intraoperative transoesophageal echocardiography as well as time spent in the echocardiography lab. This model is not as well developed here in Australia or New Zealand, with only a handful of such positions that are oversubscribed. Many cardiology-based echo labs are extremely reluctant to allow anaesthesia providers access to even observe, let alone acquire images in their departments. The fellowship model allows this in a teaching and supervised role, with an emphasis on practical and clinically relevant echocardiography, including image acquisition. Despite these challenges, I would encourage the Australian and New Zealand College of Anaesthetists and the Australian Society of Anaesthetists to take an active role in supporting hospital-based training in perioperative echocardiography for the non-cardiac anaesthetist.

Our preliminary data has shown that the most common reason for a focused cardiovascular ultrasound in our centre is the undifferentiated systolic murmur[2]. This accounts for around 50% of our requested studies. Hence, estimating severity of aortic stenosis is an important part of routine anaesthesia practice.

In my view, obtaining a velocity and gradient across the aortic valve is no more or less intuitive than any other aspect of echocardiography, such as estimation of ejection fraction or evaluation of haemodynamic state, which Drs Faris, Veltman and Royse emphasise in their H.A.R.T.scan® courses. This should be combined with qualitative views of the aortic valve from parasternal views looking for calcified, immobile and restricted leaflets as is emphasised in the review. The limitations of estimating aortic velocity and gradients in situations of low flow are also mentioned in the review. The classification of aortic stenosis severity involves aortic jet velocity, gradient and valve area, all of which are well validated[3]. For most perioperative circumstances in a limited TTE, aortic jet velocity and gradient, in conjunction with qualitative assessment, is reasonable, feasible and rapidly obtainable.

As is emphasised in the review, a focused TTE does not replace a formal cardiology-based TTE. When image quality is suboptimal, when pathology is complex or confusing, when patients require additional follow-up or there is inadequate expertise to obtain and interpret images, referral for a formal TTE needs to be considered.

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Cardiac output measurement, by means of electrical velocimetry, may be able to determine optimum maternal position during gestation, labour and caesarean delivery, by preventing vena caval compression and maximising cardiac output and placental perfusion pressure

The avoidance of aortocaval compression (ACC) has long been a key goal for the obstetrician and obstetric anaesthesiologist. Compression of the abdominal aorta by the pregnant uterus reduces uterine artery pressure below brachial artery pressure and compression of the vena cava not only reduces venous return – thereby reducing cardiac output – but also increases uterine venous pressure. Hence, ACC represents a triple threat to placental perfusion and foetal well-being: brachial artery pressure may decrease due to decreased cardiac output, uterine artery pressure may decrease below brachial artery pressure and uterine venous pressure may increase. ACC is even more dangerous in the setting of labour and anaesthesia: uterine contractions periodically deprive the placenta of maternal blood flow and neuraxial anaesthesia tends to reduce maternal brachial blood pressure further. These facts are well known, but exactly how well do we prevent ACC in practice? Is there a way of determining how much left (or right) uterine displacement is enough[1]?

For decades foetal heart rate (FHR) has been used as an indicator of foetal well-being, but it is a rather 'blunt instrument' for detecting maternal haemodynamic conditions which threaten the foetus. By the time late FHR decelerations appear, the foetus is already hypoxaemic. Might there be a way of optimising maternal haemodynamics without waiting for foetal hypoxia?

I believe that measurement of cardiac output (CO) during labour by means of electrical velocimetry (EV) may represent such a methodology. EV is a new type of impedance cardiography which is easy to apply and non-invasive. It has been shown in non-obstetric studies to correlate well with better established methods of measuring CO, such as thermodilution and echocardiography[2,3]. In a 30 patient series, EV was used without a 'gold standard' to measure the haemodynamic changes associated

with caesarean delivery and oxytocin administration[4]. In anecdotal reports without 'gold standard' verification, EV demonstrated haemodynamic changes associated with the use of intravenous magnesium sulphate, hydralazine and labetalol in pre-eclampsia[5], indigo carmine during bladder repair after caesarean delivery[6] and an episode of FHR deceleration coincident with magnesium administration[5].

When maternal CO is maximised, venous return is also maximal, since 'venous return' rapidly becomes 'cardiac output'. Venous return is maximal when the vena cava is unobstructed and an unobstructed vena cava is presumably associated with minimised uterine venous pressure. Hence, the maternal position in which CO is maximal should also maximise placental perfusion pressure. If we could easily measure and maximise CO during gestation, labour and caesarean section, the parturient could be positioned in a way that maximises placental perfusion.

Figure 1 (patient 1) demonstrates the effect of maternal position on cardiac output in an obese woman, not in labour, resting comfortably in bed, at 30 weeks gestational age. This patient's foetus had intrauterine growth restriction and absent end-diastolic flow in the umbilical artery. Figure 1 shows that any tilt toward the right side was associated with decreased cardiac index. A left tilt of 45 degrees was superior to any position with the right side down, but for maximum cardiac index the full left lateral decubitus (LLD) position was required. Blood pressure was not measured but position changes caused no maternal symptoms or FHR decelerations but reproducible changes in SI and CI, as estimated by electrical velocimetry. Chronic placental venous congestion might, in some cases, contribute to foetal growth restriction or placental abruption and it is conceivable that optimal maternal positioning during labour (based on EV or other methods of CO assessment) might reduce the incidence of placental abruption.

Figure 2 (patient 2) demonstrates the haemodynamic course of a patient with severe pre-eclampsia who received epidural analgesia for contraction pain. Both cardiac index and mean arterial pressure declined while the patient was seated for epidural placement. After epidural dosing she was positioned in the full right lateral decubitus position for taping of the catheter and then allowed to rest tilted 30 degrees to the right. Hypotension associated with low cardiac index ensued and was treated with intravenous fluid, two boluses of phenylephrine 50 μg and one bolus of ephedrine 10 mg. After placement in the full LLD position (L90, see Figure), cardiac and stroke indices increased, hypotension resolved and vasopressors were no longer required. There were no FHR decelerations during the time period shown.

Figure 3 (patient 2) provides a more detailed lookat the haemodynamic pattern after patient 2 was placed in the LLD position. The brisk increases in stroke volume and cardiac index occurring approximately every three minutes coincided with

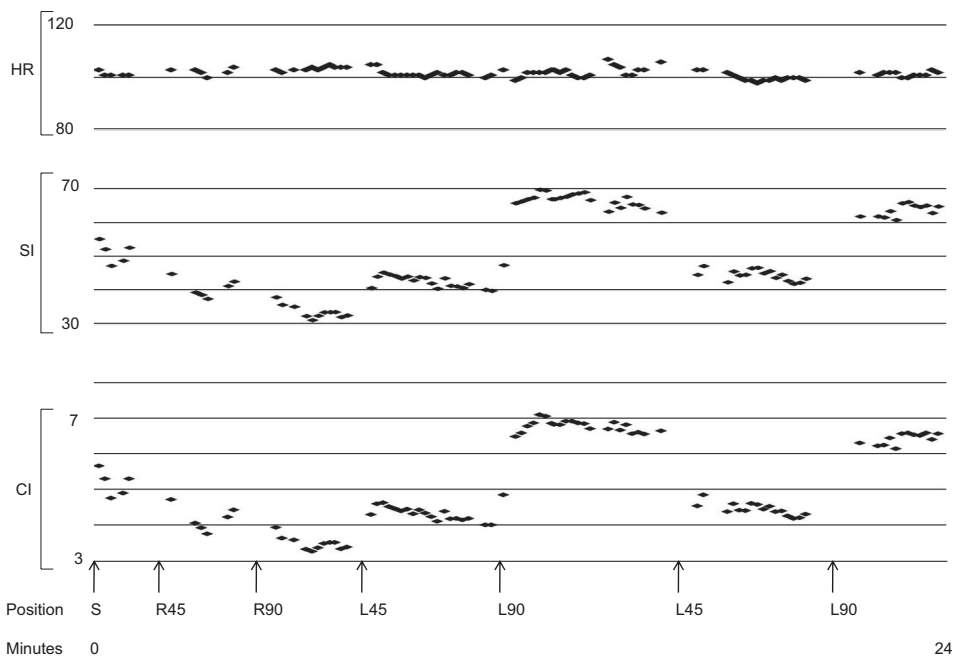


FIGURE 1: Patient 1. HR=heart rate, beats per minute; SI=stroke index, ml.m-2; CI=cardiac index, l.min^{-1}.m^{-2}; S=supine position; R=right side down; L=left side down; 45=45 degrees tilt; 90=90 degrees tilt (full lateral decubitus position).

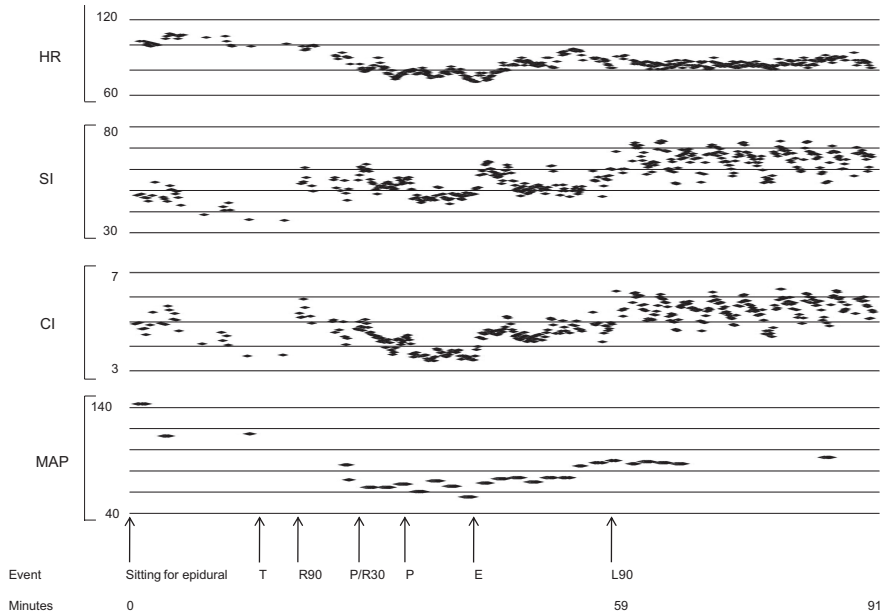


FIGURE 2: Patient 2. Before and after epidural placement in the sitting position. Test dose was 3 ml lignocaine 1.5% with adrenaline 1:200,000 then fentanyl 100 μg, 2 ml lignocaine 1.5% with adrenaline and 3 ml plain lignocaine 1.0%. Note damped excursions of cardiac index with uterine contractions before L90. HR=heart rate, beats per minute; SI=stroke index, ml.m^{-2}; CI=cardiac index, l.min^{-1}.m^{-2}; MAP=mean arterial pressure, mmHg; R=right side down; L=left side down; 30=30 degrees tilt; 90=90 degrees tilt (full lateral decubitus position); T=test dose; P=phenylephrine 50 μg; E=ephedrine 10 mg.

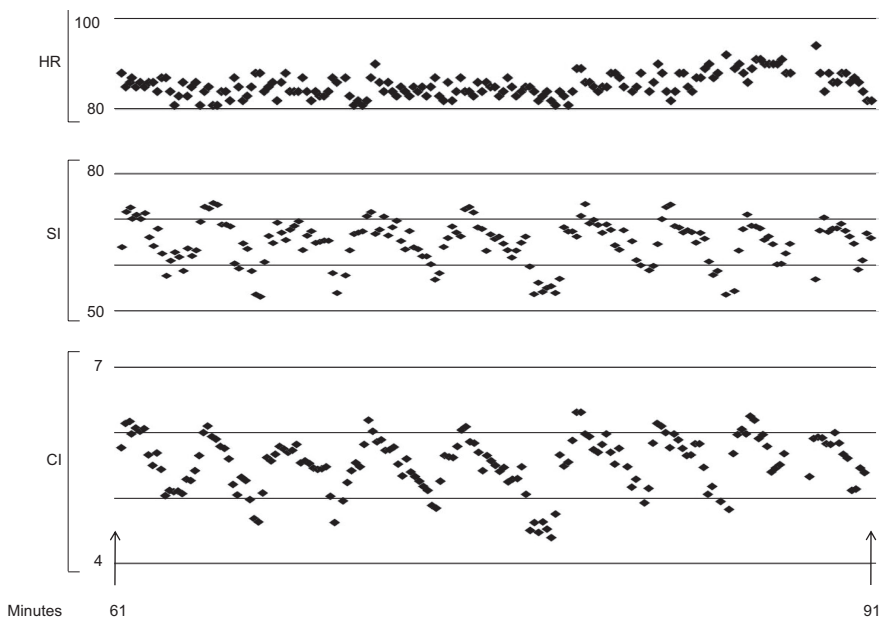


FIGURE 3: Patient 2. Thirty minute detail of increases in cardiac and stroke indices, coincident with uterine contractions that occur approximately every three minutes. Note the amplitude of periodic CI excursions before and after adoption of LLD at minute 59. HR=heart rate, beats per minute; SI=stroke index, ml.m^{-2}; CI=cardiac index, l.min^{-1}.m^{-2}.

the uterine contractions, as seen on the external tocodynamometer. The increases in cardiac index are probably due to autotransfusion in the setting of a vena cava that was presumably unobstructed in that position. The patient was pain free by this time so the haemodynamic changes were presumably to the autotransfusion.

Of note, maximised placental perfusion does not guarantee maximal oxygen delivery to the foetus because certain maternal positions can cause umbilical cord compression. This would negate any value of augmented placental perfusion, such that maximisation of CO would not replace FHR monitoring, but rather constitute an 'early warning

system' to objectively identify maternal favourable positions for placental perfusion, other things (such as the umbilical cord) being equal.

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Awake intubation using Pentax AWS video-laryngoscope after failed fibreoptic intubation in a morbidly obese patient with a massive thyroid tumour and tracheal compression

A morbidly obese 53-year-old male, weighing 134 kg with a body mass index of 45.3, was scheduled for total thyroidectomy. He presented with a massive multi-nodular goitre with pressure symptoms which included difficulty breathing and hoarseness of voice. His past medical history included severe obstructive sleep apnoea on home continuous positive airway pressure therapy, ischemic heart disease, chronic obstructive airway disease, type II diabetes mellitus and hypertension. Computed tomography of his neck demonstrated a gross enlargement of the thyroid gland with the right and left lobes measuring 11×6×5 cm and 10×6×7 cm respectively. This was associated with displacement of the trachea to the right and narrowing of the airway to a minimum transverse diameter of 7.7 mm. (Figure 1) Preoperative nasal endoscopy showed that the upper airway was very narrow, roughly 2 mm at the glottis.

Difficult intubation was acknowledged and anticipated. Multiple attempts of awake intubation with 11 Fr fibreoptic bronchoscope failed both orally and nasally by two experienced specialist anaesthetists. The surgery was cancelled for that day and the patient was re-scheduled two days later. This time an awake intubation by Pentax AWS-S100 video-laryngoscope (Pentax Corporation, Tokyo, Japan) was planned. Our alternative plan if this failed was to induce the patient with intravenous propofol and perform direct laryngoscopy, with ENT surgeons and rigid bronchoscope readily available.

After adequate topicalisation, the patient's anxiety and gag reflex were further reduced by a low dose remifentanil target-controlled infusion up to 1 ng/ml. Visualising the glottis was possible with a Cormack Lehane glottic view grade 2 using the Pentax AWS video-laryngoscope, however the glottis appeared very narrow with significant surrounding oedema and mucosal hypertrophy. An initial attempt to pass the armoured endotracheal tube failed due to the narrow glottis. Subsequently, a Frova (Cook Medical, Bjaeverskov, Denmark) airway intubation catheter was inserted under video assistance and the tube was successfully advanced into the trachea. Anaesthesia was safely induced intravenously after the airway was secured.

Massive thyroid tumours are a potential cause of difficult airway. In our case the location and size of the tumour and co-existing morbid obesity and obstructive sleep apnoea posed a high risk of complete upper airway obstruction, if anaesthesia was induced and muscle relaxation administered before intubation. If a 'can't ventilate, can't intubate' situation had occurred, it was judged that it would be impossible to perform an emergency surgical



FIGURE 1: Computed tomography topogram showing tracheal deviation with narrowing in the neck region.