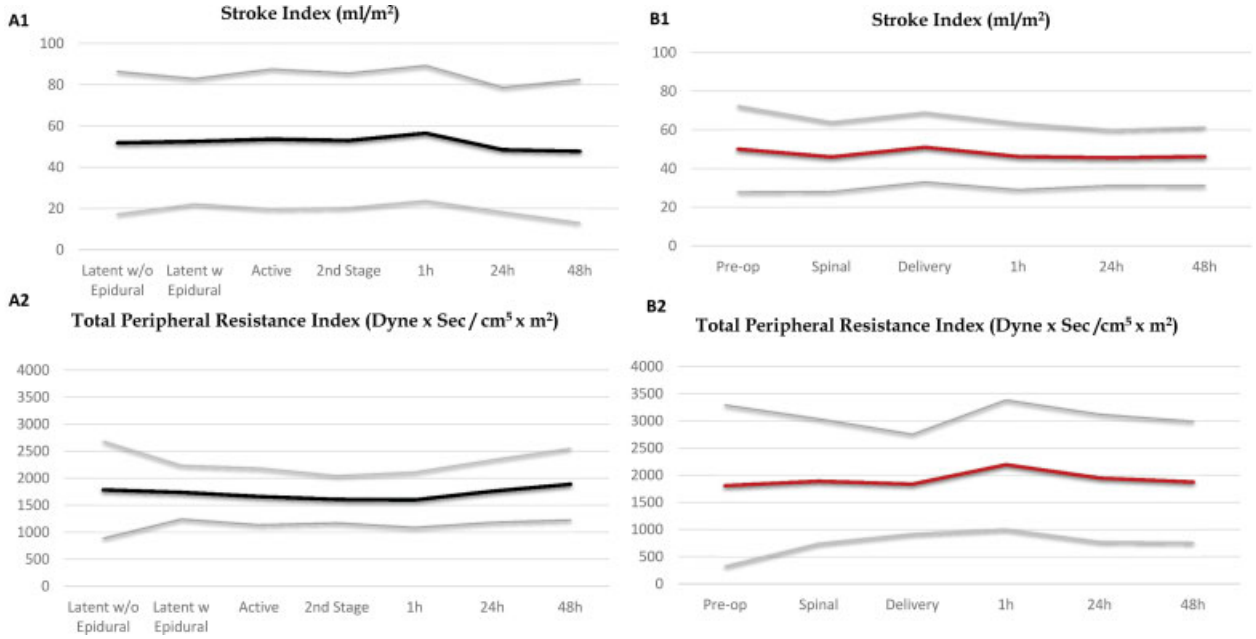

Fig. 4 Stroke index and total peripheral resistance index in normal (**A**) vaginal and (**B**) cesarean deliveries. Mean stroke index (A1) and mean total peripheral resistance index (A2) during the various points in time during vaginal delivery—latent prior to epidural anesthesia, latent with epidural anesthesia, during the active phase, during the second stage of labor, 1, 24, and 48 hours postpartum. Mean stroke index (B1) and mean total peripheral resistance index (B2) during the various points in time of elective cesarean delivery—preoperative, at spinal anesthesia, at delivery, 1, 24, and 48 hours postpartum. Mean and standard deviations are represented by the bold and gray lines, respectively.

prostaglandin F2a was administered intramuscularly. Hemoglobin in this patient dropped from 13.2 g/dL preoperatively to 10.1 g/dL postoperatively.

Discussion

Normal labor and delivery are associated with significant hemodynamic adaptations due to maternal anxiety, exertion, pain, uterine contractions, and blood loss. Previous hemodynamic studies, based on invasive and noninvasive devices, have reached nonuniform results regarding the magnitude and timing of these adaptations. This study elucidates the physiology of a brief period of time—from vaginal labor or cesarean delivery until 48 hours postpartum, utilizing the whole body bioimpedance based device. The purpose of this study was not to validate this device in the setting of parturients undergoing vaginal or cesarean deliveries, but to demonstrate the hemodynamic trends associated with delivery as determined by whole body bioimpedance.

As shown in previous studies assessing vaginal delivery,[5] our findings show that CI rises from the latent phase until the second stage of labor, reaching its peak during this time. Within 1 hour after delivery, CI declines significantly, reaching its nadir within 24 hours postpartum, all along accompanied by a gradual decline in MAP. The finding of a marked elevation in CO during the second stage has been demonstrated previously,[4] with maximal values reached in the first 10 minutes postpartum. This dramatic elevation is brief and by 1 hour postpartum, as exhibited in our study, CO declines to values approaching those of a prelabor state.[5]

Epidural anesthesia during the latent phase reduced CI and MAP, concomitant with previous findings,[8] but its effects were counterbalanced by the exertion and strain of the second stage of labor, resulting in a peaking CI and MAP. This finding emphasizes that epidural anesthesia reduces, but does not eliminate, the rise in CI.[7,30]

The second stage of a vaginal delivery is characterized by a marked increase in CI, despite a slight decrease in SI. The latter can be attributed to the straining phase and resultant Valsalva's maneuver, which decreases preload. Therefore, the elevation in CI demonstrated in our study is most likely a result of the increasing heart rate and blood pressure[31] and the decreasing TPRI, characteristic of a normal vaginal delivery. Patients exhibiting a lack of CI elevation during labor and delivery may have significantly decreased preload or SI and cardiac failure. In these cases of nonelevated CI during labor, it would be important to further study this device in this population.

The hemodynamics of cesarean deliveries differs from that of vaginal deliveries. In our study, TPRI was not significantly influenced by spinal anesthesia. While this finding is refuted by previous study,[8] it is supported by a recent study in a similar population of parturients.[27] We hypothesize that the lack of decrease in TPRI after regional anesthesia may be attributed to the fact that the preoperative measurements were taken prior to entry into the operating room. At this point, the patient was at resting state, with a mean heart rate of 85.2 $\pm$ 13.9 bpm. This is in comparison to the heightened stress reaction in the operating room, at which point a heart rate of 95.0 $\pm$ 26.4 bpm was measured (12% increase). In addition, the administration of a bolus of fluid was only

initiated in the operating room, in accordance with our departmental protocol, further raising blood pressure from 89.5 ± 13.1 to 95.8 ± 13.4 mm Hg (7% increase). As demonstrated in previous studies, CI remained constant during spinal anesthesia in our study,[4] enabling women to maintain hemodynamic stability during this time.

Contrary to past studies that showed an increase in CO after vaginal or cesarean delivery (most probably due to autotransfusion of uterine blood into the maternal systemic circulation and the administration of oxytocin),[7,27] removal of the fetus during elective cesarean sections in our study caused a marked decrease in CI, due to an increase in TPRI and a resultant decline in preload and SI.

The immediate postpartum period in vaginal deliveries and cesarean sections differs as well. While TPRI continues to decline 1 hour postpartum in normal vaginal delivery, it rises dramatically within 1 hour after cesarean delivery. Studies of resistance indices of the uterine arteries have shown that the time needed for vascular physiology to revert from a pregnant to a nonpregnant state is longer than assumed.[32] This is probably owing to a prolonged recovery and return to baseline characteristic of vaginal deliveries. During the straining phase, there is greater pelvic and uterine vessel vasodilation, an adaptation necessary for the increased workload of the muscles participating in a vaginal delivery. Therefore, in comparison to elective cesarean deliveries, longer time is needed to resume normal function. Anecdotally, the only women in our study who did not exhibit the expected increase in TPRI immediately postoperatively were those who suffered from PPH. In these patients, TPRI decreased within 1 hour postpartum. This might suggest that in parturients who demonstrate an unexpected decrease in TPRI immediately after cesarean delivery, the physician should be more alert to possible hemorrhage and prepare accordingly.

The main strength of our study is the assessment of hemodynamic changes during and after labor and delivery by a method of bioimpedance, well established in other medical settings, but utilized in parturients undergoing vaginal delivery for the first time. Its relatively large sample size of women undergoing vaginal delivery and the use of CI, as opposed to CO, a parameter that is adjusted to the patient's BMI are additional strengths. Our study is limited by a small sample size of women undergoing elective cesarean sections. Moreover, we did not perform hemodynamic studies prior to labor and this prevented us from comparing the hemodynamics of the shift from prelabor to labor. In addition, our study focused on healthy women with a low-risk pregnancy. This prevents extrapolation of the results to high-risk patients suffering of conditions associated with pathological hemodynamic alterations, such as heart disease or preeclampsia. Finally, further large studies are needed to better define the cardiovascular physiology during labor and delivery.

In conclusion, the results of this trial suggest that the whole body bioimpedance technique, as it is noninvasive, accessible and nonoperator-dependent device, can be used to assess hemodynamic changes in low-risk healthy women, during the crucial periods of labor, delivery, and 48 hours postpartum. Future studies in low- as well as high-risk populations are recommended before adopting this device to manage labor in general, and particularly in high-risk women with cardiovascular disease or preeclampsia.

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Conflict of Interest
S.L. is a consultant for NI Medical, and all other authors report no potential conflict of interest.

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