will decrease further below the threshold and result in unpleasant symptoms.

Data from Langesæter *et al.*[1] suggest that liberal administration of phenylephrine to achieve tight blood pressure control may result in lower cardiac output than a less aggressive approach. However, the clinical relevance of this in healthy elective patients is undetermined. Dyer and James[2] stated that heart rate and blood pressure are used clinically as surrogate markers of maternal cardiac output, and that maximum changes in the latter correlate better with uteroplacental blood flow than with upper arm blood pressure. However, it has yet to be proven whether a global measure of cardiac output is the ideal parameter on which to base hemodynamic therapy in obstetric patients, since this does not necessarily represent regional flow in the uterus and placenta, which have widely dilated vasculature through which flow is largely pressure-dependent.[8] Furthermore, studies in animals that have correlated fetal oxygen uptake with uterine blood flow have demonstrated that, under normal physiologic conditions, uterine blood flow is in excess of the minimum required to satisfy fetal oxygen demand.[9] This confers a margin of safety that, to a degree, protects the fetus from fluctuations in uterine blood flow.[10] If the same situation occurs in humans it could explain the lack of fetal acidosis observed when large doses of phenylephrine are used to maintain maternal blood pressure,[3–5] despite evidence that this drug has the potential to decrease both cardiac output and uteroplacental blood flow. The implication of this is that in healthy parturients, within limits, even if hemodynamic control using phenylephrine does slightly reduce cardiac output and uteroplacental blood flow, it may still be clinically acceptable if this is balanced against other benefits such as maternal wellbeing.

Langesæter *et al.*[1] concluded that that low-dose bupivacaine (with sufentanil), combined with a low-dose infusion of phenylephrine and moderate cohydration, gives the "best" hemodynamic stability. However, they can only validly make comparisons among the actual regimens included in their study. Because there were no direct comparisons with patients who received higher doses of phenylephrine or larger volumes of cohydration, it is speculative to assume that any of their regimens were superior to these other possible regimens. Moreover, a large proportion of their patients who received a low-dose phenylephrine infusion required supplementary boluses of vasopressors. Thus the mean phenylephrine requirement per patient was somewhat greater than the baseline 0.25 $\mu g \cdot kg^{-1} \cdot min^{-1}$ and it cannot be excluded that a higher infusion rate may have provided more stable hemodynamic control. Also, when assessing the optimal regimen for hemodynamic control, we believe that ease of use is also a factor to be considered. In that respect, we consider a vasopressor infusion that is titrated to a defined endpoint to be superior to the use of a hybrid technique of a fixed rate low-dose infusion to which is added repeated "rescue" boluses for times when the infusion is inadequate. This is especially relevant during single-shot spinal anesthesia when small intrathecal doses as used by Langesæter *et al.*[1] may not be appropriate.

We continue to advocate aggressive maintenance of blood pressure using phenylephrine in women having elective cesarean delivery under spinal anesthesia. Although it may be possible that cardiac output is slightly less with this method as compared with allowing blood pressure to drift 10 to 20% below baseline, unless evidence is produced that this is associated with a clinically important decrease in uteroplacental blood flow *and* with a detrimental effect on neonatal outcome, we believe that improved maternal comfort justifies our technique.

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Maternal Hemodynamic Monitoring and the Vigileo Monitor

To the Editor:—We read the recent editorial by Dyer and James,[1] referencing the paper by Langesæter *et al.*[2] with great interest. Langesæter *et al.*[2] have successfully demonstrated that a minimally invasive technology that measures cardiac output (LiDCO*plus*, LiDCO Ltd., Cambridge, United Kingdom) can be used for maternal hemodynamic monitoring. Other arterial pressure waveform–based systems that provide beat-by-beat assessment of cardiac output and stroke volume include the PiCCO*plus* (Pulsion Medical Systems, Munich, Germany) and the Vigileo monitor/FloTrac sensor (Edwards Lifesciences LLC, Irvine, CA). Dyer and James[1] note in their editorial that these less invasive methods of hemodynamic monitoring are attractive to the obstetric anesthesiologist. However, the editorial[1] view that the Vigileo monitor is unsuitable for the study of rapid maternal hemodynamic changes is not a logical conclusion.

Bland-Altman analysis is routinely used to assess precision and bias when comparing two different measurement techniques.[3–9] As acknowledged in the editorial, the acceptance of a new technique of cardiac output measurement should rely on limits of agreement of up to ± 30% between the minimally invasive techniques and the existing "gold-standard" (*i.e.,* the thermodilution pulmonary artery catheter).[5] There are currently several studies in the critical care and cardiac anesthesia literature that have evaluated precision and bias (using Bland-Altman analyses) in the measurements of cardiac output with the Vigileo monitor/FloTrac sensor.[3,4,7–10] Based on these studies, statisti-

cally and clinically acceptable precision and bias has been shown to exist in the measurement of cardiac output by the Vigileo monitor. The novel arterial pressure cardiac output algorithm used by the Vigileo monitor provides cardiac output assessments that agree satisfactorily for clinical purposes with intermittent and continuous thermodilution techniques in postoperative cardiac surgical patients[4,7–9] and in the critically ill population.[3,10] Fluid and pharmacologic therapy algorithms based on cardiac output and stroke volume variation measurements from the Vigileo monitor are now being studied (with the hope of improving outcome) in patients who are critically ill and/or undergoing major surgery.[3] Further, such studies may be needed in the noncritically ill spontaneously breathing patient populations, including pregnant women undergoing cesarean sections.

We will now examine the specific situation of the measurement of cardiac output after the administration of phenylephrine during cardiac surgery (based on which the editorial concludes that the Vigileo monitor "may not be suitable").[9] The basic physiologic principle underpinning the Vigileo device is that left ventricular stroke volume and arterial pulsatility are proportional to each other, and the proportionality constant, κ, is a number that describes the resistance and compliance of the arterial tree: Stroke volume $\approx$ pulsatility x κ.[6] Pulsatility is calculated by using the SD of the arterial pressure waveform over a 20-s period analyzed at a 100-Hz frequency. The κ value is calculated every minute by the latest operating system and is based on patient weight, height, age, mean arterial pressure, skewness, and kurtosis of the pressure wave.[11] It has been implied by Lorsomradee *et al.*[9] that immediately after a phenylephrine bolus (or after sternotomy), the Vigileo/FloTrac system overestimated the cardiac output relative to the continuous cardiac output measurement by the thermodilution pulmonary artery catheter. Phenylephrine (especially as a bolus dose) will abruptly affect the pulsatility of the arterial system. This acute arteriolar constriction will cause the left ventricular stroke volume to be ejected into a more "pressurized" central arterial tree, simulating an increase in pulsatility. The measurements obtained by the Vigileo monitor will initially interpret this increase in pulsatility as an increase in *left-sided* stroke volume and cardiac output. The *right-sided* cardiac output being measured by the thermodilution pulmonary artery catheter will not reflect this sudden change. The current software version on the Vigileo system, however, recalibrates itself constantly, and over the next 1 to 2 min the FloTrac sensor will "relearn" this new increased vascular tone, recalculate κ, and then report updated stroke volume and cardiac output values.[11] The converse may be expected acutely when a vasodilator (such as nitroprusside) is administered. The vascular tone and therefore pulsatility will acutely decrease, and the initial Vigileo cardiac output readings will underestimate thermodilution measurements. Understanding the mechanisms of cardiac output measurement by the Vigileo monitor allows us to anticipate such measurement errors. Every device has its limitations. A bolus of thermally active injectate (*e.g.,* red blood cells/fluids being administered rapidly) will produce errors in the measurement of cardiac output by the thermodilution pulmonary artery catheter. Similarly, acute changes in arterial pulsatility will produce errors in the arterial waveform based measurements systems. Since we do not need to externally calibrate the Vigileo system for measurement (the system regularly autocalibrates), we are essentially exchanging ease of use for clinically acceptable precision and bias. Waiting a few minutes for autorecalibration and the recalculation of κ is appropriate before relying on Vigileo-reported measurements.

Dyer and James[1] also state that central venous pressures and pulmonary capillary wedge pressures are unlikely to predict the response to fluid administration, and that pulse pressure variation and stroke volume variation (SVV) may be better indicators of fluid responsiveness. Systolic blood pressure variation, pulse pressure variation, and SVV are all clinically reliable measures of fluid responsiveness in nonobstetric populations.[12–14] However, these cardiorespiratory interactions have mostly been studied in mechanically ventilated, critically ill patients. Robust data in spontaneously breathing subjects are not widely available. Pulse pressure variation or SVV changes may be harder to interpret during spontaneous ventilation, given the inherent variations in respiration. The LiDCO*plus* system displays a continuous measure of SVV, and it would be interesting to know if this data were acquired by Langesæter *et al.* Since subjects in this study[2] adhered to a relatively strict fluid administration protocol (with no prehydration and moderate cohydration) and a standardized vasopressor administration protocol, we could gain useful information from any SVV data that might be available.

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