A laryngeal mask was inserted instead and the anaesthetic and operation concluded uneventfully.

The Hospital Risk Management Team was informed. The Hospital users were alerted to the possibility of a manufacturing/faulty batch. Timesco Surgical and Medical Ltd. was informed. The company confirmed that a faulty batch was responsible. A product recall was made on this batch, MHRA Reference No.: 2007/002/006/401/014.

Discussion

This is the first report of this kind of fault with a Timesco Surgical and Medical size 3 disposable laryngoscope blade. On this occasion, the patient came to no harm. The fragment may have gone undetected with the potential for it to be swallowed or inhaled. The consequences of this would have been far reaching. Also, this could have happened in the hands of advanced airway management practitioners in a different setting like A&E, intensive therapy unit or cardiopulmonary resuscitation where emergency intubation would have made it even more conducive for the fragment to have gone 'missing'.

The remnant of the optical fibre airway imaging system continued to illuminate the pharynx/larynx, unlike what would have happened with a reusable Macintosh blade with a bulb. The fact that this hazard is a possibility with fibreoptics affects many practitioners in acute hospital settings, and is one worth bearing in mind. It is not routine practice to check the disposable blade after 'single use' at intubation. Should this be part of routine equipment checking?

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References

1. Jephcott A. The Macintosh laryngoscope. A historical note on its clinical and commercial development. *Anaesthesia* 1984; **39**(5): 474–479.
2. Twigg SJ, McCormick B, Cook TM. Randomized evaluation of the performance of single-use laryngoscopes in simulated easy and difficult intubation. *Br J Anaesth* 2003; **90**(1): 8–13.
3. Hirsch N, Beckett A, Collinge J, Scaravilli F, Tabrizi S, Berry S. Lymphocyte contamination of laryngoscope blades – a possible vector for transmission of variant Creutzfeldt-Jakob disease. *Anaesthesia* 2005; **60**(7): 664–667.
4. Amour J, Marmion F, Birenbaum A *et al*. Comparison of plastic single-use and metal reusable laryngoscope blades for orotracheal intubation during rapid sequence induction of anesthesia. *Anesthesiology* 2006; **104**(1): 60–64.
5. Cormack RS, Lehane J. Difficult tracheal intubation in obstetrics. *Anaesthesia* 1984; **39**: 1105–1111.

The difference between peripheral venous pressure and central venous pressure (CVP) decreases with increasing CVP

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EDITOR:

While a universally accepted measure of volume status remains elusive, measurement of central venous pressure (CVP) remains widely utilized. The inherent requirement for the insertion and maintenance of a catheter within the thorax however garners a wide range of potential morbidity and occasional mortality. Recent reports of correlation between CVP and peripheral venous pressure (PVP) have prompted interest in PVP substitution for CVP. To date, correlation has been reported in the operating theatre and critical care settings for both adult [1–5] and paediatric [6] populations, without consensus as to whether reliance on PVP alone can be endorsed.

Investigators of isolated vascular beds have described a 'waterfall effect' to explain blood flow through collapsible tubing deformed by external pressures. Inherent in this concept is the summative effect of distending intraluminal, and compressive extraluminal pressures on vessel cross-section, and thereby resistance. We hypothesized the existence of vascular waterfall phenomena in the peripheral

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venous conduits of the upper limb that may impact the relationship between measured PVP and CVP. Specifically, we postulated elevated and erroneous PVP estimation of CVP at low CVP values secondary to venous collapse and associated elevation in conduit resistance. We therefore determined to compare CVP and PVP in mechanically ventilated subjects with emphasis on the observed differential.

The investigation was approved by the Northern Y Regional Committee of the New Zealand Health and Disability Ethics Committee. Thirty-four patients (27 male, 7 female) undergoing elective cardiac surgery (27 coronary artery grafting (CAG), 3 aortic valve replacement (AVR), 1 atrial septal defect repair, 3 combined CABG/AVR) were studied.

Preoperatively, 16-G peripheral catheter-over-needle cannula were sited in left antecubital fossa veins. Central venous catheters (7 Fr, 3 lumen, 16 cm; Arrow International, Reading, MA, USA) were introduced following induction of anaesthesia via the right internal jugular vein. *Truwave* pressure transducers (Edwards Lifesciences, Irvine, CA, USA) connected to Solar 9500 bedside monitors (Marquette Medical Systems, WI, USA) were used for acquisition of CVP and PVP.

CVP recordings were made via the distal lumen of the central venous catheter following mechanical flushing and visual confirmation of the CVP waveform. PVP recordings were performed following transfer of the CVP pressure transducer to the antecubital fossa catheter with the arm in the mid-thoracic position. Mean pressure values were recorded at end-expiration. Central and peripheral transducers were room air calibrated contemporaneously at the estimated phlebostatic axis in addition to undergoing inverted 'U-tube' levelling to eliminate zero error difference. Pressures recorded represented a time-weighted average of venous pressure waves as per manufacturers software.

Patients were studied on the first postoperative day in ICU while intubated and sedated with propofol infusion. Ventilator settings provided tidal volumes of 5–7 $mL\,kg^{-1}$ in the absence of positive end-expiratory pressure. Maintenance fluid was 5% dextrose infused at constant rate of 1 $mL\,kg^{-1}\,h^{-1}$. Inotropic and vasoactive infusions (epinephrine in 1, milrinone in 3, norepinephrine in 11) were continuing at the time of data acquisition.

CVP/PVP parings were recorded on return from theatre and subsequently before and after undergoing volume expansion. Volume expansion (250–500 mL Hemohes® (Pentastarch 10 g 100 mL^{-1} in 0.9% saline)) was undertaken to maintain haemodynamic parameters (mean arterial pressure (MAP) > 65 mmHg) according to local protocol, or at the discretion of the attending intensivist.

Statistical analysis of all variables was performed using SPSS for Windows (version 10, SPSS, Chicago, IL, USA). Linear regression analysis was performed to establish correlation between CVP and PVP. *t*-Test was used when comparing mean data. Kruskal–Wallis analysis of variance was used for subgroup analysis of PVP less CVP according to CVP. All data is presented as mean ± SD.

Fifty-four initial and pre-infusion paired CVP/PVP measures were obtained with 37 further parings recorded following volume expansion. Seventeen of these were in response to a MAP ⩽ 65 mmHg. At the time of initial sampling, pulse rates and MAP were 78 ± 11 bpm, and 72 ± 9 mmHg, respectively. Mean volume infused was 382 ± 180 mL (4.4 ± 1.9 mL kg^{-1}). CVP was 10 ± 3.6 mmHg prior to volume expansion rising to 12.6 ± 3.5 mmHg following Pentostarch infusion ($P = 0.001$). Similarly PVP rose from 11 ± 3.7 to 13 ± 3.3 mmHg ($P = 0.007$).

PVP was found to be greater than CVP by 1.13 ± 1.9 mmHg ($P < 0.001$). Mean increments in CVP and PVP recordings (∂CVP and ∂PVP) were 2.6 ± 2.6 and 1.9 ± 3.3 mmHg, respectively. There was a trend towards change in PVP being less then change in CVP (∂CVP − ∂PVP was 0.64 mmHg, $P = 0.084$).

Linear regression analysis of the initial paired recordings revealed good correlation between PVP and CVP ($r = 0.864$, $P = 0.01$). Regression of these PVP/CVP pairings yielded the equation PVP = 2.88 + (0.84 × CVP). The regression equation for PVP − CVP was 2.88 − (0.16 × CVP), ($r^2 = 0.22$, $P = 0.01$). Bar plot demonstrating distribution of PVP − CVP for CVP groupings are shown in Figure 1.

Discussion

These data demonstrate a negative correlation between central and peripherally acquired venous pressure differential and absolute CVP. Subgroup analysis proporting reduction in PVP − CVP with rising CVP, and demonstration of a trend towards volume infusion resulting in lesser PVP increment lends additional support to the study hypothesis that PVP – CVP is a decreasing function CVP. An alternative statement of this relationship is that the correlation between PVP and CVP improves at higher CVP values. This finding is not new having been previously reported in a series of hepatic transplant patients undergoing investigation for PVP/CVP correlation [4]. These authors, likewise, postulated that venous collapse may have contributed to observed PVP/CVP divergence.

We have additionally observed good correlation between PVP and CVP over a wide range of CVP values consistent with a number of recent reports

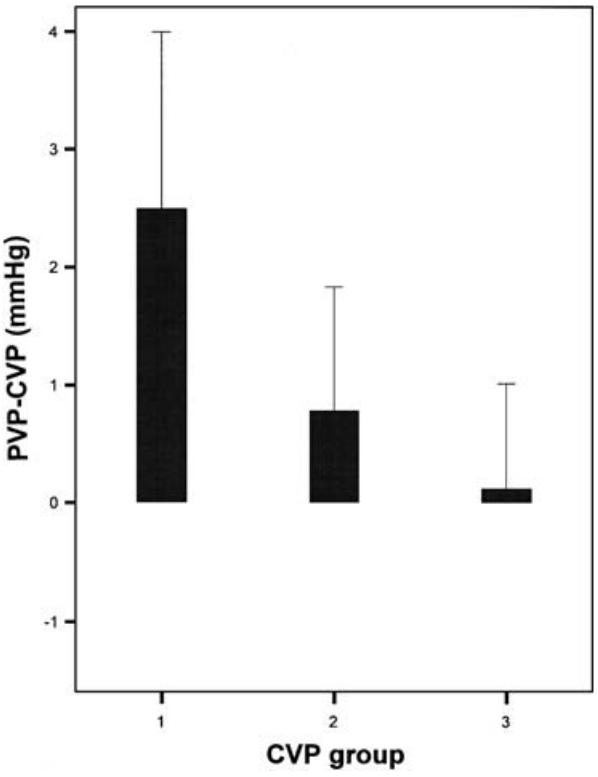

Figure 1.
Distribution of (PVP − CVP) for CVP groupings. Group 1 CVP ⩽ 8 mmHg. Group 2 CVP 9–12 mmHg. Group 3 CVP ⩾ 13 mmHg. PVP: peripheral venous pressure; CVP: central venous pressure.

demonstrating statistically significant correlation [1–5]. The limits of agreement in this study are however insufficient to advocate substitution of PVP for CVP in routine clinical practice.

The conceptual framework of the present study – investigating factors that impact on difference between CVP and PVP – represents a break from previous work, which has sought correlation alone. That we have reported positive results within this construct has implications for future research. Adopting a similar approach may be gainfully employed in designing future studies to identify subgroups of patients in which PVP and CVP are clinically interchangeable.

Earlier work [7] describing flow in compressible tubes as a function of transmural pressure described three states of resistance: (1) Collapsed conduit, where resistance is high but constant and flow occurs through open channels at the lateral aspects of the collapsed tubes only; (2) Partially open conduit, where the relationship between driving pressure (PVP − CVP) and flow is complex and non-linear. Increasing intraluminal pressure dilates the tube leading to declining resistance; and (3) Open conduit, where resistance is constant. We suggest the variation in resistance in the peripheral veins with differing transmural pressures (i.e. classic vascular waterfall effect) accounts for the relationship between PVP − CVP and CVP observed in this and other studies. Such consideration permits the cogent but unexplored hypothesis that the accuracy of PVP estimation of CVP increases markedly when unimpeded venous patency is achieved i.e. at a point wherein CVP exceeds the compressive effect of tissue pressure on venous conduits.

This study has a number of limitations. Investigators were not blinded to knowledge of the CVP before assessing PVP potentially introducing observer bias. A number of patients were at the time of data collection receiving infusions of inotropic or vasoactive medications. The effect of these on peripheral venous tone may have influenced the recorded PVP measurements. Additionally, temperature, which is known to affect correlation between CVP and PVP perhaps through an effect on venous tone [5], was not recorded. Tissue oedema may furthermore have affected the relationship between CVP and (PVP − CVP). As has been hypothesized tissue pressure may represent a critical opening pressure at the 'top of the waterfall' below which the relationship between PVP − CVP and CVP inherently non-linear. Given the small numbers in this study however, analyses other than simple linear regression to seek such a relationship were not undertaken. Finally, this study was undertaken in a population of intubated post-cardiac surgical patients. As such, the findings may not be generalizable to the more heterogeneous ICU or ward population.

In conclusion we have demonstrated (PVP – CVP) to reduce with increasing CVP, which we suggest is due to variable resistance in collapsible veins at differing transmural pressures. The clinician contemplating use of PVP in volume state assessment may find the approach of considering potential for a large PVP − CVP useful.

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References

1. Tugrul M, Camci E, Pembeci K *et al*. Relationship between peripheral and central venous pressures in different patient positions, catheter sizes and insertion sites. *J Cardiothorac Vascular Anesth* 2004; **18**: 446–450.

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2. Desjardins R, Denault AY, Belisle S *et al*. Can peripheral venous pressure be interchangeable with central venous pressure in patients undergoing cardiac surgery. *Intensive Care Med* 2004; **30**: 627–632.
3. Charalambous C, Barker TA, Zipitis CS. Comparison of peripheral and central venous pressures in critically ill patients. *Anaesth Intensive Care* 2003; **31**: 34–39.
4. Hoftman N, Braunfeld M, Hoftman G *et al*. Peripheral venous pressure as a predictor of central venous pressure during orthotopic liver transplantation. *J Clin Anesthesia* 2006; **18**: 251–255.
5. Sahin A, Salman M, Salman A *et al*. Effect of body temperature on peripheral venous pressure measurements and its agreement with central venous pressure in neurosurgical patients. *J Neurosurg Anesth* 2005; **17**: 91–96.
6. Anter AM, Bondok RS. Peripheral venous pressure is an alternative to central venous pressure in paediatric surgical patients. *Acta Anaesthesiol Scand* 2004; **48**: 1101–1104.
7. Kamm RD, Shapiro AH. Unsteady flow in a collapsible tube subject to external pressure or body forces. *J of Fluid Mech* 1979; **95**: 1–7.

Ketamine in PCA: what is the effective dose?

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EDITOR:

Although adding ketamine to patient-controlled analgesia (PCA) with morphine has proven to be safe and has shown numerous advantages, like significant lessening of morphine consumption or lessening of opioid adverse effects, these beneficial effects are far from constantly encountered. The recent randomized study of Aubrun and colleagues published in the Journal did not show any advantage of adding 0.5 mg ketamine to a 1 mg morphine bolus during postoperative period of major gynaecological surgery [1].

Javery and colleagues first found, in 1996, a significant lessening of pain scores (2.3 vs. 4.5) and of morphine consumption (51 vs. 26 mg) when ketamine was directly added to the morphine PCA using a syringe (ketamine and morphine, 1 mg mL^{-1} each). Morphine sparing resulted in less nausea, itching and urinary retention [2]. Again in 1999, a randomized study by Adriaenssens and colleagues detected a significant lessening of mean morphine consumption – 28 vs. 54 mg during the first postoperative 48 h. Ketamine was not added into the PCA, but as a 2.5 $\mu g\,kg^{-1}\,min^{-1}$ infusion [3]. There was significantly less nausea in the ketamine group. The study by Guignard and colleagues in 2002 showed that a ketamine bolus dose of 0.15 mg kg^{-1} followed by an infusion of 2 $\mu g\,kg^{-1}\,min^{-1}$ reduced perioperative remifentanil consumption and morphine consumption (46 vs. 69 mg) in the postoperative period following abdominal surgery [4].

However in 2001, a double-blind randomized trial by Reeves and colleagues concluded that small-dose ketamine combined with PCA morphine (ketamine and morphine, 1 mg mL^{-1} each) provided no benefit to patients undergoing major abdominal surgery. Analgesic efficacy was not improved, neither was opioïd consumption, but patients in the ketamine group performed worse in cognitive testing and had a relative risk of experiencing vivid dreams of 1.8 [5]. In 2002, Murdoch and colleagues did not find any benefit in the addition of ketamine to morphine in PCA for gynaecological surgery [6].

The well-designed study of Aubrun and colleagues contributes to the controversy. As the authors stated, several facts may explain the negative findings. Pain levels in the control group were not very high and morphine consumption was rather low (perhaps because of the concomitant use of an non-steroidal anti-inflammatory drug). As a consequence, the NMDA (*N*-methyl-D-aspartate) channel was not likely to stay in the open state. But we do think that the main problem resides in the chosen ketamine bolus of 0.5 mg mL^{-1}. Since ketamine dosing is closely linked to morphine PCA consumption, it may be predicted that the less the morphine consumption, the less the ketamine administration. Actually, patients received a mean dose of 44 ± 16 mg ketamine during the 48 postoperative first hours in Aubrun and colleagues's study. Because, in the smallest dose range the ketamine effective dose is not less than 2 $\mu g\,kg^{-1}\,min^{-1}$, equivalent to 6–10 mg h^{-1} in patients weighing 50–80 kg, we suggest that no effect could be expected from a dose of less than 1 mg h^{-1}.

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