Correspondence

Functional assessment tools in the intensive care unit: are we comparing apples and oranges?

Clinical interest is moving from survival towards patient functional outcomes following discharge from the intensive care unit (ICU)[1]. Currently, a large number of tools are available in assessing functional outcomes which can be especially relevant when comparing two interventions which do not have mortality benefits. However, there are difficulties in using the current functional assessment tools[2].

A. Too many tools

A recent systematic review identified 425 papers including 250 instruments assessing one or more measures of physical, cognitive, mental health, or quality of life outcomes in twenty or more survivors of critical illness[2]. This high heterogeneity decreases the ability for these studies to be compared.

Table 1

Examples of current functional assessment tools

Domain assessed	Tool
Structure and function impairment	Physical Function Test for use in the ICU (scored)* Hospital Anxiety and Depression Scale (HADS) Chelsea Critical Care Physical Assessment Tool* University of Rochester Acute Care Evaluation Functional Status Score for Intensive Care*
Activity limitation	6-minute Walk Test ICU Mobility Scale* Functional Independence Measure
Participation restriction	Follow-up Health Status Questionnaire
Quality of life	Short-Form 36 15D Health Related Quality of Life

*Tool is specifically designed for use within an intensive care unit (ICU) setting.

Table 2

Recent systematic reviews on functional assessment tools used in the intensive care unit

Reference	Papers	Unique tools	ICU validation	Quality of evidence	Criteria used
Tipping 2012[‡]	11	19	3	Poor	Not stated
Parry 2015[‡]	47	33	20	Poor to fair	COSMIN*
Robinson 2017[+#]	20	21	21	Poor to fair	COSMIN*
Peterson 2018[+‡]	34	14	14	Poor to fair	COSMIN*

*COSMIN: COnsensus-based Standards for the selection of health Measurement INstruments[6]. [+] Review of papers which had validation in an ICU setting. [#] Review of tools that assess physical function, cognitive function and quality of life. [‡] Review of physical function assessment tools only. ICU, intensive care unit.

B. Too little validation

Recent reviews have described a lack of validation for most functional assessment tools (90%) within the ICU (Table 2)[3], with quality issues including not reporting missing data, having a small sample size or lacking an a priori hypothesis.

C. Too little uptake

One review noted that ICU functional assessment comprised less than 2% of the total critical care publications between 1970 and 2013. Functional outcomes are used even less frequently as an outcome in critical care randomised controlled trials[2].

Moving functional assessment forward:

(i) Selecting a tool

Development of core outcome sets can reduce study heterogeneity, reduce selective reporting of outcomes based on results, and increase clinical relevance[4]. These outcomes are generated through discussion between the research, clinical and patient communities. Core outcome sets can be found in the Core Outcome Measurement in Effectiveness Trials (COMET) initiative database[5].

(ii) Validation studies

Validation studies will aid the development of accurate core outcome sets. The COnsensus-based Standards for the selection of health Measurement INstruments (COSMIN) checklist may be helpful[6]. This checklist discusses ten domains and two supplementary areas that should be evaluated when analysing the measurement properties of health measurement instruments. The 2011 COSMIN

Table 3

A suggested checklist for quality of a tool validation study

Property	Checklist
Study design	Will this study draw useful conclusions? 1. Are there sufficient study participants? 2. Is this study conducted in the target population?
Reliability	Is the measurement free from error? 3. Is an internal consistency statistic reported? 4. Is a measurement error statistic reported?
Validity	Does the measurement reflect reality? 5. Are measurements validated against a gold standard (if possible)? 6. Are measurements consistent across different demographics?
Responsiveness	Does the tool detect change? 7. Are patient changes reported to correlate with measurement changes?

Based on the criteria within the COSMIN (Consensus-based Standards for the selection of health Measurement INstruments) checklist four-point ranking scale[6]. An ideal study should answer "Yes" to each of these questions.

checklist four-point ranking scale provides examples of what information researchers should provide to produce the ideal validation study[6]. Table 3 is a suggested quick checklist which can be used as a preliminary method to screen the quality of a validation study. Further comparison against the COSMIN checklist is recommended to develop a more comprehensive picture of study quality.

In summary, the current functional outcome tools are vulnerable to measurement bias which can be improved through a combination of changes, including careful selection of the assessment tools, implementation of the COSMIN checklist, development of core outcome sets, and changes in publication practices. Increased commitment to reducing functional outcome research heterogeneity will produce research findings that are most relevant to our patients.

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It's hard to kill off an old paradigm—starvation in the midst of plenty

I refer to your recent excellent editorial[1] highlighting the issues surrounding euglycaemic diabetic ketoacidosis and sodium–glucose cotransporter-2 (SGLT2) inhibitors.

However, I am concerned about the statement, "In the absence of insulin, glucose remains extracellular and cells switch to ketogenic metabolism". The statement evokes the old idea of 'starvation in the midst of plenty', which was orthodox until the 1990s, when accurate measurement of radiolabelled glucose fluxes across cell membranes showed it to be wrong. Cells do not need insulin to obtain the glucose needed for their energy requirements. Glucose moves easily and without help from insulin into cells through glucose transporter 1 (GLUT1) transporters present on all cell membranes. This was comprehensively reviewed by Sonksen[2] in 2000.

Insulin does move glucose against metabolic need through glucose transporter type 4 (GLUT4) transporters, but only in liver, muscle, and adipose tissue, and then only to drive glycogen and triglyceride production. This mechanism is normal in diabetes.

Insulin works by suppressing an otherwise unchecked hepatic glucose production so as to match it to total body glucose consumption and thereby stabilise normal blood sugar levels. Less well known, insulin even at near-basal levels suppresses hepatic ketone production. Insulin-induced suppression of ketone production in patients with type 2 diabetes mellitus (T2DM) appears intact. Until now, ketoacidosis has, almost by definition, been confined to type 1 diabetes mellitus (T1DM) and has ruled out T2DM.

Stress-induced hyperglycaemia triggers a rise in insulin. Patients with T2DM are classically thought to have a normal response to the insulin ketogenic brake, but perhaps SGLT2 inhibitors are testing that normality. By blunting reactive hyperglycaemia, these drugs could be expected to also blunt a rising insulin level and this itself will affect ketone production.

Regardless, this is completely unrelated to glucose fluxes, and it is important to recognise that the statement made in the editorial is based on an old and discredited physiology idea.

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In reply

We thank Dr McLoughlin for his interest in our editorial[1]. He is indeed correct that the current proposed mechanism for intracellular ketone formation during a relative lack of insulin activity is thought to be independent of insulin-mediated glucose flux and is instead a direct effect of the hormones regulating cellular metabolism. In humans, ketones are thought to be predominantly produced in the liver in response to an elevated glucagon/insulin ratio. Sodium-glucose cotransporter-2 (SGLT2) inhibitors are associated with

an increased glucagon/insulin ratio and this is one possible explanation for euglycaemic ketoacidosis seen with their use. We apologise for discussing older proposed mechanisms for intracellular ketone formation. Nevertheless, we believe the main messages in the editorial are an accurate summary of our current understanding of SGLT2 inhibitor–induced ketoacidosis and the recommendations made are appropriate for clinical management.

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Persistent opioid use after arthroplasty

The prevalence of long-term opioid use is increasing worldwide, principally in the United States, but also in Australia. Stark et al published a large study identifying the prevalence and predictors of opioid use postoperatively in a cohort of private patients from an Australian metropolitan hospital[1]. In a subgroup of 364 orthopaedic patients, persistent opioid use at 90 days was reported in 13.7% of non–spinal surgery cases. This figure is concerning as relatively short-term prescribing may be leading to unintended longer-term opioid use in these patients. While acute pain services are well developed in most tertiary hospitals, opioid prescribing and monitoring following discharge is usually left in the hands of surgeons and general practitioners with little oversight from pain specialists.

In our smaller study from a single private metropolitan hospital we sought to determine the prevalence of opioid use at 90 days postoperatively in patients undergoing elective primary unilateral total hip or knee arthroplasty. After obtaining approval from the hospitals research ethics committee (Ethics number HPH499) and written informed consent, we asked patients to complete a questionnaire designed by a multidisciplinary team. The operations were performed by one of two experienced consultant surgeons with a standard posterolateral approach for hip arthroplasty and medial parapatellar approach for knee arthroplasty. The primary anaesthetic technique for both was central neuraxial block (spinal anaesthesia, bupivacaine 0.5%, no additives) combined with propofol-based sedation. Analgesia consisted of high-volume local anaesthetic infiltration (100 ml of ropivacaine 0.2%) and oral multimodal analgesia including paracetamol, meloxicam, pregabalin, and tapentadol. In addition, total knee arthroplasty patients received a continuous adductor canal catheter infusion for three to five days (ropivacaine 0.2% at 6 ml per hour). All patients received education regarding opioid use, multimodal analgesic regimens and opioid tapering post-discharge. This included formal preoperative education and informal bedside discussions while an inpatient, followed up with written guidelines on discharge.

One hundred and sixty-five consecutive patients were included. Nintety-three patients responded, 39 male and 54 female, predominantly American Society of Anesthesiologists physical status 2 with a mean age of 66 years and mean body mass index of 30 kg/m^2. Fifty-two patients had undergone a primary total hip arthroplasty and 41 had a primary total knee arthroplasty. Eleven patients were taking opioid analgesia preoperatively, and the remaining 82 were not taking any opioid medication (‘opioid naïve’). At three months postoperatively three of 82 (3.6%) opioid naïve patients were still taking opioids. Five of 11 patients (45%) who required opioids preoperatively remained on opioids.

Our relatively small single-centre prospective observational study showed a low prevalence of persistent opioid use in opioid naïve patients and a reduction in chronic opioid users. This compares favourably with the paper from Stark et al. However, our study had significant limitations. The number of participants was low, as was the response rate, which may have introduced bias with the potential for patients still on opioids reluctant to respond. While we collected data on risk factors for persistent opioid use, our study was underpowered to identify specific risk factors. Stark et al noted anxiety, attendance at a preoperative clinic and high self-reported pain scores at 90 days as identifiable risk factors. In a much larger study from the United States, Namba et al analysed almost 24,000 patients undergoing knee arthroplasty and listed 14 additional risk factors. In this study they noted that 60% of patients used opioids before surgery and 41.2% continued to do so three months after surgery[2]. These figures may not reflect the cohort of cases we deal with in a private hospital setting in Australia.

Although we cannot draw any conclusions from our small study we would support the important work done by Stark et al and advocate identifying persistent opioid use at 90 days as an important metric of postoperative outcome. As noted by Pratt in the accompanying editorial of Stark’s study a whole range of strategies are recommended including the use of opioid risk screening tools, education, opioid reduction strategies and defined interventions to reduce the risk of persistent opioid use[3]. These are all worthy aims but it is not clear how we can achieve these goals in the setting of a private hospital. Our single institution performs approximately 2,400 joint replacements per annum and

currently we have no defined coordinated strategy for dealing with persistent opioid use after surgery. Nationally there were 108,073 hip and knee arthroplasties performed in 2016. The majority of these (60%–70%) were performed in private hospitals and this number is increasing by 3%–4% annually[4]. Extrapolating this data using Stark's prediction of ~13.7% persistent opioid use after surgery we can estimate that joint arthroplasty may be generating at least 10,000 new persistent opioid users every year. At the very least we would advocate identification and monitoring of the prevalence of persistent opioid use postoperatively across more hospitals with greater data sharing between institutions. Multimodal strategies to deal with this problem are clearly required but as to how, and by whom these services can be delivered particularly in the private hospital setting remains unanswered.

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Primum non nocere: greening anaesthesia

Should the call to 'do no harm' apply to our environment as well as to our patients? And given that the group of potent greenhouse gases (GHGs) we use as volatile anaesthetic agents are orders of magnitude more environmentally harmful than alternatives, should we attempt to reduce the amount of GHGs in our practice[1,2]?

Our department felt that the answer to both these questions was yes, and we began a process to reduce the carbon footprint of our anaesthetic service, which staffs an acute hospital (Middlemore, 14 theatres), and a remote elective hospital (Manukau Surgery Centre, 10 theatres). This coincided with gradually changing our anaesthetic machine fleet from mostly GE Aestiva (GE Healthcare, Chicago, IL, USA) to Dräger Zeus® (Drägerwerk AG, Lübeck, Germany) during 2013. The new machines had particular appeal because we have had a penchant for low fresh gas flows (FGF) and waste reduction as part of our ethos, as well as a passion for regional anaesthesia, with its low GHG emissions. Surprisingly, volatile anaesthetic consumption initially appeared to go up when we bought our new machines (unpublished observations), despite the supposed efficiency gains and cost savings attributable to the option of target-controlled end-tidal volatile concentration, and the ability to use basal FGF rates.

Our department is certainly not the first Australasian anaesthetic department to consider how to reduce GHG emissions[3]. Others have called for jurisdictionally mandated solutions and enforced change[4].

Using the language of psychology, Johnson and May have explained that "successful behaviour change interventions operationalised in complex organisational environments are likely to require intervention types that lead to normative and relational restructuring (and hence a focus on collective rather than individual action) and the legitimisation of new practice norms through experience"[5]. In more practical terms, we wanted to change our knowledge, values, and behaviour. We needed to intervene in ways that would reframe what we all did together, as well as in our individual practice.

Our goal was to lower our carbon footprint as follows:

- Education on the relative carbon costs of different volatile agents and non-volatile techniques to our entire group, which included over 70 specialists, and the 90 Auckland anaesthesia trainees, rotating 20 at a time through our facilities, with advice to reduce carbon intensity by minimising use of desflurane (approximately 20 times environmentally worse than sevoflurane[6]), lowest possible safe flows during inhalational anaesthesia, and encouragement of regional or total intravenous anaesthesia (TIVA) techniques where possible.
- Education on optimum use of our anaesthesia machines.
- Developing an email forum for discussion, allowing other opinion setters to have a voice, and encouraging a team approach.
- A monthly calculation of carbon footprint, for simplicity based upon volatile use only (nitrous oxide use has been negligible for some time in our department). By knowing volume of volatile agents dispensed, density, and their global warming potential (GWP) (I initially used GWP20 [i.e. GWP over 20 years], later GWP 100 [GWP over 100 years]), it was possible to calculate total monthly GHG release from clinical work.
- Relating carbon footprint to anaesthetic department clinical output. This makes trends in efficiency more apparent in the context of variable workload. I used minutes of anaesthetic time (excluding local anaesthetic cases); data were recorded by theatre nurses for every case.

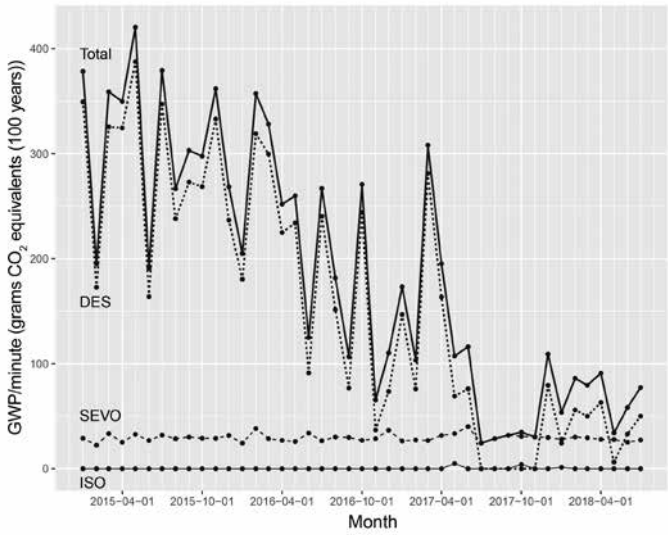

Figure 1: Global warming potential (100 years) per minute of anaesthesia, January 2015—present. Global warming potential (GWP) is shown for each component: desflurane (DES), isoflurane (ISO), sevoflurane (SEVO) and total Middlemore Hospital volatile anaesthesia (MMH).

- As interest grew, a simple group decision allowed us to set sevoflurane as the 'default' vaporiser on all anaesthetic machines (left mounted).
- Publishing monthly data in graphical form via email to all members of the department, clinical and non-clinical. In this forum, we attempted to influence, encourage and enthuse, with no attempts to compel. We appealed to competition and team spirit.

For simplicity, the data shown are from Middlemore Hospital (Figure 1), and are provided with the hospital's approval. (The data for our other, remote elective hospital were similar). The case-mix at Middlemore hospital includes (but is not limited to) surgery for burns, orthopaedics, reconstructive plastics, and hand, spine, vascular, and general surgery. It is the busiest obstetric unit in the country.

Our data show that over the past three years the use of desflurane has steadily decreased. On the basis of gram CO_2 equivalent (100 years)/minute (gCO_2e/min), our anaesthetics have dropped from approximately 250–300 to less than 100. We manage some months without opening a single bottle of desflurane. This significant reduction in volatile-derived carbon footprint (both total and per minute) has been achieved without any technological solution such as volatile capture (not commercially available to us) or recycling (unavailable anywhere at present). It may be possible for us to target 50 gCO_2e/minute or even lower, particularly if desflurane use is avoided. We feel our department can now be considered proponents of 'green anaesthesia'.

Comparisons can be thought-provoking, and individuals in other New Zealand hospitals are now collecting and sharing their data[7,8]. GHG emissions should be interpreted in the light of local factors such as surgical subspecialties, patient demographics, and entrenched practices, but their success will be measured by a downward trend in their indexed footprint rather than absolute values. The opportunities and even the motivations for sharing successful GHG strategies and manoeuvres will of course come from comparisons.

Our carbon reduction strategy—inform, audit, encourage and iterate—represents a reduction of about 600 tonnes of CO_2e per year, which we plan to maintain every year. Embedding change is more likely if our strategies support multifaceted interventions and create a facilitating framework for that change[9]. We believe we have achieved and embedded a transition, and hope to go further.

In summary, our department has embraced a philosophy and practice of which we are proud, using a process informed by theory and evidence. Without any compulsion, there has been a change in culture, behaviour, and atmospheric pollution. The environmental benefits are significant and there are cost savings; the benefits to us as collegial professionals are less easily measurable, but just as valuable.

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Use of the LMA® Gastro™ Airway, a novel dual channel laryngeal mask airway, for endoscopic retrograde cholangiopancreatography: a report of two cases

We report two endoscopic retrograde cholangiopancreatography (ERCP) procedures undertaken with the LMA® Gastro™ laryngeal mask airway (Teleflex Medical, Athlone, Ireland, Figure 1), a device shown to yield a high rate of endoscopy success, along with an excellent airway insertion success rate[1]. Written informed consent for use of this novel device and publication of the reports was obtained from each patient prior to their procedure.

Our cases involved a 72-year-old male with normal body mass index (BMI) of 24 kg/m^2, and an obese 73-year-old female (BMI 35 kg/m^2). After patient self-positioning in a conventional semi-prone ERCP position, induction of general anaesthesia was undertaken with fentanyl and propofol and no neuromuscular blockade was used. In both cases, a size 4 LMA Gastro Airway was inserted without difficulty on the first attempt, and the ERCP procedure commenced.

Maintenance of anaesthesia in both cases was uneventful. At the conclusion of the procedures, the patients were turned supine and their laryngeal masks were removed upon emergence. They were conveyed uneventfully to the recovery room, and discharged within 90 minutes.

The LMA Gastro Airway has both a large bore channel (14 mm) suitable for passage of an endoscope, and a separate channel for ventilation and monitoring of end-tidal carbon dioxide. These channels provided effective separation of the gastrointestinal and respiratory tracts in the two patients studied. In these two cases, the dual channel LMA allowed for rapid airway securement in the prone position and enabled an unobstructed airway during ERCP, providing a viable alternative to a standard LMA, endotracheal intubation, or simple airway manoeuvres during sedation.

There are many reasons why general anaesthesia with an endotracheal tube may be preferred over deep sedation alone for patients undergoing ERCP. The advantages of this technique include reduced risk of cardiopulmonary complications, a lower failure rate[2], and reduced patient discomfort[3]. Conversely, sedation alone has potential advantages, including improved operating room efficiency, patient cooperation during ERCP, and improved patient satisfaction[4]. This diversity in anaesthetic practice for patients undergoing ERCP suggests that there is a need to explore alternative, effective methods to provide an unobstructed airway and positive pressure lung ventilation.

Selection of an appropriate airway device during ERCP is complex and often based on personal preference, with no accepted uniform approach. Unwell patients or those at risk of aspiration require a higher level of airway protection, whereas others may be able to tolerate the procedure under minimal sedation. Given the significant potential for airway-related complications during ERCP, the use of the LMA Gastro Airway may provide an additional safe and effective approach to airway maintenance and may confer advantages over current practice, such as prevention of airway obstruction, provision of airway securement, and facilitation of positive pressure lung ventilation in the prone position.

Figure 1: LMA® Gastro™ (Teleflex Medical, Athlone, Ireland).

Declaration of interest

Clinical Associate Professor Marcus Skinner is the creator of the LMA Gastro Airway. He holds a part-time consultancy agreement with Teleflex. Teleflex did not influence the content of this article, nor did the company review it before submission.

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A cost comparison between total intravenous and volatile-based anaesthesia

In a recent issue of *Anaesthesia and Intensive Care*[1], Wong et al cited "additional expense" as a reason for not using total intravenous anaesthesia (TIVA). In fact, 28% of infrequent users gave this as a reason for not using TIVA.

In 2016, we conducted an extensive cost analysis of anaesthesia at Box Hill Hospital, Victoria. One of the surprising findings was the fact that TIVA was significantly less costly than a comparable sevoflurane anaesthetic at a fresh gas flow of two litres per minute. TIVA was also found to be five to ten times less costly than a desflurane anaesthetic at a fresh gas flow of a half to one litre per minute.

The exact costings of course vary considerably from case to case, but in our analysis we included consumables such as larger syringes and extension tubes for TIVA, as well as remaining propofol or remifentanil in discarded syringes.

The drug costings were obtained via personal correspondence with our pharmacy department, and represented Australian currency acquisition costs as at 2016.

Our findings are summarised in Table 1. TIVA drug discards are included as all calculations are rounded up to the next ampoule. Additional consumables are not included—A$1.43 per extension tube and A$0.40 for larger syringes. It also assumes an ideal fresh gas flow sequence[2] to minimise volatile agent wastage. At six litres per minute, one additional minute of 8% sevoflurane is over A$1, and one minute of 9% desflurane is over A$3.

Table 1

Calculated drug costs for total intravenous anaesthesia, sevoflurane, and desflurane for one to four hours

	Duration of anaesthetic			
	1 hr	2 hr	3 hr	4 hr
Propofol*	$2.88	$5.04	$6.48	$8.64
Remifentanil*	$2.20	$2.20	$4.40	$4.40
Total TIVA drug costs	$5.08	$7.24	$10.88	$13.04
Sevoflurane**				
2% at 1 l/min FGF	$4.48	$6.98	$9.48	$11.98
2% at 2 l/min FGF	$6.15	$11.16	$16.17	$21.18
Desflurane**				
6% at 0.5 l/min FGF	$25.29	$36.67	$48.05	$59.43
6% at 1 l/min FGF	$32.89	$55.66	$78.43	$101.20
6% at 2 l/min FGF	$48.07	$93.61	$139.15	$184.69

* Propofol TCI Schnider model 4 µg/ml, remifentanil TCI Minto model 3 ng/ml (age 40 years, weight 70 kg). ** 5.5 ml of sevoflurane, 12.9 ml of desflurane and 200 mg of propofol included for induction. Propofol 200 mg A$0.72, remifentanil 1 mg A$2.20, sevoflurane A$95/250 ml, desflurane A$316/240 ml. FGF, fresh gas flow; TIVA, total intravenous anaesthesia; TCI, target-controlled infusion.

We recognise that drug acquisition costs vary from time to time, from hospital to hospital, and from country to country. Also, anaesthetic drug acquisition costs make up a small fraction of the total cost of a single healthcare episode. Historically TIVA was considered to be expensive compared with inhalational anaesthesia. However, the lower costs of intravenous anaesthetics compared to the higher costs of the newer inhalational agents such as desflurane means that TIVA should no longer be seen as an additional expense compared with inhalational anaesthesia.

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In reply

We would like to thank Drs Lam and Ng for their interesting letter in response to our recent publication[1]. They presented a very clear breakdown of comparative costs of total intravenous (TIVA) versus volatile anaesthesia in their institution and it is encouraging to see that the cost of delivering TIVA is less than that of using desflurane and sevoflurane. They aptly made the point of variations in drug acquisition costs and, commensurate with this, we have calculated the comparative costs of the two techniques in our own institution, a public teaching hospital in Hong Kong. Assuming an exchange rate of A$1 to HK$5.96, the cost of drugs in Hong Kong in terms of Australian dollars are as follows: propofol $1.15; remifentanil 1 mg $14.05; remifentanil 2 mg $24.48; sevoflurane 250 ml $171.35; desflurane 240 ml $157.27. Remifentanil is particularly expensive in Hong Kong being between 5.6 to 6.4 times more expensive than in Australia. An estimate of the additional disposables would be around $3 per case. Using the same assumptions of drug consumption as Drs Lam and Ng, the cost of the different techniques is shown in Table 1.

In Hong Kong the cost of using TIVA is higher than that of 2% sevoflurane up to 2 litres per minute of fresh gas flow (FGF) and 6% desflurane at 0.5 litres per minute FGF. Of course, this is slightly simplistic as many anaesthetists using volatile agents may also use remifentanil for intraoperative analgesia. Although anaesthetists have a perception of TIVA being expensive, with generic drugs available in many countries now, this may not always be the case. One should

Table 1

Relative cost of total intravenous and volative anaesthesia in Australia versus Hong Kong.

	Duration of anaesthesia			
	1 hr	2 hr	3 hr	4 hr
Propofol	$2.88	$5.04	$6.48	$8.64
	($4.60)	($8.05)	($10.35)	($13.80)
Remifentanil	$2.20	$2.20	$4.40	$4.40
	($12.76)	($12.76)	($25.52)	($25.52)
Total TIVA drug costs	$5.08	$7.24	$10.88	$13.04
	($17.36)	($20.81)	($35.87)	($39.32)
Sevoflurane				
2% at 1 l/min FGF	$4.48	$6.98	$9.48	$11.98
	($8.08)	($12.59)	($17.10)	($21.61)
2% at 2 l/min FGF	$6.15	$11.16	$16.17	$21.18
	($11.09)	($20.13)	($29.17)	($38.20)
Desflurane				
6% at 0.5 l/min FGF	$25.29	$36.67	$48.05	$59.43
	($12.59)	($18.25)	($23.91)	($29.58)
6% at 1 l/min FGF	$32.89	$55.66	$78.43	$101.20
	($16.40)	($27.70)	($39.03)	($50.37)
6% at 2 l/min FGF	$48.07	$93.61	$139.15	$184.69
	($23.92)	($46.59)	($69.25)	($91.91)

The cost of providing the same anaesthetic in Hong Kong in terms of Australian dollars are shown in parentheses. This is calculated by multiplying the relative cost of acquiring the drug with the dollar values previously calculated by Lam and Ng[4]. For example, 200 mg of propofol costs $0.72 in Australia and A$1.15 in HK. Therefore the hourly cost of propofol in HK is (1.15/0.72) x A$2.88 = A$4.60.

also be cognisant of less tangible costs associated with surgery such as postoperative delirium, pain, and nausea and vomiting which maybe be reduced with TIVA[2,3]. While it is prudent to be aware of drug costs and not be inappropriately profligate, we have a duty of care to our patients to provide the best and safest perioperative care.

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Postoperative residual curarisation is still an issue when weaning patients in intensive care following cardiac surgery

Residual curarisation is well recognised after cardiac surgery, particularly after pancuronium[1]. Magnesium, often used in cardiac anaesthesia to prevent hypertension and arrhythmias, potentiates the action of neuromuscular blocking agents (NMBAs) and may result in prolongation of the neuromuscular blockade. Residual curarisation has become more relevant in the past 20 years with the introduction of fast-tracking after such procedures[2]. Before applying fast-tracking in our cardiosurgical unit, we sought to determine the incidence of postoperative residual curarisation after a traditional postoperative six-hour sedation period, as practised in our department after cardiac surgery until now. For the intraoperative maintenance of muscle paralysis, anaesthetists in our hospital tend to use repeated doses of NMBAs during surgery. The continuous use of NMBAs during cardiopulmonary bypass to increase tissue oxygen saturation is controversial, and continuous infusions of NMBAs during cardiac surgery are therefore not used anymore in our department[3,4].

Postoperative residual curarisation can only be avoided by neuromuscular transmission monitoring and appropriate antagonism of the neuromuscular block with reversal agents. However, such neuromuscular transmission monitoring is seldom used during cardiac surgery because cardiosurgical patients typically remain ventilated postoperatively in the intensive care unit (ICU). Frequently, the anaesthetist administers an additional bolus of NMBA at the end of surgery to 1) facilitate surgical wound closure, 2) facilitate reintubation of the patient with a single-lumen endotracheal tube after procedures such as Port-Access (involving mini-thoracotomy) or minimally invasive direct coronary artery bypass (MIDCAB) surgery where a double-lumen endotracheal tube is used intraoperatively, and 3) facilitate ventilation during transportation of the patient to the ICU. Postoperative residual curarisation following the use of NMBAs is likely frequently undetected in the ICU, as no or limited neuromuscular transmission monitoring is performed by ICU doctors and nurses. It is assumed that the time interval between the final NMBA dose and extubation of the ICU patient is sufficient to allow for the spontaneous reversal of the neuromuscular blockade[5]. Moreover, many technical and interpretive issues can interfere with the accuracy and

reproducibility of neuromuscular transmission monitoring measurements in ICU patients[6]. The level of neuromuscular blockade in ICU patients is thus often unknown. This management of neuromuscular blockade contrasts with that in the operating theatre, where anaesthetists tend to use train-of-four monitoring to interpret muscle tone.

This study (ref: 2017/067) was approved by the Ethics Committee of the Onze-Lieve-Vrouw Ziekenhuis, Aalst, Belgium, on 11 August 2017 and was registered with ClinicalTrials.gov (ref: NCT03291184). Patients gave their written informed consent. The main objective of this observational, single-centre, prospective study was to describe the incidence of postoperative residual curarisation (mean train-of-four ratio <90%) when patients admitted to the ICU were weaned from ventilation after elective cardiac surgery. We consecutively enrolled patients scheduled for cardiac surgery during a three-month period. The exclusion criteria were emergency surgery, evidence of neuromuscular disorders, or a history of medication that interfered with neuromuscular transmission. After surgery, patients were transferred to the ICU for postoperative ventilation and monitoring. Readiness for weaning was based on ICU doctor- or nurse-driven institutional weaning guidelines as follows: six hours after arrival in the ICU, and a normothermic patient who was haemodynamically stable, had normal blood gases and had no residual bleeding. The ICU nurse caring for the patient started weaning the patient from the ventilator as soon as an ICU doctor had performed three consecutive measurements of the train-of-four ratio at the adductor pollicis with the TOFscan neuromuscular transmission monitor (iDMed, Marseille, France). The TOFscan is the standard monitor used in our department and provides a reliable measurement of evoked muscle responses using three-dimensional acceleromyography. The mean of the three measurements was recorded as the train-of-four value for that individual. Each mean value below 90% was considered as postoperative residual curarisation, and in that case, the attending ICU doctor caring for the patient was informed of the result of the measurement so that he/she could reverse the patient's neuromuscular block appropriately with neostigmine or sugammadex, according to the level of block, at the discretion of the attending ICU doctor.

In the present study, we recorded the following variables: gender, age, weight and height of the patient, EuroSCORE II, preoperative serum creatinine level, preoperative bilirubin level, the preoperatively described left ventricular ejection fraction (<30%, between 30% and 50%, or higher than 50%), and type of cardiac surgery. In particular we noted whether circulatory arrest was used in any of the patients, and the bypass and cross-clamp times. We recorded the operating room time, total intraoperative NMBA dose and the time between final NMBA dose and ICU arrival, as well as the core temperature on arrival to ICU. Additionally, we collected

Table 1

Patient characteristics and perioperative data

Variable	Patients without postoperative residual curarisation (n=85)	Patients with postoperative residual curarisation (n=8)	*P*-value
Sex Male (%) Female (%)	 63 (74%) 22 (26%)	 4 (50%) 4 (50%)	0.21
Age, years	68 (10)	71 (14)	0.27
Height, cm	171 (10)	170 (8)	0.45
Weight, kg	80 (17)	75 (17)	0.52
EuroSCORE II	3.08 (2.75)	3.38 (2.46)	0.57
Creatinine level, µmol/l	97 (35)	88 (27)	0.72
Serum bilirubin level, µmol/l	8.6 (5.1)	10.3 (5.1)	0.40
Ejection fraction <30% (%)	3 (3%)	0	1.00
Type of cardiac surgery* CABG (%) Valve replacement or reconstruction (%) Combined or other procedure (%)	 39 (46%) 26 (31%) 20 (23%)	 2 (25%) 4 (50%) 2 (25%)	0.57
CPB duration, min	129 (45)	162 (44)	0.06
ACC duration, min	91 (37)	107 (38)	0.19
Operating room time, min	292 (74)	316 (68)	0.25
Total intraoperative NMBA (rocuronium) dose, mg	143 (42) †‡	187 (45)	0.01
Time between final NMBA dose and ICU arrival, min	74 (64)	69 (84)	0.14
Core temperature on arrival to ICU, °C	34.8 (0.9)	35.0 (0.8)	0.60
Decision to start ICU weaning, min after ICU arrival	363 (183)	305 (78)	0.49
Train-of-four % at decision to wean in ICU	98 (2)	59 (28)	<0.0001
Reversal agent	Not applicable	Neostigmine (n=6) Sugammadex (n=2)	
Time to extubation, min	605 (468)	582 (296)	0.72
Extubation on first attempt (yes) (%)	37 (43%)	8 (100%)	0.002
ICU stay, days	4 (3)	5 (1)	0.04
Hospital stay, days	10 (5)	11 (3)	0.14

Categorical variables are reported as frequencies (%) and were analysed using Fisher's exact test or chi-squared test. Continuous variables are reported as mean (standard deviation) and were analysed using the Mann–Whitney U test. CABG, coronary artery bypass grafting; CPB, cardiopulmonary bypass; ACC, aortic cross-clamp; NMBA, neuromuscular blocking agent; ICU, intensive care unit. *No patient underwent circulatory arrest. †Value is for 83/85 patients; only those who received rocuronium were considered; 2/85 subjects received cisatracurium. ‡One patient received an additional NMBA bolus dose in the ICU.

the following information: whether patients received an NMBA during their ICU stay prior to weaning, the time from arrival in ICU to the weaning decision, the mean train-of-four ratio at weaning from the ventilator and whether reversal agents were administered. Finally, we recorded the length of mechanical ventilation, and whether the patient's trachea was extubated on the first attempt, and other clinical outcomes, including ICU stay, hospital stay, and mortality.

Ninety-three cardiosurgical patients were enrolled in this study. No patient died in the 30-day postoperative follow-up period. In contrast to our expectations, which were based on pharmacological considerations, postoperative residual curarisation was encountered in 8/93 (9%, 95% confidence interval 4% to 16%) patients who were scheduled to be weaned from the ventilator in the ICU at six hours after elective cardiac surgery (Table 1). No differences in patient characteristics and perioperative data were observed between patients without (n=85) and with (n=8) postoperative residual curarisation. However, a significant difference was found in the total NMBA dose given intraoperatively, with a much higher drug dose administered to patients with postoperative residual curarisation. Curiously, no difference was observed in the time interval between the final NMBA dose in the operating room and the ICU arrival in patients without or with postoperative residual curarisation. All patients with postoperative residual curarisation had a train-of-four count of four, except for one subject who had a deep neuromuscular block (train-of-four count of zero with a post-tetanic count of 10) and fully recovered after an appropriate dose of sugammadex. This patient was a 79-year-old female with a weight of 66 kg, a creatinine level of 106 µmol/l, a bilirubin level of 17.1 µmol/l, and a normal left ventricular ejection fraction; however, she received a total intraoperative rocuronium dose of 190 mg, and the final dose was given 23 minutes before arrival in the ICU. One other patient was administered sugammadex by the ICU doctor after a train-of-four count of four and a train-of-four ratio of 56%. ICU length of stay was increased in patients with postoperative residual curarisation, however, we feel it is impossible to attribute this outcome to residual neuromuscular blockade in this small series of patients.

In conclusion, postoperative residual curarisation was encountered in 9% of patients who were scheduled to be weaned from ventilation in the ICU six hours after elective cardiac surgery. Although this study had some limitations (small sample size and statistical analysis between unequal groups [85 versus 8 subjects]), it suggests that routine quantitative neuromuscular monitoring should be performed in the ICU before tracheal extubation after cardiac surgery.

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