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When “Normal” Is Too Low: Iatrogenic Cerebral Hypoperfusion, Delirium Misattribution, and Sedation-Masked Examination in Acute Ischemic Stroke With Chronic Uncontrolled Hypertension

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Abstract

Blood pressure management after acute ischemic stroke is among the highest-stakes and most variable tasks in neurocritical care, and the appropriate target depends on what treatment the patient received. Recommended pressures differ for patients receiving thrombolysis, those undergoing thrombectomy, and those who get neither. In practice, general ICU order sets and an ingrained instinct to correct a high number can push toward more aggressive reduction than any of those targets intend. The risk is largest in patients with chronic, poorly controlled hypertension, whose cerebral autoregulatory range is shifted rightward. For them, a pressure that looks normal can sit below their working perfusion floor.

Using an illustrative composite scenario, this review traces a plausible cascade: rapid pressure reduction in a chronically hypertensive post-stroke patient, attribution of the resulting confusion to ICU delirium, and a dexmedetomidine infusion that may further reduce perfusion while sedating away the examination that would reveal deterioration.

Written from the vantage point of a former critical care nurse now in medical training, the piece reviews autoregulation in chronic hypertension, organizes the evidence by reperfusion status, examines the limits of delirium screening in stroke, and addresses the order-set, handoff, and education failures behind it. Recent randomized evidence suggests that hemodynamic management chosen for the individual patient may outperform a single universal target. The recommendations are low-cost and need no new validated target.

Categories: Neurology, Quality Improvement, Anesthesiology
Keywords: cerebral autoregulation, delirium, dexmedetomidine, endovascular thrombectomy, icu, interdisciplinary communication, ischemic stroke, permissive hypertension

Introduction And Background

An acute hypertensive response is present in most patients arriving with acute ischemic stroke, and what we do with that pressure in the first hours matters [1]. Cerebral autoregulation is the brain's ability to maintain its own blood flow as systemic pressure rises and falls, and the ischemic penumbra is the rim of underperfused but still viable tissue surrounding the infarct core. The rationale for permissive hypertension is well established. In the ischemic penumbra, autoregulation is disrupted, and cerebral blood flow becomes pressure-dependent, so dropping systemic pressure can extend injury into tissue that was still salvageable [2,3]. The risk is sharpest in patients with long-standing, poorly controlled hypertension, whose whole autoregulatory range shifts toward higher pressures, so a reading that is acceptable for most ICU patients can be too low for them.

The guidelines reflect the penumbral rationale and are more specific than the phrase “permissive hypertension” suggests. What counts as an acceptable pressure depends on whether the patient received intravenous thrombolysis (IVT, a clot-dissolving drug given within a set time window), underwent endovascular thrombectomy (EVT, removal of the clot by catheter), or received neither, and these three possibilities differ enough that a single order set cannot cover all of them [4,5]. The gap this review is concerned with sits downstream of the guideline, in how that intent travels from the order to the bedside. Order sets carry general ICU targets. A verbal “we’re tolerating higher pressures” carries none of the force of a documented parameter. And nurses are trained to treat a high number as a problem to fix.

This piece comes from a perspective that is uncommon in the literature: someone who spent years titrating drips at the ICU bedside as a registered nurse before entering medical school. The aim is to examine the system that connects known physiology to a 3 a.m. decision and to suggest where that system can be made safer. Specifically, this review summarizes how chronic hypertension shifts cerebral autoregulation, organizes the guideline and trial evidence by reperfusion status, examines why hypoperfusion after stroke can be mistaken for delirium and how dexmedetomidine may compound that error, and proposes low-cost

changes to order sets, handoff communication, and nursing education. No clinician or institution is being indicted here.

Review

Review type and literature selection

This is a narrative review, prepared with reference to the Scale for the Assessment of Narrative Review Articles (SANRA) [6]. Literature selection was conducted via PubMed, searched from inception through September 2026, using combinations of the terms: cerebral autoregulation, permissive hypertension, acute ischemic stroke, blood pressure management, endovascular thrombectomy, dexmedetomidine, and intensive care unit delirium. For transparency, the search was formalized during revision into five strings and rerun on October 24, 2026, limited to records published through September 30, 2026: 1. Physiology (3,518 records): autoregulation[tiab] AND (cerebral[tiab] OR brain[tiab] OR cerebrovascular[tiab]) AND (hypertension[tiab] OR hypertensive[tiab] OR "blood pressure"[tiab] OR ischemia[tiab] OR ischemic[tiab]); 2. Blood pressure trials (1,062 records): ("blood pressure"[tiab] OR antihypertensive[tiab]) AND (stroke[tiab] OR "Ischemic Stroke"[mh]) AND (acute[tiab] OR hyperacute[tiab]) AND (randomi*[tiab] OR "meta-analysis"[pt] OR "systematic review"[tiab]); 3. Dexmedetomidine (2,708 records): dexmedetomidine[tiab] AND (stroke[tiab] OR neurocritical[tiab] OR cerebral[tiab] OR "intensive care"[tiab] OR "critically ill"[tiab] OR delirium[tiab]); 4. Delirium (1,195 records): delirium[tiab] AND (stroke[tiab] OR autoregulation[tiab]); 5. Reviews and guidelines (2,700 records): ((stroke[ti] OR "ischemic cerebrovascular"[tiab] OR "cerebral ischemia"[tiab]) AND ("blood pressure"[tiab] OR hypertension[tiab] OR hypertensive[tiab] OR hemodynamics[tiab] OR "cerebral circulation"[tiab]) AND (review[pt] OR guideline[pt] OR "practice guideline"[pt])) OR ("ischemic stroke"[ti] AND "early management"[ti]).

Priority was given to current clinical practice guidelines, randomized controlled trials, and systematic or narrative reviews published in English, as well as observational, physiologic, and experimental studies that addressed one of the review's aims, including older foundational work. Pediatric studies, case reports, conference abstracts without a full report, and studies limited to intracerebral or subarachnoid hemorrhage (except those informing autoregulation monitoring) were excluded. Reference lists of retrieved articles were hand-searched for additional sources. Together, the five strings retrieved 35 of the 36 cited sources; the remaining one is the SANRA scale, cited as the reporting standard. Evidence was synthesized qualitatively and organized by reperfusion status. Selection was purposive rather than systematic; records were not screened exhaustively, so screening counts are not reported, and no formal protocol or risk-of-bias assessment was applied. The review is therefore subject to selection bias.

An illustrative scenario

What follows is a brief illustrative device rather than the evidence base for this review. It is a composite scenario, does not describe a specific patient, and contains no protected health information. The argument rests on the published literature.

A middle-aged patient with long-standing, poorly controlled hypertension (home pressures running in the 180 to 200 mmHg systolic range, with known medication non-adherence) presents with acute neurologic deficits consistent with ischemic stroke. The patient is outside the thrombolysis window and has no large-vessel occlusion amenable to thrombectomy, so no reperfusion therapy is given, and the patient is admitted to the ICU for monitoring.

A nicardipine infusion is started per protocol and titrated toward a systolic target that would be unremarkable in a general ICU population, well below this patient’s chronic baseline, and well below the threshold at which guidelines would call for any treatment at all in a patient who received no reperfusion therapy.

Over the next several hours, the patient becomes agitated and confused. The team reads this as ICU delirium, a reasonable first thought given how common delirium is in this setting. What the delirium label leaves out is the timing. The confusion follows the reduction in pressure in a brain adapted to chronically high perfusion pressures.

A dexmedetomidine infusion is started for the presumed delirium. As a highly selective alpha-2 agonist, dexmedetomidine reduces sympathetic outflow, lowers systemic vascular resistance, and can drop mean arterial pressure (MAP) and heart rate. It can have two effects here: it can nudge an already marginal perfusion pressure lower, and its sedative action flattens the very neurologic exam the team relies on to catch deterioration.

The patient continues to decline. When the infusion is paused for a full examination the next morning, the patient has a dense hemiparesis and aphasia that were not present on admission, and repeat imaging shows that the infarct has extended into tissue that had been at risk.

Cerebral autoregulation and the rightward shift in chronic hypertension

Autoregulation keeps cerebral blood flow roughly constant across a range of perfusion pressures. In a normotensive person, that plateau classically spans a mean arterial pressure of about 60 to 150 mmHg, though the lower limit varies between individuals more than the textbook curve suggests [7]. Below it, flow tracks pressure, and ischemia becomes possible. The classic plateau is itself a simplification. A review and reanalysis of 143 studies in healthy adults put the true static plateau closer to 80-100 mmHg and found the pressure-flow relationship to be asymmetric: cerebral blood flow was roughly four times more sensitive to a fall in pressure than to a rise (1.47 versus 0.37 percent per percent) [8]. The brain adapts better to a higher pressure than a falling one, so when pressure is low, the brain is least protected.

Chronic, uncontrolled hypertension shifts both limits of that range to the right [9]. The driver is structural: arteriolar wall thickening, narrower lumens, and higher cerebrovascular resistance [10]. Strandgaard's measurements in hypertensive patients placed the mean lower limit near a MAP of 113 mmHg, compared with roughly 73 mmHg in normotensive controls [9]. Those are group means from a small 1976 series, and they describe a physiologic shift rather than a target, a distinction taken up below. More recent physiological reviews support the direction of the shift while emphasizing how much the lower limit varies among individuals [7,10]. The evidence points in one direction: these patients need a higher pressure to perfuse the brain.

Guideline thresholds are written in systolic terms, while autoregulatory limits are measured as MAP, so the two are worth putting side by side. A pressure of 140/80 mmHg (a MAP of about 100), a number we would call well controlled in most patients, can fall under the autoregulatory floor of someone whose vasculature has remodeled around systolic pressures of 180 to 200 (about 180/100 to 200/110 mmHg, a MAP of roughly 127 to 140). In that patient, bringing the number to normal can induce hypoperfusion (Figure *1*). Acute ischemia compounds it, because autoregulation is already lost in the penumbra, where flow depends directly on systemic pressure [2,3]. A rightward-shifted curve coupled with focal autoregulatory failure leaves the brain unusually sensitive to any drop in pressure.

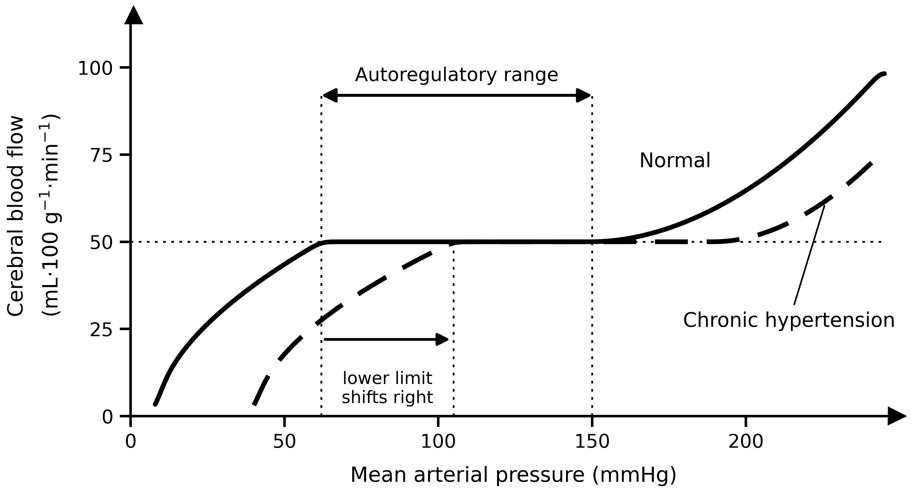


FIGURE 1: Cerebral autoregulation in the normotensive brain (solid line) and in chronic hypertension (dashed line).

Cerebral blood flow is held near 50 mL·100 g-1·min-1 across the autoregulatory range. In chronic hypertension, the curve is displaced to the right, so the lower limit of autoregulation occurs at a higher mean arterial pressure. A pressure that falls comfortably inside the normotensive range can therefore lie below the adapted brain's autoregulatory floor, where flow becomes pressure-passive. Values are illustrative and are not treatment targets. Original figure, created by the authors from published descriptions of the autoregulatory curve and its rightward displacement in chronic hypertension [7-10].

This concept tracks with the clinical data. How far pressure can fall before symptoms appear scales with where it has been running. In Strandgaard's series, patients whose hypertension had been effectively treated tolerated lower pressures before symptoms of brain hypoperfusion appeared than those whose hypertension was untreated or poorly controlled [9].

What the guidelines actually say depends on reperfusion status

Permissive hypertension is often discussed as though it were a single policy. The recommended pressure depends on what the patient received [4,5]. Setting the three situations side by side is worth doing, because it shows how far a general ICU target can sit from any of them (Table *1*).

	Intravenous thrombolysis	Endovascular thrombectomy	No reperfusion therapy
Guideline threshold	< 185/110 mmHg before bolus; < 180/105 mmHg for 24 h [4,5]	≤ 180/105 mmHg during and for 24 h after [4,5]	Lowering below 220/120 mmHg not effective in the first 48 to 72 h; about 15% reduction above it [4,5]
Nature of the number	Ceiling (hemorrhage risk)	Ceiling	Ceiling for starting treatment
Trials of lower targets	ENCHANTED [11]	BP-TARGET, BEST-II, ENCHANTED2/MT, OPTIMAL-BP [12-15]	CATIS, SCAST, ENOS, COSSACS, CHHIPS[17-21]
What lower targets did	Less hemorrhage; no better function	No benefit; worse function in two trials and the pooled analysis [16]	No reduction in death or dependency
2026 position on SBP < 140 mmHg	Not recommended (no benefit) [5]	Harmful after successful recanalization [5]	No lower bound stated [5]

TABLE 1: Blood pressure thresholds and trial evidence by reperfusion status.

SBP: systolic blood pressure.

Intravenous Thrombolysis

Blood pressure must be brought below 185/110 mmHg before the bolus and kept below 180/105 mmHg for the first 24 hours afterward, with the corresponding monitoring schedule [4,5]. That number is a ceiling set by hemorrhagic risk, not a floor, and it does not greenlight lowering the pressure any further. ENCHANTED tested that question directly and found that an intensive early target of 130 to 140 mmHg systolic failed to improve functional outcome (disability at 90 days on the modified Rankin Scale (mRS), which runs from 0, no symptoms, to 6, death) compared with the guideline target of below 180 mmHg, even though it reduced intracranial hemorrhage [11]. The 2026 American Heart Association/American Stroke Association (AHA/ASA) guideline now states that in patients with mild-to-moderate stroke, intensive lowering of systolic blood pressure (SBP) to below 140 mmHg is not recommended after thrombolysis (Class 3: No Benefit) [5].

Endovascular Thrombectomy

Guidelines recommend maintaining blood pressure at or below 180/105 mmHg during the procedure and for 24 hours afterward [4,5]. The stronger evidence here concerns the floor rather than the ceiling. Two trials found no benefit from lowering: BP-TARGET (systolic 100 to 129 versus 130 to 185 mmHg) did not reduce hemorrhage or improve function, and BEST-II, a small phase 2 trial, estimated a low likelihood that targets below 140 or 160 mmHg would help in a larger trial [12,13]. ENCHANTED2/MT was stopped early when a systolic target below 120 mmHg produced worse recovery (common OR for a worse mRS score 1.37, 95% CI 1.07 to 1.76; death or disability, mRS 3 to 6, 53% versus 39%) and more early neurologic deterioration (common OR 1.53, 95% CI 1.18 to 1.97) [14]. OPTIMAL-BP, also stopped early, found lower functional independence (mRS 0 to 2, meaning the patient can manage daily affairs without help) with a target below 140 mmHg: 39.4% versus 54.4% (adjusted OR 0.56, 95% CI 0.33 to 0.96) [15]. A pooled analysis of four post-thrombectomy trials found that intensive lowering cut the odds of functional independence (OR 0.68, 95% CI 0.51 to 0.91) without reducing hemorrhage [16]. The 2026 guideline reflects this, stating that intensive lowering of systolic pressure to below 140 mmHg is not recommended, even after complete reperfusion, and classifying it as harmful after successful recanalization (Class 3: Harm) [5].

No Reperfusion Therapy

For patients who receive neither thrombolysis nor thrombectomy and have no other indication for acute pressure lowering, permissive hypertension is acceptable up to 220/120 mmHg. Above that threshold, a reduction of about 15% during the first 24 hours may be reasonable. Below it, initiating or reinitiating antihypertensive treatment within the first 48 to 72 hours has not been shown to prevent death or dependency [4,5]. This is the situation in the scenario above, where a general ICU order set has the most potential to cause harm, because the pressure ranges the guideline sanctions and those of a standard ICU order set barely overlap.

The trials behind that recommendation point the same way. CATIS, the largest, randomized 4,071 patients with ischemic stroke who had not received thrombolysis to early lowering or stopping antihypertensives and found no difference in death or major disability [17]. SCAST (candesartan), ENOS (glyceryl trinitrate, and continuing versus stopping home agents), and COSSACS (continuing versus stopping, underpowered after early termination) also found no benefit; these trials enrolled mixed stroke types, mostly ischemic [18-20]. In SCAST, functional outcome trended worse with treatment (common OR 1.17, 95% CI 1.00 to 1.38, not significant at the trial's threshold) [18]. One small pilot study, CHHIPS, found lower 3-month mortality with treatment (9.7% versus 20.3%, of borderline significance) but was too small to assess efficacy [21]. On average, lowering pressure in these patients has not helped, and no trial was designed to find the patients it harms. Within its blood pressure section, the 2026 guideline also warns that excessive lowering "can lead to exacerbation of cerebral ischemia in the setting of impaired cerebral autoregulation" [5].

One feature is common to all three interventions: every listed number is a ceiling. The only lower bound is the statement against intensive targets below 140 mmHg after reperfusion therapy; for patients who receive no reperfusion therapy, there is none. Each threshold carries an unstated assumption that pressure will not be driven far below it. The assumption is where these patients get lost.

What the recent trials add

Recent randomized trials sharpen the caution, and the most recent one points past it. In INTERACT4, lowering pressure in the ambulance was associated with worse outcomes in the ischemic stroke subgroup (common OR for a worse mRS score 1.30, 95% CI 1.06 to 1.60), a subgroup finding outside the trial's prespecified analysis plan that its investigators cautioned against drawing conclusions from [22]. Together with the post-thrombectomy trials above, it suggests that aggressive lowering can hurt, but none of these trials says what to do instead.

The HOPE trial is the first to test the alternative head-on. Four hundred and forty patients at eleven Spanish centers with successful recanalization were randomized to a reperfusion-guided systolic target or to guideline-standard management targeting below 180 mmHg [23,24]. Reperfusion was graded by the modified Thrombolysis in Cerebral Infarction (mTICI) scale, which scores how fully the territory reperfused, with 2b meaning at least half and 2c or 3 near-complete or complete. The tailored target was 140 to 160 mmHg for mTICI 2b and 100 to 140 mmHg for mTICI 2c or 3. Functional independence at 90 days was 60.0% with the tailored strategy against 47.1% with the uniform one (adjusted OR 1.71, 95% CI 1.11 to 2.63, where an OR above 1 favors the tailored strategy). Hemorrhagic transformation was less frequent (22.3% versus 31.6%; OR 0.62, 95% CI 0.41 to 0.95), with no excess mortality [24].

At first glance, HOPE appears to argue for lower pressure because its tailored arm reduced pressure more than standard care, especially after complete reperfusion. Read by reperfusion grade, it fits the physiology above. When the vessel was fully reopened, blood flow to the threatened tissue was restored, and HOPE lowered the target. When reopening was incomplete, part of the tissue still depended on pressure, and HOPE maintained systolic pressure at 140-160 mmHg, using vasopressors as needed [24]. The graded strategy as a whole improved recovery. The patient in the scenario was never reperfused, so the whole penumbra still depends on pressure. HOPE's 100 to 140 mmHg arm also falls in the range the 2026 guideline classifies as harmful after successful recanalization [5]. The guideline, published in January 2026, predates HOPE's results and is based on trials that applied a single fixed target to everyone [14-16]. HOPE is a single open-label trial, and it does not overturn the guideline; this review does not recommend its targets.

HOPE focuses on angiographic reperfusion rather than autoregulatory physiology, and it applies only after thrombectomy, so it does nothing to validate a bedside method for identifying a chronically hypertensive patient's floor [23,24]. What it does suggest is that the choice between one number for everyone and a number chosen for the patient in front of you is a real clinical variable with a measurable effect on recovery. So far, in the one trial that has tested it, the tailored version performed better.

This evidence comes from prehospital or reperfusion-treated large-vessel populations; in contrast, the scenario above involves a non-thrombolyzed patient on nicardipine, so these trials provide directional support rather than proof for that specific case [14,15,22]. The guidelines are also not the target of the critique. The AHA/ASA thresholds stand [4,5]. The concern is narrower: a population threshold cannot be individualized, so a pressure that satisfies the guideline can still sit below a particular patient's autoregulatory floor. What carries over is supported by the data: aggressive reductions have been associated with harm, and greater early variability in systolic pressure is associated with poor functional outcome [25]. Both argue for smoothly titratable or long-acting agents over sharp correction.

The limits of an individualized target

It would be convenient to close this gap by establishing a mean arterial pressure floor for these patients, based on their elevated baseline. That number does not exist in any form we can defend. Individualized targets derived from a presumed autoregulatory floor are not standard of care in acute ischemic stroke, and no guideline supports computing a MAP target from a patient's usual pressures. Autoregulation-guided management has been studied in cardiac surgery, traumatic brain injury, and subarachnoid hemorrhage

using continuous indices such as the pressure reactivity index and the cerebral oximetry index. Still, those methods require monitoring that most stroke units do not have, and they have not been validated as a treatment target in acute ischemic stroke [26].

So, the practical claim here is deliberately smaller than a target. These patients differ from the population for which the order set was written. The difference lies in the need for more, not less, pressure. The pre-treatment pressure is the best available anchor for judging how far a reduction has already gone, and any reduction should be documented and bounded by an ordered call parameter, not left to happen as a side effect of a drip running to a generic target. The call parameter in Recommendation 1 is built the same way. It reuses the guideline's own figure of about 15% [4,5] and anchors it to this patient's documented pre-treatment pressure, so crossing it triggers a bedside reassessment rather than claiming that perfusion fails at that number. Naming a floor that has not been validated would repeat the error this review describes: treating a number as though it applied to everyone.

The diagnostic pitfall: delirium versus cerebral hypoperfusion

Reading new agitation and confusion as delirium is understandable. Delirium is common after stroke, with reported prevalence around 15 to 35% on stroke units [27]. A trained nurse or physician will reasonably put it on the differential.

The trouble is that the standard screening tools, Confusion Assessment Method for the Intensive Care Unit (CAM-ICU) and Intensive Care Delirium Screening Checklist (ICDSC), were not built for patients with a primary neurologic injury. Deficits such as impaired consciousness, aphasia, and hemineglect track with positive delirium screens, which raises the possibility of false positives in stroke patients whose mental status change is neurologic in origin [28]. The Austrian Stroke Society has said as much, cautioning that delirium screening strategies drawn from non-stroke populations transfer poorly to stroke [27].

There is randomized evidence that this link runs in the direction assumed by the scenario. In a trial nested within a larger cardiac surgery study, 199 patients older than 55 undergoing nonemergency surgery had their individual lower limit of autoregulation identified before cardiopulmonary bypass; the intervention arm then maintained mean arterial pressure above that limit, while the control arm was managed according to institutional practice. Delirium occurred in 53% of the usual-care group versus 38% of the intervention group, a 45% reduction in the odds (OR 0.55, 95% CI 0.31 to 0.97) [29]. The population is cardiac surgery rather than stroke, and the exposure is bypass rather than a titrated antihypertensive, so no clear target carries over, but the underlying mechanism does. Perfusion held below an individual patient's autoregulatory limit is associated with delirium, and in that trial, maintaining pressure above the limit prevented some of it.

The question standard delirium protocols do not prompt is the one that matters here. Could this altered mental status be worsening perfusion, rather than or on top of delirium? In a patient with recent ischemic stroke, chronic hypertension, and a recent aggressive pressure drop, the timing alone should raise it before any sedative goes up. Figure *2* puts that question into a short pre-sedation check.

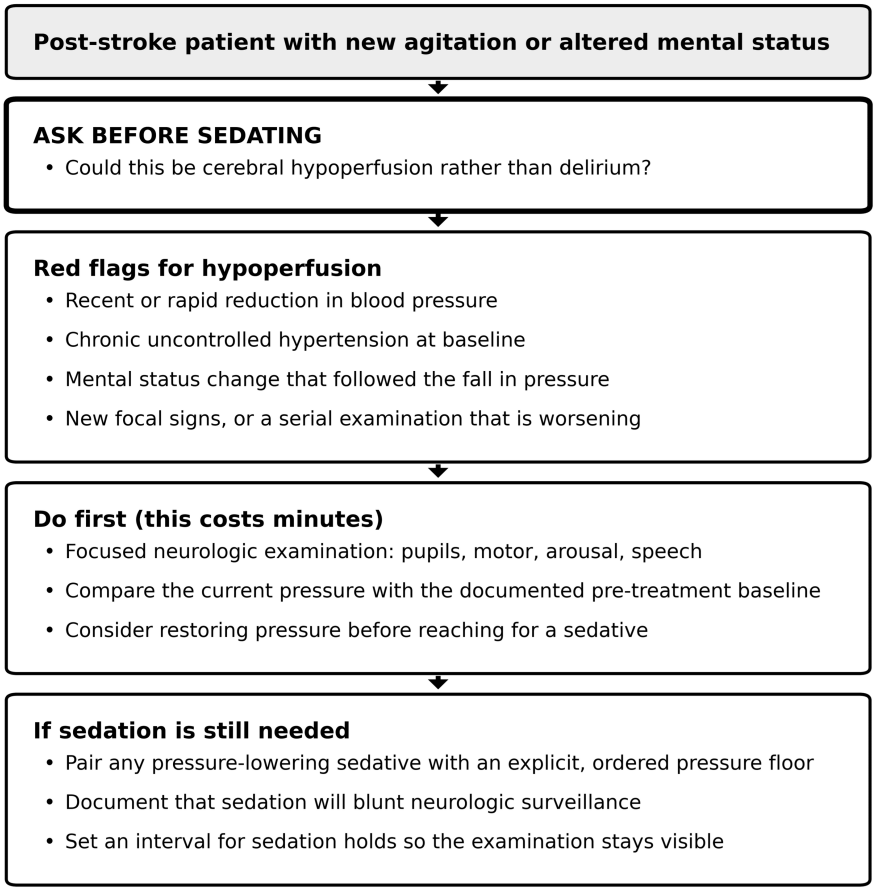


FIGURE 2: A one-page pre-sedation check.

In a post-stroke patient with new agitation, the clinician asks whether the change could be hypoperfusion before sedating, screens for red flags, performs a focused neurologic exam, compares the current pressure with the documented pre-treatment baseline, and, if sedation is still needed, pairs it with an explicit ordered call parameter and a scheduled sedation hold. Original figure, developed by the authors from the sources cited in the text [4,5,27,28,30].

The dexmedetomidine problem

Dexmedetomidine is popular in the ICU for good reasons: sedation without much respiratory depression, anxiolysis, and an opioid-sparing effect. In mechanically ventilated patients with agitated delirium, it has been linked to faster resolution of delirium [31], and it is often a first-line agent for agitation.

Its pharmacology is also what makes it risky in this particular setting. As an alpha-2 agonist, it reduces sympathetic tone and lowers systemic vascular resistance, and hypotension and bradycardia are well-documented effects [30]; both were more common with dexmedetomidine in SPICE III, the largest trial of early dexmedetomidine sedation [32]. Reviews of dexmedetomidine in neurocritical care note that its cerebral hemodynamic effects remain unclear, and the open question is whether perfusion is maintained during low-pressure episodes [33,34]. In a patient with no intracranial pathology, this may never matter. In a post-stroke patient with little autoregulatory reserve and an already low mean pressure, even a modest further drop may extend the injury.

The second effect is subtler. Sedation makes serial neuro exams unreliable. Monitoring a stroke patient depends on someone who can be roused and participate in the assessment, including assessment of pupils, motor function, level of consciousness, and speech. Sedation dulls all four, and it does so in the window where a change in any of them is worth catching. Because the evidence for cerebral hemodynamic effects is unsettled, this remains a precaution rather than an established synergy, but the concern is concrete. A sedative given for presumed delirium may lower perfusion and conceal the neurologic decline at the same time. We call this pairing perfusion-blind sedation, treating the agitation while remaining blind to the brain's perfusion state, and it runs through the broader argument for keeping the examination visible whenever pressure is in play.

Systems: the space between the order and bedside care

The system centers on three converging issues, each addressed by a recommendation below. Each is a point where what the physician intends and what the nurse is trained to do can come apart.

Order Set Design (Recommendations 1 and 5)

Standardized blood pressure parameters are written for general ICU populations. “Hold nicardipine for SBP < 120” or “titrate to SBP 140 to 160” is fine for most patients and dangerous for the chronically hypertensive stroke patient who received no reperfusion therapy. A printed range implies it applies to everyone. Overriding it takes active effort, context, and usually a call to the provider, the kind of steps that fall away on a busy night with rotating coverage.

Communication (Recommendation 1)

When a physician intends permissive hypertension, that intent has to reach the bedside nurse, the charge nurse, and the next shift. “We’re tolerating higher pressures” is not enough without a specific number tied to this patient. If a nurse’s training, experience, and unit culture all say a systolic of 180 in the ICU demands action, a vague verbal plan will usually lose to that instinct. The target belongs in an order, not a hallway conversation.

Nursing Education (Recommendation 4)

Nursing trains vital sign monitoring as a core duty, and high blood pressure is taught as a problem to be resolved. Documentation, alarms, and supervisory expectations all reinforce it. A nurse who sees a systolic pressure of 190 in a stroke patient and does nothing risks looking negligent to peers and to the team. The idea that an elevated pressure is justified and should be protected runs counter to that training, and it is rarely taught.

Put a generic order set, a vague plan, and that training together, and you get the scenario above, where each action is defensible on its own. The failure lies in the system that never connects them into a single picture.

Recommendations

Each recommendation below answers one of the failure points identified above.

Recommendation 1

Write the pressure plan as an order, with the reperfusion pathway on its face: Every acute ischemic stroke admission order should state which pathway the patient is on (thrombolysis, thrombectomy, or neither), the corresponding guideline ceiling, the documented pre-treatment baseline pressure, and a call parameter for how far down is too far. For a patient who received no reperfusion therapy: “No IVT/EVT. Permissive hypertension per AHA/ASA: do not treat unless SBP > 220 or diastolic blood pressure (DBP) > 120. If treated, target approximately 15% reduction over 24 h. Documented baseline home SBP 180 to 200; pre-treatment BP 200/105 (MAP ≈ 137). Call provider if SBP falls more than 15% below the pre-treatment value (below 170) or for any new neurologic change.” The call parameter is a communication device anchored to this patient’s baseline, and it should be labeled as such rather than presented as a validated perfusion threshold.

Recommendation 2

A neuro check before sedation: Before starting any sedative in a post-stroke patient with new altered mental status, perform and document a focused neurologic assessment that asks explicitly whether the change could be hypoperfusion. A one-line decision aid, “delirium or a neuro change?”, can live in the nursing workflow and in the electronic medical record (EMR), with Figure *2* as the template.

Recommendation 3

Caution with dexmedetomidine in acute stroke: Where it is used, pair it with continuous pressure monitoring against the ordered call parameter and a scheduled sedation hold, and have the team acknowledge up front that the sedation will blunt neurologic surveillance.

Recommendation 4

Teach autoregulation across disciplines, with the curve in hand: The counterintuitive point is the whole point - in these patients, allowing a higher pressure can be neuroprotective rather than negligent, consistent with experimental data showing that mild induced hypertension improves blood flow and oxygen metabolism in the penumbra [35]. Nursing curricula, in particular, treat elevated pressure as a problem to be

solved. The stroke exception has to be taught explicitly, or it will not translate to the moments it's needed the most.

Recommendation 5

Reform the order sets: Stroke blood pressure order sets should require the baseline pressure, the reperfusion pathway, and a flag for whether permissive hypertension is in effect. Decision support can fire when titration parameters conflict with stroke-specific guidance. Technically, this is the same problem as any other dose-range check, and it is already solved elsewhere in the EMR.

Future directions

The argument in this review is mechanistic, and the mechanism is better established than its clinical footprint. Nobody knows how often the sequence described here actually occurs. The first study worth doing is descriptive and needs no new equipment: in chronically hypertensive patients admitted with acute ischemic stroke, how far and how fast does pressure fall in the first 48 hours, how often is a delirium diagnosis or a new sedative order entered within hours of that fall, and does the pairing predict worse discharge outcomes than either event alone? A retrospective cohort would establish whether perfusion-blind sedation is a real clinical entity or an argument that only works on paper. The recommendations above are likewise untested. Whether rewriting a stroke blood pressure order set to require a documented baseline, a reperfusion pathway, and a call parameter changes what actually happens at the bedside is a question a single institution could answer with a before-and-after design.

The second question is whether the individual limit can be found at the bedside in a stroke unit. Continuous autoregulation indices derived from transcranial Doppler or near-infrared spectroscopy can locate an individual's lower limit in real time, and the near-infrared method is the more feasible of the two [29,36]. The wide variance in that limit is itself the argument for treating hypotension as an individual rather than a population-based definition [29]. Brown and colleagues located it in the controlled setting of the operating room, where pressure is managed over a matter of hours, not on a stroke unit, where it may fluctuate over days in a brain that already has an injured region. Whether an autoregulation index derived from near-infrared spectroscopy stays stable across the days a stroke patient spends in the ICU is unknown [29,36]. Acute ischemic stroke also complicates the picture, because the injured brain and the uninjured brain may not behave the same way, and a single number for the whole head may not describe either.

Third, and most useful if it works, is whether the pre-treatment pressure closely predicts the measured lower limit, serving as a bedside proxy. A negative result would also be worth publishing, as anchoring to the documented baseline (recommended above, since there's no better alternative) is currently untested. Only after those three does a trial of an autoregulation-guided pressure floor after ischemic stroke become designable.

Limitations

This is an illustrative composite scenario intended to demonstrate a documented physiology, rather than a report of a specific patient or a formal case report with verifiable data. No protected health information is included. The proposed sequence is the most physiologically plausible reading of this pattern, but it remains hypothesis-generating. Alternative explanations, including stroke progression independent of blood pressure, other causes of delirium, and concurrent events, cannot be excluded, and no causal claim about any individual patient is made or intended.

The trial evidence assembled here has its own boundaries. HOPE enrolled only patients with successful recanalization after thrombectomy and individualized the analysis by angiographic reperfusion grade, so its results support the general principle of tailoring rather than any specific target for the patients described here [24]. The INTERACT4 ischemic finding is a subgroup result. Figure *1* is a schematic. The pressures marked on it show the direction of the shift and should not be read as thresholds for any individual patient.

Conclusions

The scenario traces a foreseeable cascade: aggressive pressure reduction in a chronically hypertensive post-stroke patient, the resulting hypoperfusion read as delirium, and a dexmedetomidine infusion that may both worsen perfusion and hide the deteriorating exam. What makes it instructive is that no step requires anyone to act carelessly. The nurse treats high pressure because that is the training. The order set includes parameters that were never written for this physiology. The team names delirium because the picture fits, and reaches for a first-line agent. Each choice is defensible on its own, but the gap is the system's failure to assemble them into a coherent read.

The field is already moving toward tailoring interventions. The one trial that has tested it suggests that a blood pressure target chosen for the patient in front of you can outperform a generalized target. The patients in this review are further from that evidence and closer to the bedside, and for them, the tool available today is a documented baseline, a bounded reduction, and an examination that stays visible. The

lesson is that knowing the guideline only counts if it reaches the order and the handoff. That depends on shared understanding: when the physician writing the order and the nurse titrating the drip read the same curve, a systolic of 180 means the same thing to both of them, and the patient is safer for it. The fix lies in how intent gets communicated, individualized, and executed at the bedside. The distance between what we know about autoregulation and what happens when the monitor alarms for a systolic of 195 at 3 a.m. is where these patients are lost.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Ugo Okwudiri, Tyler Rogers

Acquisition, analysis, or interpretation of data: Ugo Okwudiri

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