electrocardiography (ECG), 24-hour ambulatory ECG, transthoracic echocardiogram and stress ECG were normal. Since his previous procedure he had remained normotensive without medications and had been asymptomatic apart from his hernia.

He was 182 cm tall and weighed 90 kg. All preoperative investigations were normal and the patient was well on the day of surgery. Pre-induction vital signs were normal. Anaesthesia was induced with fentanyl 100 μg intravenously in two divided doses and propofol 200 mg intravenously. A laryngeal mask airway was inserted easily. Post-induction his vital signs remained stable. Anaesthesia was maintained with 2% sevoflurane a mixture of air and oxygen. About ten minutes after induction, midway during the cleaning of the surgical site with cetrimide solution, his heart rate decreased to 22 /minute. The surgeon was asked to stop cleaning and intravenous atropine 0.6 mg was administered immediately, which increased his heart rate to 84 /minute. Surgical preparation then resumed and the operation, emergence and postoperative recovery proceeded uneventfully.

The cause of perioperative bradycardia is often multifactorial which may be both pharmacological and physiological. Opioids and inhalational agents such as sevoflurane have been associated with bradycardia. Fentanyl in particular has been associated with bradycardia. Mechanisms include central vagal effects likely via the cardioinhibitory parasympathetic vagal neurons in the nucleus ambiguous[1] and the effect on vagal nerve endings and cardiac opiate receptors[2]. Inhalational agents have been associated with cardiovascular depressant effects[3], although not severe. There are also case reports of bradycardia, especially in children[4], attributed to sevoflurane.

Bradycardia resulting from vagal stimulation secondary to peritoneal or pleural irritation, or the triggering of the oculocardiac or trigeminocardiac reflexes is also not uncommon intraoperatively. Although unusual, vagal stimulation to cold resulting in syncope[5] and even cardiac arrest after a cold drink[6] have been reported, but the exact mechanisms are unclear. The genital area is innervated by branches from the sacral plexus, which include parasympathetic nerve fibres. It is thus reasonable to postulate that with a background of higher sensitivity to parasympathetic stimulation, the coldness of the cetrimide cleaning solution may have stimulated the area resulting in the bradycardia.

Postoperative follow-up by the surgical and anaesthetic teams is essential. Our patient has been made aware of his susceptibility to profound bradycardia during anaesthesia. Written information regarding the above event likely related to the sensitivity to cold cetrimide was given to the patient. He has also been advised to discuss this with his anaesthetist and surgeon prior to any future surgery. This case demonstrates the need for continued high level vigilance and preparedness, even in otherwise well patients undergoing minor procedures.

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Maximisation of maternal cardiac output during labour might help to prevent not only foetal hypoxaemia but also myometrial ischaemia, dysfunctional labour, uterine atony and postpartum endometritis

We previously noted that positioning the parturient so as to maximise cardiac output has a high probability of maximising both placental perfusion and foetal oxygenation[1]. It has since occurred to us that the same logic applies to overall uterine perfusion and specifically to myometrial perfusion. This may have implications for optimising all aspects of uterine function during and after labour. Since uterine acidosis and hypoxia are associated with both dysfunctional labour[2] and postpartum uterine atony, it is possible that uterine venous decompression and good uterine perfusion could protect the foetus, improve uterine function and leave the uterus less susceptible to postpartum infection. It is also conceivable that maximising cardiac output by proper maternal positioning during gestation and labour could play a role in palliating pre-eclampsia, since poor placental perfusion is a factor in that condition.

This 'uterine ischaemia hypothesis' might also explain why obese patients have an increased risk of dysfunctional labour and postpartum uterine atony. Obese patients have increased intra-abdominal pressure which, in conjunction with the gravid uterus, may compress venous return. Hence, uterine perfusion and contractility may be particularly precarious in obese patients.

Women who have a uterus that is overdistended by polyhydramnios or multiple gestation have an increased risk of postpartum haemorrhage. How might this fit with our "uterine ischaemia hypothesis"? The answer lies in La Place's Law: uterine wall tension depends on both intrauterine pressure and uterine diameter. As with the myocardium, myometrial oxygen consumption probably depends on 1) frequency and duration of contraction and 2) wall tension. For a given intrauterine pressure, a large uterus is likely to consume more oxygen than a normal-sized uterus and thus be more susceptible to ischaemia and consequent dysfunction.

Our hypothesis is that maternal positioning during labour, to maximise cardiac output, might improve both foetal and maternal outcomes. We are initiating such research and intend to look at several outcomes, including caesarean section, dysfunctional labour, uterine atony and postpartum endometritis. Perhaps we are closer to answering Paech's question, "Should we take a different angle in managing pregnant women at delivery?"[3]. We speculate that in some cases the uterus – as well as the foetus – needs oxygen more than oxytocin.

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Kaolin activated thromboelastography can result in false positive fibrinolytic trace

Using whole blood, this method can yield information relating to the cumulative effects of various blood components (e.g. plasmatic factors, platelets) on all coagulation phases. As the generation of the thromboelastography (TEG) trace using calcium activated blood is slow (within citrated method), activators such as kaolin are increasingly used to reduce the time to trace generation. However, kaolin can initiate an intristic coagulation pathway and has a complex effect on platelets[1]. Therefore some differences in results between kaolin activated and non-kaolin activated samples may be possible. Thalheimer and his colleagues found differences in many TEG parameters in healthy volunteers and also in patients[2]. When performing TEG, we often find a pattern of secondary fibrinolyis, which does not correlate with the clinical status of the patient nor with laboratory haematological parameters. We performed kaolin activated and non-kaolin activated TEG (using citrated method) in patient with cereberal bleeding caused by tumour eroding blood vessels (Haemoscope Corp., Niles, Illinois, USA). In

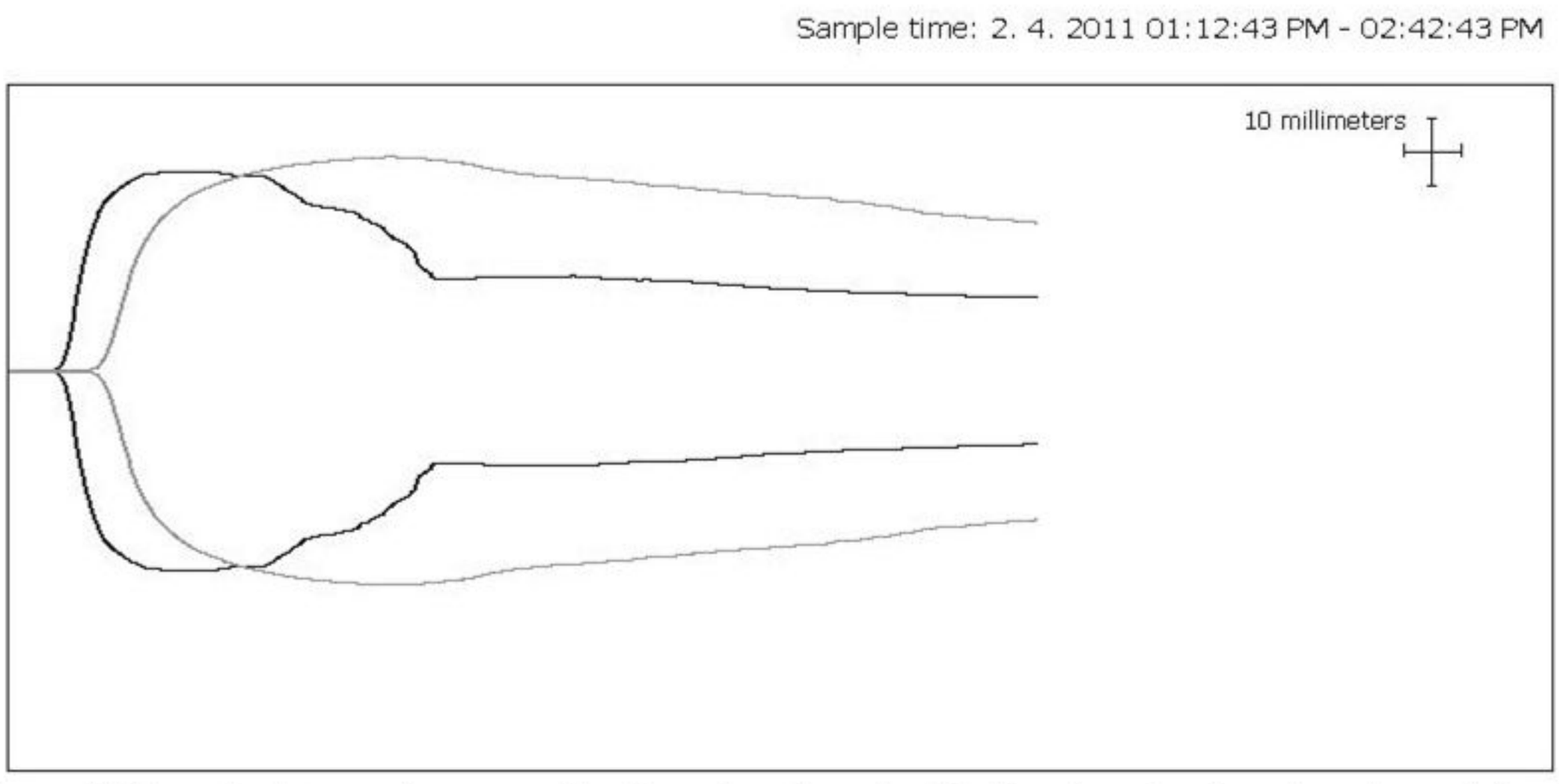


FIGURE 1: Thromboelastography traces of kaolin activated sample (black) and non-kaolin activated sample (grey).