Preliminary guideline- and pathophysiology-based protocols for neurocritical care

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Abstract

Background: Because of the complex pathophysiological processes involved, neurocritical care has been driven by anecdotal experience and physician preferences, which has led to care variation worldwide. Standardization of practice has improved outcomes for many of the critical conditions encountered in the intensive care unit.

Main body: In this review article, we introduce preliminary guideline- and pathophysiology-based protocols for (1) prompt shivering management, (2) traumatic brain injury and intracranial pressure management, (3) neurological prognostication after cardiac arrest, (4) delayed cerebral ischemia after subarachnoid hemorrhage, (5) nonconvulsive status epilepticus, and (6) acute or subacute psychosis and seizure.

Conclusion: These tentative protocols may be useful tools for bedside clinicians who need to provide consistent, standardized care in a dynamic clinical environment. Because most of the contents of presented protocol are not supported by evidence, they should be validated in a prospective controlled study in future. We suggest that these protocols should be regarded as drafts to be tailored to the systems, environments, and clinician preferences in each institution.

Keywords: Neurocritical care, Protocols, Guidelines, Pathophysiology, Shivering, Neurological prognostication, Delayed cerebral ischemia, Nonconvulsive status epilepticus, Psychosis, Seizure

Background

The art of neurocritical care requires an understanding of the pathophysiology of the highly complex central nervous system. Because of its complexity and the lack of evidence, the approach to neurocritical care is often clinician-dependent, i.e., driven by anecdotal experience and physician preferences, which leads to care variation. Overall, standardization of practice has improved outcomes for many critical conditions in the intensive care unit; thus, greater emphasis should be placed on reducing variation in neurocritical care practice.

Guideline- and pathophysiology-based protocols are concise yet comprehensive and are useful for bedside clinicians who need to provide consistent, standardized practice in a dynamic clinical environment. We introduce five preliminary protocols in this article. Because most of the text of the protocols addresses management in neurocritical care fields that lack firm evidence, and because of the varied availability of medical resources among institutions, we recommend that these protocols be used as drafts to be customized for the systems, environments, and clinical preferences of each institution.

Protocols

Prompt shivering management (Fig. 1)

Shivering is a physiological homeostatic response to maintain or raise temperature in hypothermia or fever when the set point temperature is elevated. However, shivering counteracts the effort of fever management and targeted temperature management (TTM)/therapeutic hypothermia, which are critical interventions to mitigate secondary brain injury. With inadequate shivering management, target temperature is difficult to achieve in a timely manner and may potentially worsen...
outcome. Furthermore, shivering increases the cerebral metabolic rate and may result in increased intracranial pressure (ICP) and brain oxygen consumption [1, 2]. Lastly, shivering increases the total body metabolic rate and total CO₂ production, which may raise the partial pressure of CO₂ and raise ICP. Therefore, shivering should be regarded as a neurological emergency requiring immediate control in patients with acute brain injury, and any protocol for shivering management should encourage clinicians to expedite treatment. The present draft proposal for shivering management refers to the Bedside Shivering Assessment Scale and shivering protocol proposed by Badjatia and Brophy, respectively, [1, 2] and was refined, based on our practice, to achieve prompt shivering control (Fig. 1).

**Traumatic brain injury and ICP management (Fig. 2)**

Intracranial hypertension, commonly defined as persistent elevation of ICP above 20–22 mmHg, is a relatively common neurologic complication seen after traumatic brain injury (TBI). If untreated, it can lead to cerebral ischemia, brain herniation, and possibly brain death. Therefore, care providers must promptly recognize the early clinical and radiographic features of elevated ICP and aggressively treat with a goal of reducing mortality and morbidity. The adult brain is a nearly incompressible substance enclosed in a fixed cranium. Therefore, ICP will inevitably be affected by a volume change in any of the three main intracranial components—cerebrospinal fluid (CSF), brain parenchyma, and blood [3]. In addition, if there is a new space-occupying lesion within the fixed cranium (i.e., hematoma), it will inevitably increase ICP. When assessing a patient with elevated ICP, it is important to determine whether the contributing factor is a focal, global, or mixed process since the treatment strategy may be different for each type of mass effect. If there is a focal, new mass-occupying lesion that is causing a regional mass effect and brain tissue compression, the

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**Record Bedside Shivering Assessment Scale (BSAS) every 1 hours and notify house officer if BSAS > 0**

| Score | Description                                                                 |
|-------|------------------------------------------------------------------------------|
| 0     | No shivering noted on palpation of the masseter, neck, or chest wall and no electrophysiological evidence of shivering (using ECG) |
| 1     | Electrophysiological evidence of shivering (using ECG), without clinical evidence of shivering                             |
| 2     | Shivering localized to the neck and/or thorax only                            |
| 3     | Shivering involves gross movement of the upper extremities (in addition to neck and thorax)                                |
| 4     | Shivering involves gross movements of the trunk, upper, and lower extremities |

**STEP 1 (Go to step 2 if BSAS still > 0 in 20 minutes)**
- Acetaminophen 1000mg IV bolus then 650-1000 mg PO every 6H
- Magnesium 2-4g IV bolus; if necessary, followed by 0.5-2g/H for goal 3-4mg/dL (Check Mg level every 6H)
- Buspirone 30mg q8H
- Skin counter-warming (e.g. Bair Hugger set at 43 °C)

**STEP 2 (Go to step 3 if BSAS still > 0 in 20 minutes)**
- Meperidine 25-50mg IV bolus every 1h as needed
- Fentanyl 25-100mcg IV bolus followed by 25mcg/H
- Then, consider adding the following to maintain BSAS 0
- Dexametadomidine IV infusion 0.2mcg/kg/H

**STEP 3 (Go to step 4 if BSAS still > 0 in 20 minutes)**
- Propofol 20-40mg bolus IV followed by 30-120mg/H if BP tolerates or
- Midazolam bolus 3-5mg IV followed by 2-15mg/H
- Intubate if not already done

**STEP 4**
- Cisatracurium 0.1–0.2mg/kg IV bolus followed by 2-10mcg/kg/min
- Rocuronium 0.6–1.2mg/kg IV bolus followed by 3-12mg/H
- Wean paralytics to maintain BSAS score 0 and 1-2 twitches on Train of Four (TOF)

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Fig. 1 Preliminary protocol for prompt shivering management [1, 2]. Modified from Brophy [2] with permission. Abbreviations: BSAS Bedside Shivering Assessment Scale, ECG electrocardiogram, IV intravenously, PO per oral, H hour, min minute
Fig. 2 (See legend on next page.)
first step is to consider surgical evacuation. Once the focal mass effect is ruled out or treated, global elevation of ICP must be addressed. The overarching strategy to control globally elevated ICP is to (1) optimize cerebral perfusion, oxygenation, and venous drainage; (2) prevent fever, hypercapnia, hyponatremia, hypo/hyperglycemia, and seizure; (3) provide adequate cerebral metabolic suppression with sedation; and (4) reduce cerebral edema with osmotic therapy. For refractory intracranial hypertension, treatments to be considered include pentobarbital-induced coma, therapeutic hypothermia, ventriculostomy placement for CSF diversion, and decompressive craniectomy. Ideally, a protocol for TBI management should include not only ICP control but also indications of initial surgical intervention for intracranial hematoma and basic management to prevent secondary brain injury. Step 1 (indications for surgical intervention) of the preliminary protocol is based on the recommendations by Bullock et al. [4–7], and step 2 (indications for ICP monitoring) and step 3 (basic management of TBI and ICP control) were developed in accordance with the guidelines for management of severe traumatic brain injury of the Brain Trauma Foundation and our practice (Fig. 2) [8].

Neurological prognostication after cardiac arrest (Fig. 3)
Cardiac arrest causes complete cessation of cerebral perfusion and rapidly depletes oxygen and glucose delivery to cerebral tissue. Cell death, which includes ion channel dysfunction and cell membrane destabilization, release

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**Fig. 2** Preliminary protocol for traumatic brain injury and ICP management [4–8]. Abbreviations: CT computed tomography, SBP systolic blood pressure, U units, 3PCC 3-factor prothrombin complex concentrate, 4PCC 4-factor prothrombin complex concentrate, ICP intracranial pressure, CVP central venous pressure, PCWP pulmonary capillary wedge pressure, SaO2 arterial oxygen saturation, PaO2 arterial pressure of oxygen, PaCO2 arterial pressure of carbon dioxide.

**Fig. 3** Preliminary protocol for neurological prognostication after cardiac arrest [13, 16]. Modified from Nolan [13] with permission. Abbreviations: ROSC return of spontaneous circulation, EEG electroencephalography.
**Baseline management**
- Maintain head-of-bed at 30 degrees
- Hold antplatelet or anticoagulant until aneurysm is secured
- Aneurysm repair within 24h
- Systolic blood pressure <140mmHg before aneurysm repair
- Systolic blood pressure <180mmHg (immediately post aneurysmal repair if no vasospasm after aneurysm repair)
- Nimodipine 60mg orally every 4h for 21 days (30mg every 2h if hypotension side effect is significant)
- Start DVT chemoprophylaxis on day 2-3 if bleeding is controlled.
- Again after coil embolization
- Routine use of seizure prophylaxis is not indicated unless clinical seizure was present.
- Fever and shivering management per protocol
- Insert ventriculostomy for obstructive hydrocephalus
- ICP management for elevated ICP per protocol
- Sodium level 135-140mEq/L
- Avoid hypertonic or dextrose containing IV fluids
- Maintain blood glucose 100-180 mg/dl

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**Risk stratification and monitoring of DCI**

### Low risk
- World Federation of Neurological Surgeons (WFNS) 1-2 or Hunt Hess 1-2 AND
- Modified Fisher scale 1 (thin SAH and no IVH)
  - Neurological assessment every 4-8 hours, transcranial Doppler (TCD) daily for 5-10 days
  - Go to “Diagnosis of delayed cerebral ischemia (DCI) for any new neurological changes"
  - Enhance level of monitoring for increased TCD mean flow velocities (>120cm/s or Lindegaard ratio>3) without neurological changes

### Moderate risk
- WFNS 3-5 or Hunt Hess 1-3 AND
- Modified Fisher scale 2, 3 (thin SAH with IVH or thick SAH without IVH)
  - Neurological assessment every 2-6 hours, TCD daily for 10-14 days
  - Go to “Diagnosis of DCI for any new neurological changes"
  - Enhance level of monitoring for increased TCD mean flow velocities (>120cm/s or Lindegaard ratio>3) without neurological changes

### High risk
- WFNS 3-5 or Hunt Hess 4-5 AND/OR
- Modified Fisher scale 4 (thick SAH with IVH)
  - Neurological assessment every 1-4 hours, TCD twice daily for 14 days
  - Go to “Diagnosis of DCI for any new neurological changes"
  - Enhance level of monitoring for increased TCD mean flow velocities (>120cm/s or Lindegaard ratio>3) without neurological changes
  - Consider surveillance CT or MR angiography (day 5-8 & day 13-15)

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**Diagnosis of DCI**
- Neurological deterioration (GCS<2 points or NIHSS<2 points)
or
- New focal neurologic deficits lasting for at least 1 hour
  - Not explained by other causes

**Findings supporting diagnosis of DCI**
- DSA or CTA : severe angiographic vasospasm [defined as a narrowing of at least 70% from baseline]
- TCD:
  - Mean flow velocities >180 cm/s or Lindegaard ratio>4 (severe)
  - Mean flow velocities 140-180 cm/s or Lindegaard ratio 4-4.5 (moderate)
  - Mean flow velocities 120-140 cm/sec or Lindegaard ratio 3-4 (mild)
- CT perfusion parameters : CBF>25mL/100g/minute, MTT (mean transit time)>6.5 seconds
- EEG : reduced alpha variability
- PI/PSO2 : <20mmHg
- Cerebral microdialysis (CQMO) : LPR>40, glucose<0.5mM, glutamate>40mM

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**Treatment of DCI**
1. 500-1000ml crystalloid bolus
   - Target euolema (hypervolemic is not superior than euolema) and evaluate neurological fluid responsiveness
2. Induced hypertension
   - Start phenylephrine or noradrenaline after excluding intracranial hemorrhage on CT
   - Increase vasopressor to titrate up SBP by 10mmHg to SBP 160-220mmHg until neurological improvements
3. Inotropic agents
   - Start dobutamine or milrinone if cardiac function is low (i.e. Takotsubo or baseline cardiomyopathy)
4. Intracerebral therapy
   - Percutaneous balloon angioplasty / intra-arterial vasodilators (i.e. Calcium-channel blocker)

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Fig. 4 Preliminary protocol for monitoring and diagnosis of delayed cerebral ischemia after subarachnoid hemorrhage [23, 24]. Abbreviations: SAH subarachnoid hemorrhage, DVT deep vein thrombosis, IVH intraventricular hemorrhage
of destructive enzymes, cell swelling, and, eventually, apoptosis, can begin within 5 min after complete cessation of cerebral perfusion [9–11]. After return of spontaneous circulation, neurological prognostication is essential because it enables clinicians to provide information to family members or surrogates who must consider decisions to limit care for patients with little hope of meaningful neurological recovery [11]. To date, there is no definitive diagnostic test to accurately predict functional outcome after cardiac arrest. Moreover, clinical findings shortly after cardiac arrest have little relationship to patient outcomes [12]. However, the use of a systematic approach allows for reliable prediction of a very poor neurological outcome (persistent vegetative state) and provides family members and surrogates with the information necessary to make decisions [13–16]. A protocol that clearly addresses “what to do next” would be helpful for clinicians. The present preliminary protocol is based on the 2015 European Society of Intensive Care Medicine Guidelines for Post-resuscitation Care [13], but includes additional detailed stepwise instructions (Fig. 3).

Delayed cerebral ischemia after subarachnoid hemorrhage (Fig. 4)
After subarachnoid hemorrhage, notably from a ruptured cerebral aneurysm, cerebral vasospasm can develop, thus leading to delayed cerebral ischemia (DCI) and possibly infarction. The mechanism of DCI is complex and is not solely attributed to large-vessel narrowing and associated low blood flow distally [17, 18]. Other postulated mechanisms are early brain injury, microcirculatory dysfunction with loss of cerebral autoregulation, cortical spreading depolarization, and microthrombosis [19, 20]. A DCI diagnosis is made clinically on the basis of symptoms such as new changes in mental status and neurologic deficits. Additional relevant information includes findings of CT or MRI angiography, digital subtraction angiography, and transcranial Doppler (TCD) ultrasound. In addition, other reversible causes of neurological changes must be ruled out, such as delayed hydrocephalus, nonconvulsive seizure, rebleeding, toxic-metabolic encephalopathy from infection, and medication side effects. To date, nimodipine, a calcium-channel blocker with cerebral vasodilatory

![NCSE protocol for patients without known epileptic encephalopathy](image)

**When to suspect non-convulsive seizures epilepticus?**
- Altered mental status which can not be explained by brain images or toxicological / metabolic causes
- Altered mental status or waking-waning exam in the context of known epilepsy, acute ischemic stroke, ICH, TBI, SAH, etc.
- Altered mental status which does not improve after cessation of clinical seizure (i.e. convulsion)
- Altered mental status with eye deviation, nystagmus, myoclonus or minor twitching of mouth, peri-orbital region, or extremities

**Start cEEG**

Frequency of epileptiform discharges (EDs) >2.5Hz lasting for more than 10 seconds?
- Spike and wave complex
- Polyspikes

Evolution:
- at least 2 ictal/vocal, sequential changes in one of the followings
- Frequency (e.g. 1Hz–2Hz–3Hz–1Hz)
- Morphology (e.g. slow wave–sharp wave–spike–slow wave)
- Location (e.g. F4-C4→F4-C4, C4-P4→F4-C4, C4-P4, P4-O2 → F4-C4)

**Spatiotemporal evolution of**
- epileptiform discharge (spike, sharp waves, polyspikes)
- rhythmic activity (RA) >0.5Hz

**Pathological clinical phenomena such as**
- minor twitching of mouth, peri-orbital region, or extremities
- One of the followings
  - EDs<2.5Hz with fluctuation
  - RA>0.5Hz with fluctuation

**Seek other causes for altered mental status**

**One of the followings within 10 min after benzodiazepine (e.g. Midazolam 2mg iv) and/or AED given**
- Improvement in mental status
- Improvement in EEG

Start treating as non-convulsive status epilepticus (NCSE)

**Fig. 5** Preliminary protocol for diagnosis of nonconvulsive status epilepticus [32]. Abbreviations: AED anti-epileptic drug
Step 1: Differential diagnoses and evaluations
- Drug use/toxicology screening
- Bacterial or fungal meningitis/encephalitis: Cerebrospinal fluid (CSF) glucose, protein, cell count with differential, VDRL, bacterial culture, acid-fast stain (AFB) culture, Cryptococcus culture, serum lytic antibody
- Viral encephalitis: Brain MRI with contrast, CSF glucose, protein, cell count with differential, CSF HSV/12 PCR and VZV PCR (Epstein-Barr virus PCR, CMV PCR, and HIV RNA if serum HIV positive)
- Hashimoto encephalopathy: anti-TPO antibody, anti-thyroglobulin antibody
- Acute disseminated encephalomyelitis (ADEM): Check for preceding infection or vaccination, brain MRI with contrast
- Reversible posterior leukocerebroencephalopathy syndrome: Brain MRI, check for history of preceding visual disturbance, episode of acute severe hypertension and causative agent such as cyclosporine, thalidomide, and methotrexate
- Leptomeningeal metastasis: CSF cytology, brain MRI with contrast
- CNS Vasculitis: serum ANA, ANCA, CT/MRI angiography or conventional angiography

No clear diagnosis

Step 2: Evaluate the possibility of autoimmune encephalitis with Antibody Prevalence in Epilepsy (APE) score

| Clinical Feature                                      | Value |
|-------------------------------------------------------|-------|
| New-onset, rapidly progressive mental status changes of 1-6 weeks, or new-onset seizure activity | 1     |
| Neuroaesthetic changes: agitation, aggressiveness, emotional lability | 1     |
| Autonomic dysfunction (presenting as tachycardia, labile heart rate, persistent hypertension) | 1     |
| Viral prodrome (unspecific symptoms only) | 2     |
| Facial dyskinesias or focal/infrequent dystonic movements | 2     |
| Seizure refractory to at least 2 antiseizure medications | 2     |
| CSF findings consistent with inflammation (elevated CSF protein level >50 mg/dl and/or lymphocytic pleocytosis >5 cells/μl) with the total number of CSF RBCs is <1000 cells/μl | 2     |
| Brain MRI showing signal changes consistent with limbic encephalitis (mild temporal lobe T2 signal changes) | 2     |
| Presence of underlying malignancy (excluding cutaneous squamous cell or basal cell carcinomas) | 2     |
| Total | 15   |

Therapeutic options including methylprednisolone, intravenous immunoglobulins or plasma exchange might be considered for patients with APE score >4

Step 3: Send autoimmune encephalitis panel for serum and CSF

- Antibodies against intracellular antigens
  - Hu/ANNA-1: Small cell lung cancer (SCLC)
  - CV2/CRMP5: SCG1, thymoma
  - Ri/JO-1: Breast cancer
  - Yo/ACA-1: Ovarian tumor
  - Scl/70: Connective tissue disease
  - ENA: Scleritis
  - EMA: Thymoma
  - ANNA-2: Thymoma
  - ANNA-3: Thymoma

- Antibodies against cell surface or synaptic proteins
  - NMDA: Ovarian teratoma
  - VGLial: Thymoma
  - Anti-PR: GC
  - Anti-Ri: GC
  - Anti-SSA: GC
  - Anti-SSB: GC

Step 4: Modify treatments once the antibody results are back

- Antibodies against intracellular antigens: consider adding second-line therapies such as rituximab or cyclophosphamide
- Antibodies against cell surface or synaptic proteins: continue first-line treatment (plasma exchange, IVIG, or plasma exchange) and consider adding second-line treatment if no clinical improvement
- Antibodies negative: consider adding second-line treatment if no clinical improvement evaluate for other causes especially infectious causes

Fig. 6 Preliminary protocol for acute or subacute psychosis and newly onset seizure [33, 35]. Modified from Dubey [33] with permission. Abbreviations: VDRL, venereal disease research laboratory, HSV herpes simplex virus, VZV varicella zoster virus, HIV human immunodeficiency virus, CMV cytomegalovirus, Anti-TPO anti-thyroid peroxidase, ANA anti-nuclear antibody, ANCA antineutrophil cytoplasmic antibody.
effect, is the only drug that has been shown to improve neurological outcomes in patients with subarachnoid hemorrhage [21]. Other calcium-channel blockers, such as nicardipine, have been used in countries where nimodipine is unavailable [22] but have not been shown to improve outcomes. Hemodynamic augmentation to increase oxygen delivery to the brain, including volume optimization and induced hypertension, is the mainstay for management of new-onset DCI. For refractory cases where medical management is ineffective, intra-arterial interventions such as balloon angioplasty and intra-arterial administration of calcium-channel blockers are a second-line treatment [23]. A protocol should include risk stratification and stepwise treatment for DCI in individual patients. The present preliminary protocol describes basic management of subarachnoid hemorrhage and risk stratification and monitoring, based on our practice and the existing literature [24–26]. Diagnosis and management of DCI are based on recommendations from the neurocritical care society and our practice [23] (Fig. 4).

Diagnosis of nonconvulsive status epilepticus (Fig. 5)
Nonconvulsive status epilepticus (NCSE) is characterized by electrographic seizure activity without clinical convulsions in patients who do not fully recover consciousness between attacks [27]. Although the impact of treating NCSE on clinical outcomes has not been investigated in a randomized controlled trial, the prognosis of NCSE is believed to be poor if not treated since untreated seizure is associated with secondary brain injury [28–30]. A diagnosis of NCSE should be considered in any patient with discrepancies between his/her neurological findings and clinical history or imaging findings such as CT or MRI. The typical example is a patient who develops sudden unexpected neurological deterioration after successful management of a structural brain injury without new findings on CT or MRI. Although the condition is referred to as nonconvulsive, patients with NCSE may have subtle motor symptoms such as sustained eye deviation, nystagmus, lip smacking, and twitching in the face or extremities [31]. Definitive diagnosis requires electroencephalography (EEG), and continuous EEG monitoring increases the sensitivity and specificity of NCSE diagnosis. We attempted to develop an algorithmic protocol to interpret EEG and diagnose NCSE for bedside clinical use. Based on the current guidelines [27, 32], this draft protocol for diagnosis of NCSE is designed to simplify NCSE diagnosis and management (Fig. 5).

Acute or subacute psychosis and seizure (Fig. 6)
Many medical conditions and pharmacologic side effects can cause unexpected psychosis or seizure. However, some treatable and reversible conditions, such as viral encephalitis and autoimmune encephalitis, are frequently missed. Delays in diagnosis and treatment of encephalitis may result in poor neurological outcomes. It is essential to review all possible causes of unexpected psychosis and seizure and to start empirical treatment for a potentially treatable condition before obtaining all examination results. The present draft protocol for diagnosis and management of autoimmune encephalitis includes comprehensive differential diagnoses and algorithms for diagnostic evaluation and was developed on the basis of a comprehensive literature review by Francesc et al., antibody prevalence in epilepsy (APE) score [33], and empirical treatment for autoimmune encephalitis in cases of unexpected psychosis or seizure, as reflected by the expert opinions of the European Federation of the Neurological Societies (EFNS) task force [34] and our practice (Fig. 6).

Conclusion
The present guideline- and pathophysiology-based protocols that can be customized for particular clinical environments may help providing consistent, standardized care in neurocritical care. Because most of the contents of presented protocol are not supported by evidence, they should be validated in a prospective controlled study in future.

Abbreviations
CSF: Cerebrospinal fluid; DCI: Delayed cerebral ischemia; EEG: Electroencephalography; ICP: Intracranial pressure; NCSE: Nonconvulsive status epilepticus; TBI: Traumatic brain injury; TCD: Transcranial Doppler; TTM: Targeted temperature management

Author contributions
YN is the guarantor of the manuscript content and protocols. YF and KN substantially contributed to the manuscript and protocols. All authors read and approved the final manuscript.

Consent for publication
Not required

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