We then noted that the Genius 1 was set to 'oral' mode and Genius 2 was set to 'tympanic' mode. A temperature displayed by these instruments may differ by up to 1.16 °C depending on the equivalence modes to which they are set. The thermometer adds a value to the measured reading to give an estimated equivalent temperature, for example axillary 0.04 °C, oral 0.60 °C, core 1.04 °C, rectal 1.16 °C and tympanic 0 °C. Clearly this has implications when a 'non-core' temperature mode is chosen and guidelines are based on core values. This issue is addressed in the NICE recommendations: 'healthcare professionals should be aware of, and carry out, any adjustments that need to be made in order to obtain an estimate of core temperature from that recorded at the site of measurement' [1].

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Correcting the individual measurements to their core equivalents reduced the number below the 36.0 °C threshold to 4/50 and 5/50 for Genius 1 and 2 thermometers, respectively. However, there was also variability between measurements with mean (SD [range]) core corrected temperatures for Genius 1 of 36.3 (0.2 [35.4-36.6]) °C and for Genius 2 of 36.5 (0.4 [35.5-37.2]) °C.

Previous studies have highlighted the inaccuracy of infrared tympanic thermometers in children [2, 3] and adults [4, 5]. One of these studies showed that the inter-brand difference between two such thermometers was 0.6 °C and individual right-left ear differences were as large as 2.5 °C, concluding that the use of these devices may be potentially hazardous [5]. We have abandoned the use of pre-operative core temperature thresholds for patients undergoing elective surgery. We continue to use these devices (set to core mode) but have concerns about their reliability, particularly in the detection of hypothermia.

The literature suggests infrared tympanic thermometers are neither accurate nor reproducible. Most research has centred on the ability of these devices to detect fever in paediatric practice. More research is needed regarding the performance of these devices in adults and in the setting of hypothermia.

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## Rivaroxaban for thromboembolism prophylaxis after orthopaedic surgery

We read with interest the editorial concerning rivaroxaban for thromboembolism prophylaxis after orthopaedic surgery [1]. We have recently completed an audit into the use and compliance of rivaroxaban used for thromboprophylaxis in patients undergoing elective hip arthroplasty. We retrospectively collected the data from 50 patients who underwent hip arthroplasty via medical notes and a telephone survey. We looked at the length of treatment with our hospital's standard agent, low molecular weight heparin (LMWH), complications and patient satisfaction. Our trust then introduced a new thromboprophylactic agent, rivaroxaban, and we then completed the audit cycle with a further 50 patients.

The editorial highlighted that current clinical practice is to treat patients with LMWH during their postoperative stay in hospital and discontinue it on discharge. This was true in our trust, as we found that our patients had a mean duration of thromboprophylaxis with LMWH of 5.2 days.

Warwick et al. [2] showed (in a sample of 13 000 patients) that the peak incidence of venous thromboembolism following total hip arthroplasty was at 21.5 days.

Considering this evidence and the National Institute of Clinical Excellence guidelines [3] our trust replaced LMWH with rivaroxaban for thromboprophylaxis with a guideline for patients to receive rivaroxaban 10 mg per day, continued after discharge for a total treatment time of 35 days. Our second audit found that all 50 patients had received a total of 35 days of rivaroxaban and were therefore receiving thromboprophylaxis during the peak incidence of venous thromboembolic events.

The median verbal satisfaction score for the LMWH group was 3 compared with 5 in the rivaroxaban group, indicating good acceptability (1 being most dissatisfied up to 5 being most satisfied). Within the two groups there was no difference in the documented associated complications. One patient in the LMWH group developed a pulmonary embolus on day 23 after surgery.

In addition to rivaroxaban, another oral anticoagulant, which is a direct thrombin inhibitor, has been licensed for thromboprophylaxis. The efficacy of dabigatran (Pradaxa, Boehringer Ingelheim, Germany) has been demonstrated when compared to LMWH [4, 5]. No studies have compared rivaroxaban with dabigatran and little evidence exists concerning the bleeding risk of these new agents.

Our audit suggests that rivaroxaban is easy to introduce into clinical practice as a thromboprophylactic agent to cover the recommended extended period and has high patient satisfaction rates. These newer agents should allow hospitals to improve venous thromboprophylaxis cover as outlined as a key priority within the NHS.

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# Sedation by non-anaesthetists: an unacceptable risk?

Edwards et al. [1] are to be applauded for their efforts to provide a sedation service delivered by non-anaesthetists, using propofol and opioids. There is no doubt that there is increasing pressure on anaesthetists to relinquish control of what is seen to be less than general anaesthesia. However, most anaesthetists would consider sedation more challenging than general anaesthesia and mortality data would bear this out. Quine et al.'s seminal paper of 1995 [2] presented approximately 12 000 gastroscopies under sedation with a 30-day mortality due to the procedure of 1:2000; at least two of these deaths could be attributed to the sedation, giving a 1:6000 mortality; compared with a mortality for general anaesthesia of approximately 1:100 000 [3]. Are patients sedated by non-anaesthetists told at consent that their risk of death is approximately 15 times greater than it would be for a general anaesthetic administered by a trained anaesthetist? The problem is that most patients, and healthcare workers, wrongly consider sedation to be safer than general anaesthesia.

There are many published series of sedations by non-anaesthetists administering propofol in the literature, but it is important to remember the 'rule of 3' when attempting to work out the mortality in a group where no mortality has occurred. This is that risk = 3/no. of observations [4], i.e. Edwards et al.'s series of 4342 cases without a death means that they can be 95% confident that the mortality of the service is no worse than 1:1447. This is quite an achievement, but no better than Quine et al.'s series in 1995, and still approximately 70 times the likely risk of a general anaesthetic given by a trained anaesthetist.

I believe that sedation can be safe in the very controlled circumstances described in this study, but the danger is 'mission creep,' such that others dumb down the rigidly controlled system in Glasgow, so that propofol sedation given by non-anaesthetists for a wide range of procedures becomes accepted, which would undoubtedly lead to unnecessary deaths.

In my own institution, it has been suggested that sedation of this nature should be given for electrophysiological procedures such as atrial fibrillation ablation, where the patient must remain completely still for over 4 h, as this is accepted practice in Germany and the USA. I would suggest that 'sedation' achieving immobility for such a period of time is, in fact, anaesthesia with an unprotected airway.

The low mortality risk for general anaesthesia of approximately 1:100 000 is yet to be achieved by any sedation series. I believe that the minimum training for the use of anaesthetic induction agents for sedation should remain the Royal College of Anaesthetists Fellowship Examination. We should not be forced into lowering standards of training and practice and therefore potentially putting patients at increased risk.

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