Land of confusion

Venema, Allart M.; Uyttenboogaart, Maarten; Absalom, Anthony R.

Published in:
British Journal of Anaesthesia

DOI:
10.1016/j.bja.2018.12.004

Funding

Supported in part by grant R37 GM48085 to JCE and R01 DA15353 to TLY from the National Institutes of Health, Bethesda, MD, USA.

References

1. Zel J, Hadzik A, Cvetko E, et al. Neurological and histological outcomes after subarachnoidal injection of bupivacaine liposome injectable suspension in pigs: a pilot study. *Br J Anaesth* 2019; **122**: 379–87
2. Lambert LA, Lambert DH, Strichartz GR. Irreversible conduction block in isolated nerve by high concentrations of local anesthetics. *Anesthesiology* 1994; **80**: 1082–93
3. Yamashita A, Matsumoto M, Matsumoto S, Itoh M, Kawai K, Sakabe T. A comparison of the neurotoxic effects on the spinal cord of tetracaine, lidocaine, bupivacaine, and ropivacaine administered intrathecally in rabbits. *Anesth Analg* 2003; **97**: 512–9
4. Li DF, Bahar M, Cole G, Rosen M. Neurological toxicity of the subarachnoid infusions of bupivacaine, lignocaine, or 2-chloroprocaine in the rat. *Br J Anaesth* 1985; **57**: 424–9
5. Takenami T, Yagishita S, Murase S, Hiruma H, Kawakami T, Hoka S. Neurotoxicity of intrathecally administered bupivacaine involves the posterior roots/posterior white matter and is milder than lidocaine in rats. *Reg Anesth Pain Med* 2005; **30**: 464–72
6. Ganem EM, Vianna PT, Marques M, Castiglia YM, Vane LA. Neurotoxicity of subarachnoid hyperbaric bupivacaine in dogs. *Reg Anesth* 1996; **21**: 234–8
7. Rosen MA, Baysinger CL, Shnider SM, et al. Evaluation of neurotoxicity after subarachnoid injection of large volumes of local anesthetic solutions. *Anesth Analg* 1983; **62**: 802–8
8. Moussa T, Abdoulaye D, Youssouf C, Oumar GC, Karim TS, Traore TJ. Cauda equina syndrome and profound hearing loss after spinal anesthesia with isobaric bupivacaine. *Anesth Analg* 2006; **102**: 1863–4
9. Chen X, Xu Z, Lin R, Liu Z. Persistent cauda equina syndrome after cesarean section under combined spinal–epidural anesthesia: a case report. *J Clin Anesth* 2015; **27**: 520–3
10. Drasner K, Sakura S, Chan VWS, Bollen AW, Ciriales R. Persistent sacral sensory deficit induced by intrathecal local anesthetic infusion in the rat. *Anesthesiology* 1994; **80**: 847–52
11. Drasner K, Rigler ML, Sessler DI, Stoller ML. Cauda equina syndrome following intended epidural anesthesia. *Anesthesiology* 1992; **77**: 582–5
12. Rigler ML, Drasner K, Krejcie TC, et al. Cauda equina syndrome after continuous spinal anesthesia. *Anesth Analg* 1991; **72**: 275–81
13. Muguruma T, Sakura S, Kirihara Y, Saito Y. Comparative somatic and visceral antinociception and neurotoxicity of intrathecal bupivacaine, levobupivacaine, and dextrobupivacaine in rats. *Anesthesiology* 2006; **104**: 1249–56
14. Guo Z, Liu Y, Cheng M. Resveratrol protects bupivacaine-induced neuro-apoptosis in dorsal root ganglion neurons via activation on tropomyosin receptor kinase A. *Biomed Pharmacothe* 2018; **103**: 1545–51
15. Gold MS, Reichling DB, Hampl KF, Drasner K, Levine JD. Lidocaine toxicity in primary afferent neurons from the rat. *J Pharmacol Exp Ther* 1998; **285**: 413–21
16. Emami A, Tepper J, Short B, et al. Toxicology evaluation of drugs administered via uncommon routes: intranasal, intraocular, intrathecal/intraspinal, and intra-articular. *Intl J Toxicol* 2018; **37**: 4–27
17. Butt MT. Evaluation of the adult nervous system in preclinical studies. In: Butt MT, editor. *Fundamental neuropathology for pathologists and toxicologists: principles and techniques*. Hoboken, NJ: John Wiley & Sons; 2011. p. 321–8
18. Kohn FR, Malkmus SA, Brownson EA, Rossi SS, Yaksh TL. Fate of the predominant phospholipid component of DepoFoam drug delivery matrix after intrathecal administration of sustained-release encapsulated cytarabine in rats. *Drug Deliv* 1998; **5**: 143–51
19. Legoabe LJ, Breytenbach JC, N'Da DD, Breytenbach JW. In-vitro transdermal penetration of cytarabine and its N_4-alkylamide derivatives. *J Pharm Pharmacol* 2010; **62**: 756–61
20. Viscusi ER, Candiotti KA, Onel E, Morren M, Ludbrook GL. The pharmacokinetics and pharmacodynamics of liposome bupivacaine administered via a single epidural injection to healthy volunteers. *Reg Anesth Pain Med* 2012; **37**: 616–22

British Journal of Anaesthesia, 122 (3): 300–304 (2019)
doi: 10.1016/j.bja.2018.12.004
Advance Access Publication Date: 17 January 2019

Land of confusion: anaesthetic management during thrombectomy for acute ischaemic stroke

Allart M. Venema[1,*], Maarten Uyttenboogaart[2,3] and Anthony R. Absalom[1]

[1]Department of Anaesthesiology, University Medical Center Groningen, University of Groningen, Groningen, The Netherlands, [2]Department of Neurology, University Medical Center Groningen, University of Groningen, Groningen, The Netherlands and [3]Department of Radiology, University Medical Center Groningen, University of Groningen, Groningen, The Netherlands

*Corresponding author. E-mail: a.m.venema@umcg.nl

Stroke is a major global health issue, contributing to an age-standardised global death rate for cerebrovascular diseases of 86.5 per 100 000.[1–4] In the USA and Europe, the majority of strokes are ischaemic.[2,3] Since the introduction of intravenous thrombolytic agents, systemic reperfusion therapy has become the gold standard in the management strategy for acute ischaemic strokes.[5] However, a more recent series of studies on intra-arterial thrombectomy published in 2015 has significantly changed "the way we walk" the acute stroke management landscape.[6–11]

These abovementioned studies investigated the influence of intra-arterial thrombectomy together with standard care on outcome after acute ischaemic strokes. The first study to be published was the MR CLEAN (Multicenter Randomized Clinical Trial of Endovascular Treatment for Acute Ischemic Stroke in The Netherlands) trial.[6] The evidence of benefit was so compelling that investigators involved in other trials felt obliged to perform interim analyses, which confirmed the efficacy of intra-arterial thrombectomy—which resulted in these trials being terminated early.[7–11] A meta-analysis of five randomised trials confirmed the impressive efficacy of intra-arterial thrombectomy: the number-needed-to-treat for a favourable outcome (reduced disability of at least 1 point on the modified Rankin scale) was 2.6.[12]

These results have led to a substantial increase in the use of intra-arterial thrombectomy.[13] This increase in caseload has significant consequences for anaesthetists, as anaesthetic support for patients undergoing intra-arterial thrombectomy poses particular challenges.[14] For the treatment of acute ischaemic strokes, speed is essential, leading to the frequently stated aphorism 'time is brain'.[15–17] When intra-arterial thrombectomy is indicated and general anaesthesia is required, the personnel and equipment must be available at short notice in a radiology suite that is usually remote from the operating theatre, with potential adverse consequences for the elective or emergency procedure schedule. The increasing use of intra-arterial thrombectomy where general anaesthesia is required might necessitate changes in the logistics and staffing of operating theatres, in particular of on-call teams. Besides the logistical challenges, another emerging challenge for anaesthetists is to determine how they can best assist interventionists to optimise treatment and clinical outcome for patients undergoing intra-arterial thrombectomy. This is a hotly debated topic, and much more research is needed before there is a sufficiently solid evidence base upon which to base management guidelines.[5,11]

One debated issue is whether general anaesthesia, sedation, or local anaesthesia alone is best for patient outcome. Most of the evidence so far comes from studies not primarily designed to answer this question as the studies were designed to investigate the influence of intra-arterial thrombectomy on outcome. Campbell and colleagues[18] performed a meta-analysis of the data from seven such studies. They found that of those patients who underwent intra-arterial thrombectomy, those who did so under general anaesthesia had worse outcomes than those who received sedation or only local anaesthesia. This led the authors to conclude that general anaesthesia is harmful and, when possible, should be avoided during intra-arterial thrombectomy. Soon afterwards several groups began to avoid general anaesthesia, thereby attenuating the additional burden on their anaesthetic departments.

Do the abovementioned data[18] mean that anaesthetists can relax and retreat to the familiar territory of the operating theatre, and leave the radiologist to perform these procedures with no or only minimal operator-administered sedation? Our view is certainly not. As Dinsmore[19] pointed out in the excellent accompanying editorial, the strength of this conclusion was somewhat overstated given that the study suffered from most of the potential weaknesses of a retrospective secondary analysis of data from studies with a different goal. Among these were that the patients enrolled in these studies were randomised to treatment groups (intra-arterial thrombectomy *vs* standard care) and the choice of anaesthetic technique was

Table 1 Research suggestions

Topic	Suggested research strategy
Optimal ventilation strategy and Pa_{CO_2}	Randomisation to mild hypocarbia, normocarbia, and mild hypercarbia to determine optimal Pa_{CO_2} Investigate influence of Pa_{CO_2} on cerebral autoregulation after recanalisation
Optimal FiO_2 and Pa_{O_2}	Randomisation to different Pa_{O_2} Investigate influence of Pa_{O_2} on arteriolar tone after recanalisation
Optimal blood pressure management (targets, medications, reperfusion injury/haemorrhagic complications)	Randomisation to different arterial pressure thresholds Separate consideration of optimal haemodynamic targets before and after reperfusion Effects of arterial pressure variability during intra-arterial thrombectomy on outcome Influence of the combination of volume status and different vasoactive/inotropic agents on cerebral tissue oxygenation
Influence of anaesthetic maintenance agent on outcome	Randomised controlled trial to investigate biomarker and clinical outcome variables after different anaesthetic agents and techniques
Neurological monitoring	Feasibility and clinical and prognostic utility of cerebral oximetry/near-infrared spectroscopy/transcranial Doppler ultrasound during intra-arterial thrombectomy
Uniform reporting template and definitions	Multi-disciplinary Delphi procedure and consensus statement on uniform definitions and research parameters

at the discretion of the responsible clinicians, thereby introducing the risk of selection bias as it is plausible that patients in worse condition would be selected to receive general anaesthesia.[19,20] Moreover, important information such as whether or not there were pre-specified protocols for choice and dose of anaesthetic or sedative agents, depth of anaesthesia or sedation, and blood pressure (BP) management was often lacking.

More recently, three RCTs have specifically addressed this issue. Patients were randomly assigned to general anaesthesia or non-general anaesthesia groups.[21–23] Even though the care provided in both groups differed between studies, without exception these studies either found no difference in outcome or a tendency to better outcomes in the general anaesthesia group. These data, combined with the benefit to the interventionalist if the patient is immobile, suggest that general anaesthesia may well be the optimal option.

To be able to better guide the management of patients undergoing intra-arterial thrombectomy under general anaesthesia, important questions remain to be answered (Table 1). For example, which hypnotic agent is most beneficial for maintenance of anaesthesia? By definition each successful intra-arterial thrombectomy procedure generates an iatrogenic ischaemia—reperfusion injury. Vlisides and colleagues[24] have suggested that these procedures provide an ideal 'model' for studying the potential neurotoxic or neuroprotective effects of currently used anaesthetic agents on neurological recovery and survival. Should such studies show beneficial effects on clinical outcomes, it would provide a strong argument for using general anaesthesia during all intra-arterial thrombectomy procedures and provide guidance for the choice of anaesthetic technique to be used: total intravenous anaesthesia or inhalational.

An argument often used against providing general anaesthesia during intra-arterial thrombectomy is that general anaesthesia can lead to absolute or relative decreases in arterial BP compared with the pre-induction pressure. Radiologists, neurologists, and anaesthetists seem convinced that hypotension is harmful during intra-arterial thrombectomy procedures. However, the optimal BP management during intra-arterial thrombectomy has not yet been determined. Although general anaesthesia is associated with more hypotension and higher vasopressor requirements than conscious sedation,[21–23] no correlations between BP changes and early and late neurological outcomes have been found.[25,26] It has been argued that this lack of association is because of good compliance with strict BP management protocols in these studies.[25] Despite the weak or absent evidence, it seems logical and prudent for anaesthetists to maintain a sufficiently high BP.[25,26] The challenge remains to define an adequate individualised BP, as it is unlikely that 'one size fits all'. It is also conceivable that there are other parameters, such as duration of hypotension and variability of periprocedural BP, that are important as well.[27] Once we know the optimal BP, we also need to determine the optimal method of keeping the BP on target. Research in volunteers shows that phenylephrine and norepinephrine might actually decrease cerebral tissue oxygenation.[28]

We also need to understand better the influence of recanalisation on cerebral autoregulation, as well as the influence of Pa_{O_2} and Pa_{CO_2} on vascular tone in the affected vascular beds. Although little is known about this, it is likely that after recanalisation autoregulation is not present in the territory of the affected vessel, and that blood flow in the recanalised vessel is pressure passive (so called vasoplegia). If this is the case, then we might need different BP management strategies before and after recanalisation, or tailor-made strategies based on individual and real-time assessments of autoregulatory capacity in order to prevent haemorrhagic complications of reperfusion injury.

High Pa_{O_2} can cause vasospasm in some vascular beds.[29] Does this apply to the affected vascular bed after recanalisation such that high FiO_2 should be avoided after recanalisation? Does CO_2 reactivity remain in the affected vascular bed after re-canalisation? It seems unlikely to be present if autoregulation is absent. However, we need to know for certain because if CO_2 reactivity is absent then hyperventilation should be actively avoided, as it may cause a steal phenomenon with hyperaemia in the affected bed. If CO_2 reactivity remains, then hypoventilation (and hypercarbia) should also be avoided. If there is vasoplegia in the reperfused vascular territory, then it is also likely that the resulting regional hyperaemia influences the availability of blood for other territories.

There are many research opportunities in this area. Many of the published studies seem to have been performed without help from anaesthesia colleagues. Anaesthetists should become more involved as their deep understanding of the complex interplay between BP, Pa_{CO_2}, Pa_{O_2}, and cerebral blood flow can provide useful input during the planning and conduct of studies. We can use intra-arterial thrombectomy as a model to explain important questions of general relevance to anaesthesia (e.g. anaesthetic conditioning). Anaesthetists can also play an important role in clinical practise, even when general anaesthesia is not used. Where sedation is desirable, anaesthetists are experts at providing safe sedation to vulnerable patients and at rescuing patients from the adverse consequences of sedation.

Even if future studies show compelling evidence for avoiding general anaesthesia, there will always be situations where general anaesthesia is still needed (restless patient, failed sedation, excessive sedation, threatened airway in deeply comatose patient). In such cases, and especially when we do eventually know what BP is optimal, anaesthetists will be uniquely qualified to manage the haemodynamic status of these patients. Achievement of optimal outcomes after acute ischaemic strokes depends on factors such as speed and optimal haemodynamic management, and for these multidisciplinary teamwork and communication are essential.

Further intra-arterial thrombectomy research might identify additional ideas to implement in future studies. In resuscitation research the 'Utstein template' has been in use for more than two decades to create more uniformity in definitions and reporting of results.[30] This template has been updated, and for several specific areas of resuscitation research (e.g. drowning) additional templates have been introduced.[31] They now form a solid base for conducting resuscitation research and are used in the disciplines (anaesthesiology, cardiology, etc.) involved in this area of research. Such a consensus-based template might be of value in studying the care of patients undergoing intra-arterial thrombectomy as well.

In conclusion, currently available data are not yet strong enough to clearly guide anaesthetic management during intra-arterial thrombectomy; results are conflicting and there is a lack of standardised reporting of relevant parameters. We encourage anaesthetists working in centres performing intra-arterial thrombectomy to get actively involved in their neuro-interventional team. They can make an important contribution in researching and formulating the optimal strategy for treating and monitoring stroke patients during the perioperative phase of intra-arterial thrombectomy.

Authors' contributions

Conception of the material presented: AMV, ARA, MU.
Writing of the manuscript: AMV, ARA.
Critical revision of the material presented: AMV, ARA, MU.

Declarations of interest

AMV and MU report no conflicts of interest. ARA is an editor of the *British Journal of Anaesthesia*.

Funding

The University Medical Center Groningen.

References

1. Feigin VL, Roth GA, Naghavi M, et al. Global burden of stroke and risk factors in 188 countries, during 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet Neurol* 2016; **15**: 913–24
2. Benjamin EJ, Virani SS, Callaway CW, et al. Heart disease and stroke statistics—2018 update: a report from the American Heart Association. *Circulation* 2018; **137**: e492
3. Béjot Y, Bailly H, Durier J, Giroud M. Epidemiology of stroke in Europe and trends for the 21st century. *Presse Méd* 2016; **45**: e398
4. Naghavi M, Abajobir AA, Abbafati C, et al. Global, regional, and national age–sex specific mortality for 264 causes of death, 1980–2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet* 2017; **390**: 1151–210
5. Powers WJ, Rabinstein AA, Ackerson T, et al. 2018 Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke* 2018; **49**: e46–99
6. Berkhemer OA, Fransen PSS, Beumer D, et al. A randomized trial of intraarterial treatment for acute ischemic stroke. *N Engl J Med* 2015; **372**: 11–20
7. Goyal M, Demchuk AM, Menon BK, et al. Randomized assessment of rapid endovascular treatment of ischemic stroke. *N Engl J Med* 2015; **372**: 1019–30
8. Campbell BCV, Mitchell PJ, Kleinig TJ, et al. Endovascular therapy for ischemic stroke with perfusion-imaging selection. *N Engl J Med* 2015; **372**: 1009–18
9. Saver JL, Goyal M, Bonafe A, et al. Stent-retriever thrombectomy after intravenous t-PA vs. t-PA alone in stroke. *N Engl J Med* 2015; **372**: 2285–95
10. Jovin TG, Chamorro A, Cobo E, et al. Thrombectomy within 8 hours after symptom onset in ischemic stroke. *N Engl J Med* 2015; **372**: 2296–306
11. White PM, Bhalla A, Dinsmore J, et al. Standards for providing safe acute ischaemic stroke thrombectomy services (September 2015). *Clin Radiol* 2017; **72**: 175.e9
12. Goyal M, Menon BK, van Zwam WH, et al. Endovascular thrombectomy after large-vessel ischaemic stroke: a meta-analysis of individual patient data from five randomised trials. *Lancet* 2016; **387**: 1723–31
13. Smith EE, Saver JL, Cox M, et al. Increase in endovascular therapy in get with the guidelines—stroke after the publication of pivotal trials. *Circulation* 2017; **136**: 2303–10
14. Dinsmore J, Elwishi M, Kailainathan P. Anaesthesia for endovascular thrombectomy. *BJA Educ* 2018; **18**: 291–9
15. Froehler MT, Saver JL, Zaidat OO, et al. Interhospital transfer before thrombectomy is associated with delayed treatment and worse outcome in the STRATIS Registry (Systematic Evaluation of Patients Treated With Neurothrombectomy Devices for Acute Ischemic Stroke). *Circulation* 2017; **136**: 2311
16. Campbell BCV, Donnan GA, Davis SM. Optimizing systems of care for endovascular thrombectomy in ischemic stroke. *Circulation* 2017; **136**: 2322
17. Mulder MJHL, Jansen IGH, Goldhoorn RB, et al. Time to endovascular treatment and outcome in acute ischemic stroke: MR CLEAN Registry results. *Circulation* 2018; **138**: 232–40
18. Campbell BCV, van Zwam WH, Goyal M, et al. Effect of general anaesthesia on functional outcome in patients with anterior circulation ischaemic stroke having endovascular thrombectomy versus standard care: a meta-analysis of individual patient data. *Lancet Neurol* 2018; **17**: 47–53
19. Dinsmore J. HERMES: a helpful messenger in the anaesthesia for thrombectomy debate? *Lancet Neurol* 2018; **17**: 21–3
20. Brinjikji W, Pasternak J, Murad MH, et al. Anesthesia-related outcomes for endovascular stroke revascularization. *Stroke* 2017; **48**: 2784–91
21. Schönenberger S, Uhlmann L, Hacke W, et al. Effect of conscious sedation vs general anesthesia on early neurological improvement among patients with ischemic stroke undergoing endovascular thrombectomy: a randomized clinical trial. *JAMA* 2016; **316**: 1986–96
22. Löwhagen Hendén P, Rentzos A, Karlsson J, et al. General anesthesia versus conscious sedation for endovascular treatment of acute ischemic stroke. *Stroke* 2017; **48**: 1601–7
23. Simonsen CZ, Yoo AJ, Sørensen LH, et al. Effect of general anesthesia and conscious sedation during endovascular therapy on infarct growth and clinical outcomes in acute ischemic stroke: a randomized clinical trial. *JAMA Neurol* 2018; **75**: 470–7
24. Vlisides PE, Avidan MS, Mashour GA. Reconceptualising stroke research to inform the question of anaesthetic neurotoxicity. *Br J Anaesth* 2018; **120**: 430–5
25. Schonenberger S, Uhlmann L, Ungerer M, et al. Association of blood pressure with short- and long-term functional outcome after stroke thrombectomy: post hoc analysis of the SIESTA trial. *Stroke* 2018; **49**: 1451–6
26. Rasmussen M, Espelund US, Juul N, et al. The influence of blood pressure management on neurological outcome in endovascular therapy for acute ischaemic stroke. *Br J Anaesth* 2018; **120**: 1287–94
27. Wesselink EM, Kappen TH, Torn HM, Slooter AJC, van Klei WA. Intraoperative hypotension and the risk of postoperative adverse outcomes: a systematic review. *Br J Anaesth* 2018; **121**: 706–21
28. Poterman M, Vos JJ, Vereecke HE, et al. Differential effects of phenylephrine and norepinephrine on peripheral tissue oxygenation during general anaesthesia: a randomised controlled trial. *Eur J Anaesthesiol* 2015; **32**: 571–80

29. Kety SS, Schmidt CF. The effects of altered arterial tensions of carbon dioxide and oxygen on cerebral blood flow and cerebral oxygen consumption of normal young men. *J Clin Invest* 1948; **27**: 484–92
30. Chamberlain D, Cummins RO, Abramson N, et al. Recommended guidelines for uniform reporting of data from out-of-hospital cardiac arrest: the 'Utstein style': prepared by a task force of representatives from the European Resuscitation Council, American Heart Association, Heart and Stroke Foundation of Canada, Australian Resuscitation Council. *Resuscitation* 1991; **22**: 1–26
31. Idris AH, Bierens JJLM, Perkins GD, et al. 2015 revised Utstein-style recommended guidelines for uniform reporting of data from drowning-related resuscitation. *Resuscitation* 2017; **118**: 147–58

British Journal of Anaesthesia, 122 (3): 304–306 (2019)
doi: 10.1016/j.bja.2018.12.003
Advance Access Publication Date: 3 January 2019

Beyond 'failure to rescue': the time has come for continuous ward monitoring

Daniel I. Sessler[1,*] and Bernd Saugel[2]

[1]Department of Outcomes Research, Anesthesiology Institute, Cleveland Clinic, Cleveland, OH, USA and [2]Department of Anesthesiology, Center of Anesthesiology and Intensive Care Medicine, University Medical Center Hamburg-Eppendorf, Hamburg, Germany

*Corresponding author. E-mail: DS@OR.org

When patients having major surgery reach the post-anaesthesia care unit (PACU), families naturally assume that they have survived the most dangerous part of the perioperative experience. Their assumption is wrong. Mortality in the 30 days after surgery is 1000 times higher than intraoperative mortality.[1,2] In fact, if the month after surgery were considered a disease, it would be the third leading cause of death in the USA.[3] Most of this mortality occurs during the initial hospitalization, that is, under direct medical care in our highest-level facilities.

Most postoperative deaths are a consequence of major bleeding or cardiovascular complications.[4] Respiratory deaths are less common, but of special interest because nearly all are preventable. As might be expected from cardiovascular and respiratory deaths, hypotension and hypoxaemia on surgical wards is common, profound, and prolonged. For example, nearly 20% of postoperative patients have at least 15 continuous minutes with an MAP <65 mm Hg (unpublished data[5]). In addition, more than a third of these patients have more than a continuous hour with oxygen saturation <90% by pulse oximetry (SpO_2).[6]

Most ward hypoxaemia and hypotension is missed because ward monitoring in most hospitals consists largely of vital signs assessed at intervals of 4–8 h. For example, in recent studies routine 4 h vital signs missed about half of all patients who had an MAP <65 mm Hg for at least 15 min (unpublished data[5]) and 90% of the patients who had a continuous hour of SpO_2 <90%.[6]

Failure to detect clinically important physiological perturbations is preventable. Life-threatening ward complications including cardiocirculatory and respiratory failure are usually preceded by abnormalities in vital signs that occur minutes to hours earlier.[7] Recognition of even subtle changes in basic vital signs might allow clinical deterioration to be identified well before serious adverse events occur. Enhanced ward monitoring therefore seems likely to identify deterioration earlier than routine sparse intermittent monitoring.

Ward monitoring has hardly changed over the past half-century, although there have been major changes in hospital populations. Major changes include: (i) ambulatory surgery is routine for relatively healthy patients, with 60% of all surgery in the USA now done on an out-patient basis; (ii) it is now common to perform large operations in elderly and frail patients; and (iii) even when patients are admitted after surgery, the duration of hospitalisation is usually short. Ward patients are therefore much sicker than previously.

Battery-powered, untethered ward systems that continuously monitor a combination of physiologic variables, such as BP, electrocardiogram, HR, oxygen saturation, ventilatory frequency, body position, activity, and location, are currently available.[8,9] New biosensor material and digital developments will allow further miniaturisation of measurement systems, and non-invasive continuous measurement of other physiological variables.[10] Continuous, real-time assessment of vital signs will soon be routine, which will facilitate rapid detection of abnormalities and trigger clinical interventions.[11]

Ward monitoring will only improve outcomes if data are processed and analysed in real-time to identify patients at risk (efferent arm) and trigger an intervention by medical caregivers (afferent arm). A challenge with continuous ward monitoring is how to handle dense data streams from numerous patients.