Ten false beliefs in neuro-critical care

Geert Meyfroidt*
Department of Intensive Care Medicine, University Hospitals Leuven, Belgium
Associate Professor, Faculty of Medicine, KU Leuven, Belgium
geert.meyfroidt@uzleuven.be

David Menon
Head, Division of Anaesthesia, University of Cambridge
Honorary Consultant, Neurosciences Critical Care Unit, Addenbrooke's Hospital
Professorial Fellow, Queens' College, Cambridge
Emeritus Senior Investigator, National Institute for Health Research, UK
Chair, European Brain Injury Consortium
dkm13@cam.ac.uk

Alexis F. Turgeon
Associate Professor and Director of Research, Division of Critical Care Medicine, Department of Anesthesiology and Critical Care Medicine, and CHU de Québec – Université Laval Research Center, Université Laval, Québec City, Québec, Canada
Canada Research Chair in Critical Care Neurology and Trauma
Alexis.Turgeon@fmed.ulaval.ca

*Corresponding author

Search terms:
Traumatic brain injury – targeted temperature management – acute ischemic stroke – subarachnoid hemorrhage - intracerebral hemorrhage – intensive care unit

Word count: 1000

1. Only neuro-intensivists should care about the brain.

In acute brain injury, the need for specific expertise on central nervous pathophysiology is evident. However, even when the primary reason for ICU admission is extracranial, the brain may be affected too, through inadequate substrate and oxygen delivery, blood brain barrier leak, harmful effects of sedatives, and excitotoxicity. The resulting spectrum of brain dysfunction includes delirium, encephalopathy, coma, and non-convulsive seizures. Therefore, all intensive care should integrate neuro-intensive care, with the primary goal to preserve the brain [1].

2. Clinical examination of neuro-critically ill patients is impossible.

The patient's clinical state is our most important neuro-monitor. Clinical assessment of consciousness, cognition, brainstem, and motor function, should be attempted at least upon admission and daily [2]. Sedatives confound neurological examinations, and should be used sparingly in severe brain injuries, except for specific indications, such as intracranial pressure (ICP) control, seizure treatment, or targeted temperature management (TTM). They should be titrated and stopped if no longer indicated.

3. We should no longer monitor ICP in traumatic brain injury (TBI).

The ICP monitor has been accused of increasing therapeutic intensity, potentially harming patients without improving their outcomes. The Best-TRIP trial [3] is often wrongfully interpreted as evidence against ICP monitoring – a view that inappropriately conflates monitoring use and therapy titration. The controversy is not whether to monitor or treat ICP, but how this signal should be interpreted and responded to.

4. The threshold to treat ICP is 20 or 22 mmHg.

Changing ICP treatment thresholds from 20 to 22 mmHg in influential guidelines [4] imply that an ICP of 21 mmHg is fine, whereas an ICP of 23 mmHg should be treated aggressively. The absurdity of such strategy is obvious, because it neglects measurement errors, and ignores modern concepts such as the ICP intensity-time burden [5]. Interventional studies where aggressive treatments, decompressive craniectomy [6] or hypothermia [7], were applied early after crossing the 20 mmHg threshold, showed harm rather than benefit. A tiered approach is rational, and aggressive measures should probably be reserved for sustained ICP elevations above 25-30 mmHg unresponsive to lower-tier therapy.

5. Ketamine increases the ICP.

Ketamine-induced ICP elevations were reported in small studies in non-ventilated (and not acutely brain-injured) patients [8]. In fact, ketamine, as an adjunct in sedated mechanically ventilated patients, might decrease the ICP [9, 10], produce neuroprotection through NMDA-receptor antagonism [11], suppress harmful cortical spreading depolarisations [12], and control refractory seizures [13].

6. Subarachnoid hemorrhage (SAH) patients should get Triple H therapy.

Aggressive fluid loading (with consequent haemodilution), coupled with vasopressor administration to increase arterial blood pressure (ABP), was used in the past in the hope of preventing delayed cerebral ischemia (DCI) and vasospasm. This strategy is discredited, and might be deleterious in this population, where the median age is 50-60 years, and cardiopulmonary complications are common. Current recommendations [14] advise against haemodilution, and to aim for normovolemia. Clinical DCI should prompt a stepwise trial of ABP augmentation, titrated to neurological assessment. Where diagnosis or response to therapy are uncertain, additional investigations can help confirm or refute the diagnosis of DCI - not all late neurological deterioration in SAH is due to vasospasm.

7. There is no need to control the temperature after cardiac arrest (CA).

The evidence for TTM at 32-34°C after out-of-hospital CA, is less robust than initially assumed [15]. No difference in outcome was found between TTM at 33°C or at 36°C [16]. However, these results do not justify neglecting temperature control after CA. Targeting 32-34°C may still be defensible because of equipoise between targets, but many sites involved in the TTM trial have adopted the 36°C target [17], since it avoids potential adverse events of more aggressive cooling.

8. Hypoglycemia is harmful for the brain, hyperglycemia is not.

The optimal glycemic target for the injured brain is controversial. Tight blood glucose control to the normal fasting range (TGC) increases the risk of (deep) hypoglycemia, especially in inexperienced hands. In small observational studies, the (lower) normoglycemic range has been associated with low cerebral microdialysis glucose values [18], raising concerns about substrate delivery. On the other hand, hyperglycemia is an independent predictor of poor neurological outcome and death after CA [19]. According to a recent meta-analysis, the use of TGC showed a small but significant reduction in the risk of poor neurological outcomes in TBI [20]. So, even when the discussion on the optimal glycemic target is far from resolved, it is important not to ignore that both hypo-and hyperglycemia are associated with worse clinical outcomes in neuro-critically ill patients.

9. In acute ischemic stroke (AIS), revascularization should be done within 3 hours of symptom onset.

The conventional time window for thrombolysis in AIS is 3 hours, extendable to 4.5 hours in patients ≤80 y of age, without a history of both diabetes mellitus and prior stroke combined, a National Institutes of Health Stroke Scale (NIHSS) score of ≤25, not taking any oral anticoagulants, and without imaging evidence of ischemic injury involving more than 1/3 of middle cerebral artery territory [21]. When mechanical thrombectomy is considered, the recommended timeframe is 6 hours post-ictus, but specific penumbra-like conditions on perfusion imaging allow for longer time-windows up to 16 hours [22]. General anesthesia during thrombectomy should be avoided [23].

10. Blood pressure control in intracerebral hemorrhage (ICH): contradictory trials.

Interpreting recent trials [24–26] on the treatment of hypertension after ICH is complicated by differences in inclusion criteria, intervention timing, outcomes, antihypertensive drugs, and systolic blood pressure (SBP) targets. All these studies were conducted in patients with relatively small ICH volumes, with varying latency to achieving target SBP (ranging from 4.5 to 24h). Early intensive SBP control to targets above 140 mmHg reduces hematoma expansion, but does not improve neurological outcome or mortality. More aggressive SBP reduction to 110-140 mmHg in the ATACH-2 study [26] found no incremental benefit as compared to the 140-180 mmHg range, but a higher rate of renal complications. In brief, SBP control in ICH may reduce hematoma expansion, but not below 140 mmHg.

Conflict of interest

On behalf of all authors, the corresponding author states that there is no conflict of interest.

References

1. Tasker RC, Menon DK (2016) Critical Care and the Brain. JAMA 315:749.
2. Sharshar T, Citerio G, Andrews PJD, et al (2014) Neurological examination of critically ill patients: A pragmatic approach. Report of an ESICM expert panel. Intensive Care

Med 40:484–495.
3. Chesnut RM, Temkin N, Carney N, et al (2012) A Trial of Intracranial-Pressure Monitoring in Traumatic Brain Injury. N Engl J Med 367:2471–2481.
4. Carney N, Totten AM, O'Reilly C, et al (2017) Guidelines for the Management of Severe Traumatic Brain Injury, Fourth Edition. Neurosurgery 80:6–15.
5. Güiza F, Depreitere B, Piper I, et al (2015) Visualizing the pressure and time burden of intracranial hypertension in adult and paediatric traumatic brain injury. Intensive Care Med 41:1067–1076.
6. Cooper JD, Rosenfeld J V., Murray L, et al (2011) Decompressive Craniectomy in Diffuse Traumatic Brain Injury. New Engl J Med 364:1493–1502.
7. Andrews PJD, Sinclair HL, Rodriguez A, et al (2015) Hypothermia for Intracranial Hypertension after Traumatic Brain Injury. N Engl J Med 373:2403-2412
8. Gardner AE, Olson BE, Lichtiger M (1971) Cerebrospinal-fluid Pressure during Dissociative Anesthesia with Ketamine. Anesthesiology 35:226–228.
9. Zeiler FA, Teitelbaum J, West M, Gillman LM (2014) The ketamine effect on ICP in traumatic brain injury. Neurocrit Care 21:163–73.
10. Zeiler FA, Teitelbaum J, West M, Gillman LM (2014) The ketamine effect on intracranial pressure in nontraumatic neurological illness. J Crit Care 29:1096–1106.
11. Sakai T, Ichiyama T, Whitten CW, et al (2000) Ketamine suppresses endotoxin-induced NF-kappaB expression. Can J Anaesth 47:1019–24.
12. Hertle DN, Dreier JP, Woitzik J, et al (2012) Effect of analgesics and sedatives on the occurrence of spreading depolarizations accompanying acute brain injury. Brain 135:2390–2398.
13. Gaspard N, Foreman B, Judd LM, et al (2013) Intravenous ketamine for the treatment of refractory status epilepticus: a retrospective multicenter study. Epilepsia 54:1498–503.
14. Diringer MN, Bleck TP, Claude Hemphill J, et al (2011) Critical Care Management of Patients Following Aneurysmal Subarachnoid Hemorrhage: Recommendations from the Neurocritical Care Society's Multidisciplinary Consensus Conference. Neurocrit Care 15:211–240.
15. Nielsen N, Friberg H, Gluud C, et al (2011) Hypothermia after cardiac arrest should be further evaluated—A systematic review of randomised trials with meta-analysis and trial sequential analysis. Int J Cardiol 151:333–341.
16. Nielsen N, Wetterslev J, Cronberg T, et al (2013) Targeted Temperature Management at 33°C versus 36°C after Cardiac Arrest. N Engl J Med 369:2197–2206.
17. Nielsen N, Friberg H (2015) Temperature management after cardiac arrest. Curr Opin Crit Care 21:202–208.
18. Oddo M, Schmidt JM, Carrera E, et al (2008) Impact of tight glycemic control on cerebral glucose metabolism after severe brain injury: A microdialysis study*. Crit Care Med 36(12):3233-8.
19. Borgquist O, Wise MP, Nielsen N, et al (2017) Dysglycemia, glycemic variability, and outcome after cardiac arrest and temperature management at 33°C and 36°C. Crit Care Med 45:1337–1343.
20. Hermanides J, Plummer MP, Finnis M, et al (2018) Glycaemic control targets after traumatic brain injury: a systematic review and meta-analysis. Crit Care 22:11.
21. Powers WJ, Rabinstein AA, Ackerson T, et al (2018) 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 (Epub ahead of print).
22. Albers GW, Marks MP, Kemp S, et al (2018) Thrombectomy for Stroke at 6 to 16 Hours with Selection by Perfusion Imaging. N Engl J Med 378:708–718.
23. Campbell BC V, van Zwam WH, Goyal M, et al (2018) 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 17:47–53.
24. Butcher KS, Jeerakathil T, Hill M, et al (2013) The intracerebral hemorrhage acutely decreasing arterial pressure trial. Stroke 44:620–626.
25. Anderson CS, Heeley E, Huang Y, et al (2013) Rapid Blood-Pressure Lowering in Patients with Acute Intracerebral Hemorrhage. N Engl J Med 368:2355–2365.
26. Qureshi AI, Palesch YY, Barsan WG, et al (2016) Intensive Blood-Pressure Lowering in Patients with Acute Cerebral Hemorrhage. N Engl J Med 375:1033-1043.

Table 1: 10 false beliefs in neurocritical care.

False belief	New concept
Only neuro-intensivists should care about the brain.	All intensive care integrates neuro-intensive care.
Clinical examination of neuro-critically ill patients is impossible.	Clinical assessment of neuro-critically ill patients is more reliable than any neuromonitor.
We should no longer monitor the intracranial pressure in traumatic brain injury.	Don't confuse the monitor for the treatment.
The threshold to treat the intracranial pressure is 20 or 22 mmHg.	There is no universal ICP threshold for all patients. ICP should be interpreted together with clinical signs, imaging, and other multimodality monitors. Aggressive and potentially harmful therapeutic measures should be reserved for sustained ICP elevations above 25-30 mmHg unresponsive to lower-tier therapy.
Ketamine increases the ICP	In mechanically ventilated patients receiving other sedatives, ketamine can reduce ICP, provide neuroprotection, control seizures and reduce cortical spreading depression.
Subarachnoid hemorrhage patients should get 'Triple H' therapy.	Haemodilution is not recommended, and euvolemia should be targeted initially. A clinical picture of delayed cerebral ischemia (DCI) should promote blood pressure augmentation titrated to neurology. Remember that not all neurological deterioration in SAH is DCI.
There is no need to control the temperature after cardiac arrest.	Pre-hospital cooling is not beneficial. Strict normothermia (~36°C) or hypothermia (~33°C) are equally beneficial; the former has fewer side effects.
Hypoglycemia is harmful for the brain, hyperglycemia is not.	Both hypo-and hyperglycemia are associated with worse clinical outcomes.
In acute ischemic stroke, revascularization should be done within 3 hours of symptom onset.	In selected patients, the window for IV thrombolytic therapy can be extended to 4,5 hours. Thrombectomy can be beneficial up to 16 h in some patients (selected with advanced imaging).

Blood pressure control in intracerebral hemorrhage (ICH): contradictory trials.	In ICH, early blood pressure control is feasible, and reduces the rate of hematoma expansion, but does not improve outcome. There is no additional benefit for blood pressure reduction in the 110-139 mmHg range, compared with goals of 140-179.

Ten false beliefs in neuro-critical care

Geert Meyfroidt*
Department of Intensive Care Medicine, University Hospitals Leuven, Belgium
Associate Professor, Faculty of Medicine, KU Leuven, Belgium
geert.meyfroidt@uzleuven.be

David Menon
Head, Division of Anaesthesia, University of Cambridge
Honorary Consultant, Neurosciences Critical Care Unit, Addenbrooke's Hospital
Professorial Fellow, Queens' College, Cambridge
Emeritus Senior Investigator, National Institute for Health Research, UK
Chair, European Brain Injury Consortium
dkm13@cam.ac.uk

Alexis F. Turgeon
Associate Professor and Director of Research, Division of Critical Care Medicine, Department of Anesthesiology and Critical Care Medicine, and CHU de Québec – Université Laval Research Center, Université Laval, Québec City, Québec, Canada
Canada Research Chair in Critical Care Neurology and Trauma
Alexis.Turgeon@fmed.ulaval.ca

*Corresponding author

Search terms:
Traumatic brain injury – targeted temperature management – acute ischemic stroke – subarachnoid hemorrhage - intracerebral hemorrhage – intensive care unit

Word count: 1000

1. Only neuro-intensivists should care about the brain.

In acute brain injury, the need for specific expertise on central nervous pathophysiology is evident. However, even when the primary reason for ICU admission is extracranial, the brain may be affected too, through inadequate substrate and oxygen delivery, blood brain barrier leak, harmful effects of sedatives, and excitotoxicity. The resulting spectrum of brain dysfunction includes delirium, encephalopathy, coma, and non-convulsive seizures. Therefore, all intensive care should integrate neuro-intensive care, with the primary goal to preserve the brain [1].

2. Clinical examination of neuro-critically ill patients is impossible.

The patient's clinical state is our most important neuro-monitor. Clinical assessment of consciousness, cognition, brainstem, and motor function, should be attempted at least upon admission and daily [2]. Sedatives confound neurological examinations, and should be used sparingly in severe brain injuries, except for specific indications, such as intracranial pressure (ICP) control, seizure treatment, or targeted temperature management (TTM). They should be titrated and stopped if no longer indicated.

3. We should no longer monitor ICP in traumatic brain injury (TBI).

The ICP monitor has been accused of increasing therapeutic intensity, potentially harming patients without improving their outcomes. The Best-TRIP trial [3] is often wrongfully interpreted as evidence against ICP monitoring – a view that inappropriately conflates monitoring use and therapy titration. The controversy is not whether to monitor or treat ICP, but how this signal should be interpreted and responded to.

4. The threshold to treat ICP is 20 or 22 mmHg.

Changing ICP treatment thresholds from 20 to 22 mmHg in influential guidelines [4] imply that an ICP of 21 mmHg is fine, whereas an ICP of 23 mmHg should be treated aggressively. The absurdity of such strategy is obvious, because it neglects measurement errors, and ignores modern concepts such as the ICP intensity-time burden [5]. Interventional studies where aggressive treatments, decompressive craniectomy [6] or hypothermia [7], were applied early after crossing the 20 mmHg threshold, showed harm rather than benefit. A tiered approach is rational, and aggressive measures should probably be reserved for sustained ICP elevations above 25-30 mmHg unresponsive to lower-tier therapy.

5. Ketamine increases the ICP.

Ketamine-induced ICP elevations were reported in small studies in non-ventilated (and not acutely brain-injured) patients [8]. In fact, ketamine, as an adjunct in sedated mechanically ventilated patients, might decrease the ICP [9, 10], produce neuroprotection through NMDA-receptor antagonism [11], suppress harmful cortical spreading depolarisations [12], and control refractory seizures [13].

6. Subarachnoid hemorrhage (SAH) patients should get Triple H therapy.

Aggressive fluid loading (with consequent haemodilution), coupled with vasopressor administration to increase arterial blood pressure (ABP), was used in the past in the hope of preventing delayed cerebral ischemia (DCI) and vasospasm. This strategy is discredited, and might be deleterious in this population, where the median age is 50-60 years, and cardiopulmonary complications are common. Current recommendations [14] advise against haemodilution, and to aim for normovolemia. Clinical DCI should prompt a stepwise trial of ABP augmentation, titrated to neurological assessment. Where diagnosis or response to therapy are uncertain, additional investigations can help confirm or refute the diagnosis of DCI - not all late neurological deterioration in SAH is due to vasospasm.

7. There is no need to control the temperature after cardiac arrest (CA).

The evidence for TTM at 32-34°C after out-of-hospital CA, is less robust than initially assumed [15]. No difference in outcome was found between TTM at 33°C or at 36°C [16]. However, these results do not justify neglecting temperature control after CA. Targeting 32-34°C may still be defensible because of equipoise between targets, but many sites involved in the TTM trial have adopted the 36°C target [17], since it avoids potential adverse events of more aggressive cooling.

8. Hypoglycemia is harmful for the brain, hyperglycemia is not.

The optimal glycemic target for the injured brain is controversial. Tight blood glucose control to the normal fasting range (TGC) increases the risk of (deep) hypoglycemia, especially in inexperienced hands. In small observational studies, the (lower) normoglycemic range has been associated with low cerebral microdialysis glucose values [18], raising concerns about substrate delivery. On the other hand, hyperglycemia is an independent predictor of poor neurological outcome and death after CA [19]. According to a recent meta-analysis, the use of TGC showed a small but significant reduction in the risk of poor neurological outcomes in TBI [20]. So, even when the discussion on the optimal glycemic target is far from resolved, it is important not to ignore that both hypo-and hyperglycemia are associated with worse clinical outcomes in neuro-critically ill patients.

9. In acute ischemic stroke (AIS), revascularization should be done within 3 hours of symptom onset.

The conventional time window for thrombolysis in AIS is 3 hours, extendable to 4.5 hours in patients ≤80 y of age, without a history of both diabetes mellitus and prior stroke combined, a National Institutes of Health Stroke Scale (NIHSS) score of ≤25, not taking any oral anticoagulants, and without imaging evidence of ischemic injury involving more than 1/3 of middle cerebral artery territory [21]. When mechanical thrombectomy is considered, the recommended timeframe is 6 hours post-ictus, but specific penumbra-like conditions on perfusion imaging allow for longer time-windows up to 16 hours [22]. General anesthesia during thrombectomy should be avoided [23].

10. Blood pressure control in intracerebral hemorrhage (ICH): contradictory trials.

Interpreting recent trials [24–26] on the treatment of hypertension after ICH is complicated by differences in inclusion criteria, intervention timing, outcomes, antihypertensive drugs, and systolic blood pressure (SBP) targets. All these studies were conducted in patients with relatively small ICH volumes, with varying latency to achieving target SBP (ranging from 4.5 to 24h). Early intensive SBP control to targets above 140 mmHg reduces hematoma expansion, but does not improve neurological outcome or mortality. More aggressive SBP reduction to 110-140 mmHg in the ATACH-2 study [26] found no incremental benefit as compared to the 140-180 mmHg range, but a higher rate of renal complications. In brief, SBP control in ICH may reduce hematoma expansion, but not below 140 mmHg.

Conflict of interest

On behalf of all authors, the corresponding author states that there is no conflict of interest.

References

1. Tasker RC, Menon DK (2016) Critical Care and the Brain. JAMA 315:749.
2. Sharshar T, Citerio G, Andrews PJD, et al (2014) Neurological examination of critically ill patients: A pragmatic approach. Report of an ESICM expert panel. Intensive Care

Med 40:484–495.
3. Chesnut RM, Temkin N, Carney N, et al (2012) A Trial of Intracranial-Pressure Monitoring in Traumatic Brain Injury. N Engl J Med 367:2471–2481.
4. Carney N, Totten AM, O'Reilly C, et al (2017) Guidelines for the Management of Severe Traumatic Brain Injury, Fourth Edition. Neurosurgery 80:6–15.
5. Güiza F, Depreitere B, Piper I, et al (2015) Visualizing the pressure and time burden of intracranial hypertension in adult and paediatric traumatic brain injury. Intensive Care Med 41:1067–1076.
6. Cooper JD, Rosenfeld J V., Murray L, et al (2011) Decompressive Craniectomy in Diffuse Traumatic Brain Injury. New Engl J Med 364:1493–1502.
7. Andrews PJD, Sinclair HL, Rodriguez A, et al (2015) Hypothermia for Intracranial Hypertension after Traumatic Brain Injury. N Engl J Med 373:2403-2412
8. Gardner AE, Olson BE, Lichtiger M (1971) Cerebrospinal-fluid Pressure during Dissociative Anesthesia with Ketamine. Anesthesiology 35:226–228.
9. Zeiler FA, Teitelbaum J, West M, Gillman LM (2014) The ketamine effect on ICP in traumatic brain injury. Neurocrit Care 21:163–73.
10. Zeiler FA, Teitelbaum J, West M, Gillman LM (2014) The ketamine effect on intracranial pressure in nontraumatic neurological illness. J Crit Care 29:1096–1106.
11. Sakai T, Ichiyama T, Whitten CW, et al (2000) Ketamine suppresses endotoxin-induced NF-kappaB expression. Can J Anaesth 47:1019–24.
12. Hertle DN, Dreier JP, Woitzik J, et al (2012) Effect of analgesics and sedatives on the occurrence of spreading depolarizations accompanying acute brain injury. Brain 135:2390–2398.
13. Gaspard N, Foreman B, Judd LM, et al (2013) Intravenous ketamine for the treatment of refractory status epilepticus: a retrospective multicenter study. Epilepsia 54:1498–503.
14. Diringer MN, Bleck TP, Claude Hemphill J, et al (2011) Critical Care Management of Patients Following Aneurysmal Subarachnoid Hemorrhage: Recommendations from the Neurocritical Care Society's Multidisciplinary Consensus Conference. Neurocrit Care 15:211–240.
15. Nielsen N, Friberg H, Gluud C, et al (2011) Hypothermia after cardiac arrest should be further evaluated—A systematic review of randomised trials with meta-analysis and trial sequential analysis. Int J Cardiol 151:333–341.
16. Nielsen N, Wetterslev J, Cronberg T, et al (2013) Targeted Temperature Management at 33°C versus 36°C after Cardiac Arrest. N Engl J Med 369:2197–2206.
17. Nielsen N, Friberg H (2015) Temperature management after cardiac arrest. Curr Opin Crit Care 21:202–208.
18. Oddo M, Schmidt JM, Carrera E, et al (2008) Impact of tight glycemic control on cerebral glucose metabolism after severe brain injury: A microdialysis study*. Crit Care Med 36(12):3233-8.
19. Borgquist O, Wise MP, Nielsen N, et al (2017) Dysglycemia, glycemic variability, and outcome after cardiac arrest and temperature management at 33°C and 36°C. Crit Care Med 45:1337–1343.
20. Hermanides J, Plummer MP, Finnis M, et al (2018) Glycaemic control targets after traumatic brain injury: a systematic review and meta-analysis. Crit Care 22:11.
21. Powers WJ, Rabinstein AA, Ackerson T, et al (2018) 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 (Epub ahead of print).
22. Albers GW, Marks MP, Kemp S, et al (2018) Thrombectomy for Stroke at 6 to 16 Hours with Selection by Perfusion Imaging. N Engl J Med 378:708–718.
23. Campbell BC V, van Zwam WH, Goyal M, et al (2018) 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 17:47–53.
24. Butcher KS, Jeerakathil T, Hill M, et al (2013) The intracerebral hemorrhage acutely decreasing arterial pressure trial. Stroke 44:620–626.
25. Anderson CS, Heeley E, Huang Y, et al (2013) Rapid Blood-Pressure Lowering in Patients with Acute Intracerebral Hemorrhage. N Engl J Med 368:2355–2365.
26. Qureshi AI, Palesch YY, Barsan WG, et al (2016) Intensive Blood-Pressure Lowering in Patients with Acute Cerebral Hemorrhage. N Engl J Med 375:1033-1043.

Table 1: 10 false beliefs in neurocritical care.

False belief	New concept
Only neuro-intensivists should care about the brain.	All intensive care integrates neuro-intensive care.
Clinical examination of neuro-critically ill patients is impossible.	Clinical assessment of neuro-critically ill patients is more reliable than any neuromonitor.
We should no longer monitor the intracranial pressure in traumatic brain injury.	Don't confuse the monitor for the treatment.
The threshold to treat the intracranial pressure is 20 or 22 mmHg.	There is no universal ICP threshold for all patients. ICP should be interpreted together with clinical signs, imaging, and other multimodality monitors. Aggressive and potentially harmful therapeutic measures should be reserved for sustained ICP elevations above 25-30 mmHg unresponsive to lower-tier therapy.
Ketamine increases the ICP	In mechanically ventilated patients receiving other sedatives, ketamine can reduce ICP, provide neuroprotection, control seizures and reduce cortical spreading depression.
Subarachnoid hemorrhage patients should get 'Triple H' therapy.	Haemodilution is not recommended, and euvolemia should be targeted initially. A clinical picture of delayed cerebral ischemia (DCI) should promote blood pressure augmentation titrated to neurology. Remember that not all neurological deterioration in SAH is DCI.
There is no need to control the temperature after cardiac arrest.	Pre-hospital cooling is not beneficial. Strict normothermia (~36°C) or hypothermia (~33°C) are equally beneficial; the former has fewer side effects.
Hypoglycemia is harmful for the brain, hyperglycemia is not.	Both hypo-and hyperglycemia are associated with worse clinical outcomes.
In acute ischemic stroke, revascularization should be done within 3 hours of symptom onset.	In selected patients, the window for IV thrombolytic therapy can be extended to 4,5 hours. Thrombectomy can be beneficial up to 16 h in some patients (selected with advanced imaging).

Blood pressure control in intracerebral hemorrhage (ICH): contradictory trials.	In ICH, early blood pressure control is feasible, and reduces the rate of hematoma expansion, but does not improve outcome. There is no additional benefit for blood pressure reduction in the 110-139 mmHg range, compared with goals of 140-179.

Table 1: 10 false beliefs in neurocritical care.

False belief	New concept
Only neuro-intensivists should care about the brain.	All intensive care integrates neuro-intensive care.
Clinical examination of neuro-critically ill patients is impossible.	Clinical assessment of neuro-critically ill patients is more reliable than any neuromonitor.
We should no longer monitor the intracranial pressure in traumatic brain injury.	Don't confuse the monitor for the treatment.
The threshold to treat the intracranial pressure is 20 or 22 mmHg.	There is no universal ICP threshold for all patients. ICP should be interpreted together with clinical signs, imaging, and other multimodality monitors. Aggressive and potentially harmful therapeutic measures should be reserved for sustained ICP elevations above 25-30 mmHg unresponsive to lower-tier therapy.
Ketamine increases the ICP	In mechanically ventilated patients receiving other sedatives, ketamine can reduce ICP, provide neuroprotection, control seizures and reduce cortical spreading depression.
Subarachnoid hemorrhage patients should get 'Triple H' therapy.	Haemodilution is not recommended, and euvolemia should be targeted initially. A clinical picture of delayed cerebral ischemia (DCI) should promote blood pressure augmentation titrated to neurology. Remember that not all neurological deterioration in SAH is DCI.
There is no need to control the temperature after cardiac arrest.	Pre-hospital cooling is not beneficial. Strict normothermia (~36°C) or hypothermia (~33°C) are equally beneficial; the former has fewer side effects.
Hypoglycemia is harmful for the brain, hyperglycemia is not.	Both hypo-and hyperglycemia are associated with worse clinical outcomes.
In acute ischemic stroke, revascularization should be done within 3 hours of symptom onset.	In selected patients, the window for IV thrombolytic therapy can be extended to 4,5 hours. Thrombectomy can be beneficial up to 16 h in some patients (selected with advanced imaging).

Blood pressure control in intracerebral hemorrhage (ICH): contradictory trials.	In ICH, early blood pressure control is feasible, and reduces the rate of hematoma expansion, but does not improve outcome. There is no additional benefit for blood pressure reduction in the 110-139 mmHg range, compared with goals of 140-179.

Author replies with annotations