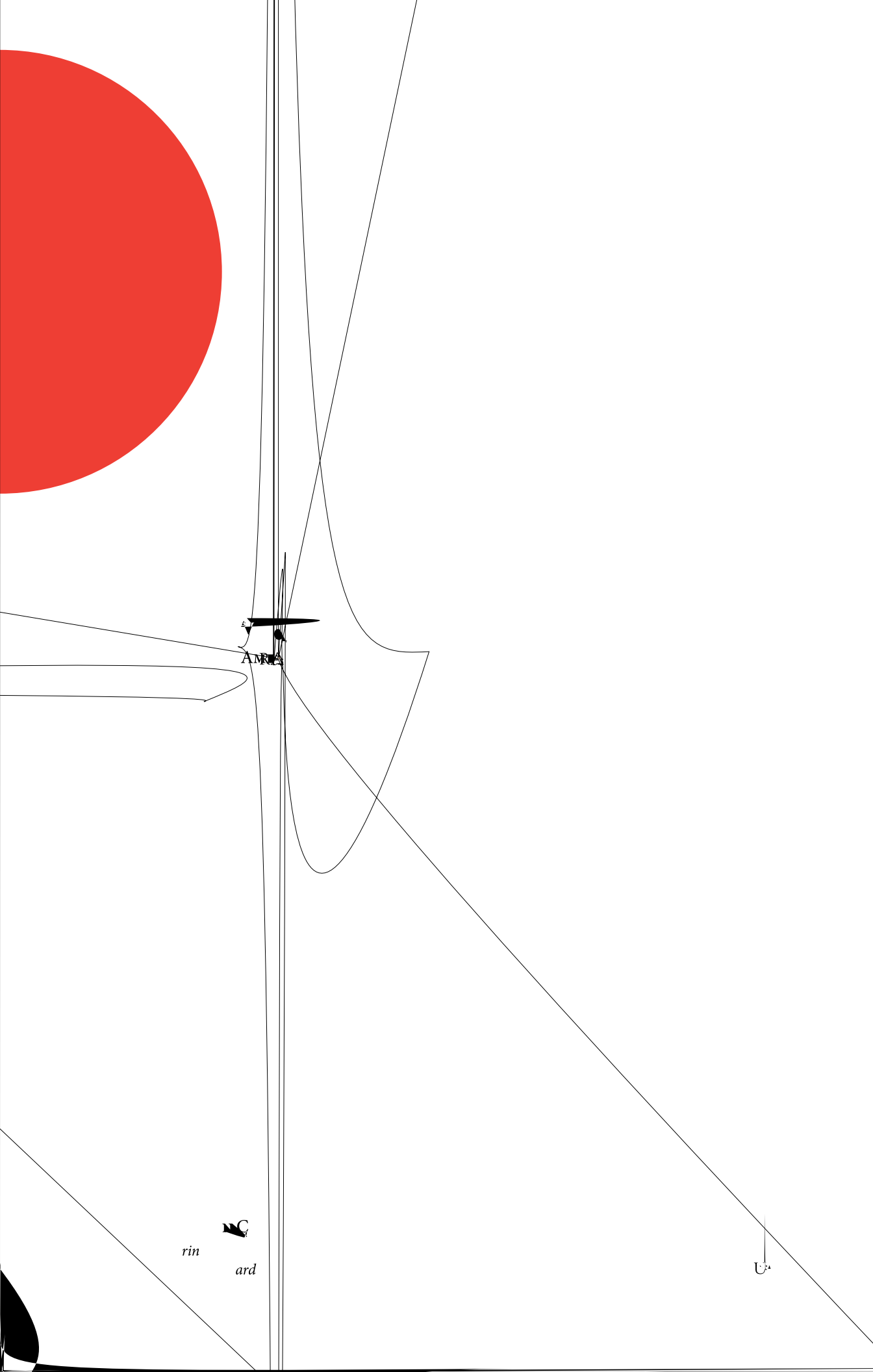

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A score of ≥13 correlates with a mild brain injury, 9 to 12 is a moderate injury, and ≤8 a severe brain injury.

If a GCS component is untestable due to intubation, sedation, or another confounder, the reason for this should be recorded. Although often done, a score of 1 should not be assigned because di□erentiation between a "true 1" and an untestable component is relevant. Graphical display of the three GCS components over time may facilitate earlier detection of changes.

Assessment requires either a spontaneous response or response following application of a stimulus. At more severely disturbed levels of consciousness, the motor score has better discrimination, but in milder injuries the eye and verbal components are more relevant. Thus, each component of the scale (Eye, Verbal, Motor) provides complementary information. Strengths of the GCS are that it covers a broad spectrum of disorders of consciousness, is widely applicable, and o□ers an important tool for monitoring changes in the level of consciousness. Standardized approaches to both its assessment and its reporting are required in order to be able to compare evaluations over time or when communicating with other health care professionals. Spontaneous responses are □rst observed without stimulating the patient in any way. First, verbal stimuli are applied, such as asking a patient to obey commands and at the same time observing whether, e.g., an eye opening occurs. If a patient is not responsive, a stimulus is applied to elicit a response. The location of the stimulus (central or peripheral)e □l)e1.7 ()3.5 (t)]

To pressure

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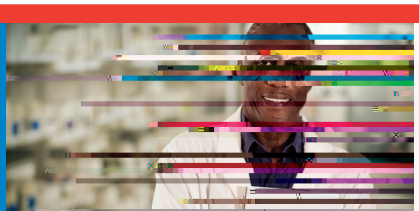


TRIAGE AND TRANSPORT

Patients with a Glasgow Coma Scale (GCS) ≤ 13 should be rapidly transported directly from the scene to the highest level trauma center available in a de□ned trauma system to allow for expedient neurosurgical assessment and intervention

Patients with a combination of TBI (GCS score ≤ 15) and moderate to severe extra-cranial anatomic injuries

hyperglycemia and hypoglycemia are detrimental to the outcome of patients with TBI. Serum glucose levels must be monitored closely in all TBI patients. More frequent monitoring is required

ICP monitoring is indicated in comatose patients (GCS ≤ 8) and if there is evidence of structural brain damage on initial CT imaging

ICP monitoring is generally not indicated in comatose patients without evidence of structural brain damage or elevated ICP (compressed/absent basal cisterns) on initial CT imaging. Patients may be observed with repeat CT imaging and forego ICP monitoring if there is no progression

ICP monitoring should be considered in patients with a GCS > 8 who have structural brain damage with high risk for progression (large/ multiple contusions, coagulopathy)

ICP monitoring should be considered in patients who require urgent surgery for extracranial injurie who n[h)1.1 (-.8 (ng)0 -)1.2imaging and forego ICP monini

The gold standard for ICP measurement is via an external ventricular drain (EVD), attached to an external strain-gauge transducer. The monitor, centrally

TIER 1

Head of bed elevated at 30 degrees (reverse Trendelenburg) to improve cerebral venous out□ow

Sedation and analgesia using recommended short-acting agents (for example, propofol, fentanyl, midazolam) in intubated patients

Ventricular drainage performed intermittently. Continuous drainage is not recommended unless an additional ICP monitor is placed, as when the drain is open, it does not accurately re□ect the true ICP

Repeat CT imaging and neurological examination should be considered to rule out the development of a surgical mass lesion and guide treatment

If ICP remains ≥ 20 - 25 mmHg proceed to Tier 2

TIER 2

PaCO2 goal of 30 - 35 mmHg should be maintained, as long as brain hypoxia is not encountered. Additional neuromonitoring (e.g., PbtO2, SjvO2, CBF) may help determine optimal PaCO2

Repeat CT imaging and neurological examination should be considered to rule out development of a surgical mass lesion and guide treatment

ADVANCED NEUROMONITORING

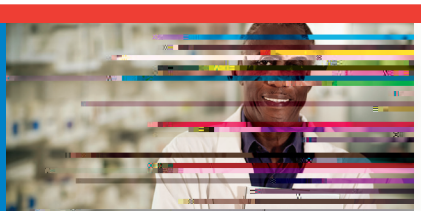
Advanced neuromonitoring and assessment of cerebral autoregulation may be helpful in identifying a more individualized approach to treatment

Impaired cerebral oxygenation can occur in the face of normal ICP and CPP

Cerebrovascular pressure reactivity index (PRx) and cerebral blood flow (CBF) monitoring can assess autoregulation status, which may help determine patient-specific CPP and ICP goals

TBI is a complex disease with substantial heterogeneity. ICP monitoring alone cannot detect all potential insults to the brain; ensuring adequate cerebral blood flow and oxygenation are important goals. Multiple studies have demonstrated an association between low brain tissue oxygen tension (PbtO2 ≤ 15 mm Hg) and episodes of jugular venous oxygen desaturation (SJvO2 ≤ 50 %) with poor outcome in TBI. Importantly, brain tissue hypoxia can occur even when ICP and CPP are normal. A recently completed Phase II prospective randomized clinical trial investigating PbtO2-based management of severe TBI compared treatment guided by ICP alone to treatment guided by both ICP and PbtO2 (BOOST, NCT00974259). The ICP+PbtO2 management group had statistically significant decreased duration and severity of brain hypoxia along with a 10% reduction in mortality and a trend toward reduced mortality and improved neurologic outcome at 6 months. This trial supports the value of advanced multimodality monitoring in TBI patients.

Cerebral pressure autoregulation is the brain's intrinsic ability to maintain constant CBF over a range of systemic blood pressures. This mechanism protects against cerebral ischemia due to hypotension and against excessive flow that can lead to elevated ICP. Cerebral autoregulation can be assessed at the bedside in the ICU with cerebrovascular pressure reactivity index (PRx) monitoring, CBF monitoring, and Transcranial Doppler (TCD) ultrasonography monitoring. The PRx is quantified as the slope of the regression line relating MAP and ICP and can be used to establish patient-specific CPP thresholds. For patients with impaired cerebral autoregulation (PRx slope > 0.13), a lower CPP (50 – 60 mm Hg) should be considered as an option for treatment. Patients with intact autoregulation (PRx slope < 0.13) may benefit from a higher CPP (50 – 60 mm Hg). When CBF is monitored directly, autoregulation status can be assessed with a hemodynamic challenge. In patients with intact autoregulation, CBF will change minimally in response to an increase in MAP. Conversely, CBF will rise with increasing MAP in patients with impaired autoregulation. Once determined, autoregulation status can be used to set CPP goals as described above. In a similar fashion, TCD ultrasonography and hemodynamic



maximal medical therapy. However, critics of this trial have highlighted unbalanced treatment groups, variability in medical treatments for the control group, high crossover rate to the surgical arm, and short-term follow-up (six months) as arguments against the conclusions of the study. The application of decompressive craniectomy for severe TBI remains a topic of lively debate.

Depressed skull fractures are commonly elevated if the depression is greater than the depth of the adjacent inner table, especially if located in a cosmetically important area like the forehead. Open depressed fractures are best treated surgically to prevent infection, but nonoperative management may be attempted in selected cases, limited to those without dural laceration, gross contamination or evidence of infection, or injury to the frontal sinus. In general, a depressed skull fracture over the sagittal sinus should not be treated surgically because of the high risk of uncontrollable hemorrhage.

NUTRITIONAL SUPPORT

Nutrition should begin early, as soon as the patient is hemodynamically stable, and ideally within 24-48 hours of injury

Enteral nutrition is recommended over the use of parenteral nutrition

Post-pyloric feeding methods are preferred as they are associated with a lower rate of pneumonia

Full nutritional supplementation should be achieved within 7 days of injury

Patients with TBI demonstrate

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TRACHEOSTOMY

If level of consciousness remains persistently depressed, TBI patients should undergo tracheostomy to facilitate liberation from mechanical ventilation; this can decrease risk of pneumonia and ventilator-induced lung injury

Relative contraindications to tracheostomy include high intracranial pressure, hemodynamic instability, and severe respiratory failure

All TBI patients deemed not likely to improve rapidly should be considered for early tracheostomy, within 8 days of injury

Patients su□ering severe TBI require mechanical ventilation in intensive care units as a component of their initial post-injury care. If the leluid4nBT11 0 0(. I)-alniapton.8 (r)-29 (s d)2 Tw 0trii stset tie4 (n)11.8ho ipte8 (t)-5d.3

Laparoscopic procedures
should be avoided

Close monitoring is required

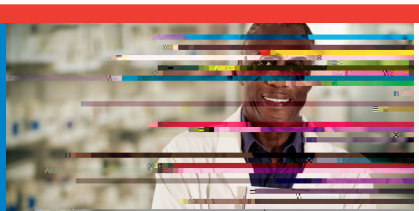
TIMING OF PHARMACOLOGIC VENOUS THROMBOEMBOLISM PROPHYLAXIS

- Patients with TBI are at high risk for venous thromboembolism (VTE), with rates as high as 20-30%
- VTE prophylaxis should be considered within the first 72 hours following TBI in most patients. Earlier initiation of pharmacologic prophylaxis (<72 hours) appears to be safe in patients at low risk for progression of intracranial bleeding and have a stable repeat head CT scan
- Placement of a prophylactic inferior vena cava (IVC) filter should be considered in patients at high risk for progression of intracranial hemorrhage who cannot receive pharmacologic prophylaxis, including those with lower extremity long bone fractures or pelvic fractures in addition to TBI

Patients with TBI are at high risk for venous thromboembolism (VTE) with rates as high as 20-30%, even with appropriate mechanical prophylaxis. In spite of these risks, providers have traditionally erred on the side of withholding pharmacologic VTE prophylaxis, accepting a higher risk of a VTE event in order to prevent potential progression of intracranial hemorrhage following TBI. The challenge in deciding when to initiate pharmacologic prophylaxis lies in determining when the risk of progression of intracranial hemorrhage has become sufficiently low. Evidence suggests that delays in initiation of > 4 days after injury substantially increases the risk of VTE, so balancing these risks is critical. One approach is to ensure that the brain injury has stabilized on CT before initiation of prophylaxis. In several studies, pharmacologic prophylaxis is withheld pending a CT scan at intervals ranging from 24-72 hours post injury. In the absence of any changes on CT scan, prophylaxis with a 01 (n C)3 (r)7.8 (o)1.7 (ph)20J0 T(h a 01 (n C)3

retrievable IVC filter can be considered in these patients, particularly those who are very high risk for VTE (e.g., patients with lower extremity long bone fractures or pelvic fractures) and removed after the risk is reduced. Alternatively, surveillance duplex ultrasound of the lower extremity can be undertaken and if a DVT is identified, a IVC filter can be conconsidered. Finally, some centers initiate LMWH in patients with ICP monitors and following craniotomy after a stable head CT, although this practice has not been investigated.

MANAGEMENT CONSIDERATIONS FOR PEDIATRIC PATIENTS WITH TBI



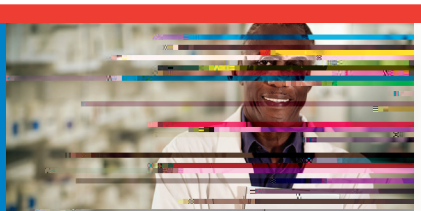
All aspects of care of the pediatric patient with TBI should be optimized

to reverse dabigatran (with or without hemodialysis) and Prothrombin Complex Concentrate (PCC) for rivaroxaban/apixaban. It is suggested that each center develop its own protocol for rapid reversal of anticoagulants using local expertise. For more information about reversal of anticoagulants in the elderly, please refer to the ACS TQIP Geriatric Trauma Management Guidelines.

Neurologic evaluation of the elderly patient with TBI can often be complicated by pre-existing dementia, cognitive decline, or hearing/vision de□cits. Family and caregivers can be invaluable sources of information when trying to determine a neurologic "baseline." Determining the appropriate level of diagnostic evaluation is important. One study found that in elderly patients with mild head injury, 14% of patients had evidence of traumatic lesion on head CT, with 20% of those lesions requiring neurosurgical intervention. Therefore, the American College of Emergency Physicians recommends that a head CT be obtained in any patient age ≥ 65 years who presents with mild head injury.

There is a paucity of information related to acute management of intracranial hypertension resulting from TBI in the elderly. Age-related changes in intracranial space are known to lower ICP signi□cantly, with a concomitant rise in CPP. Further, cerebral autoregulation and pressure reactivity indices are known to decrease over time. These changes can be complicated by comorbid conditions and medications that are more common in the elderly patient sustaining TBI. Well-studied recommendations for optimal CPP thresholds in the elderly are lacking.

It is clear that as age advances, the risks of mortality and poor functional outcome from TBI increase. This is true for all types of brain injury, but most striking with a GCS < 9. Despite this grim prognosis, 30% of elderly TBI patients with severe TBI can survive to leave the hospital. There is tremendous variability in the aggressiveness of medical care following traumatic brain injury. This likely is due to local, regional, and cultural di□erences in how care is provided. Many of those deaths occur early after brain injury and likely re□ect early decisions to withdraw life-sustaining therapy. At this time, due to the lack of su□cient prognostic tools, it is di□cult to determine which patients may go on to have a meaningful recovery. Arbitrary age thresholds for limitations of care should be avoided. Rather, a detailed discussion with the family and decision-makers should center around the severity of injury, comorbid conditions, and respect for a patient's previously expressed wishes.



Given these concerns, the advocated best practice is to provide all severe TBI patients with a trial of aggressive therapy and not limit any interventions for at least 72 hours post-injury. While this time period is somewhat arbitrary, it represents a minimum period during which the e□ectiveness of initial interventions and the likelihood of patient survival can be assessed. Exceptions would be patients who are brain-dead or in whom a pre-injury Advance Directive states that such intervention is not desired. A longer period of treatment and observation is typically needed for prognosis of neurological recovery. Age, taken in isolation, should not be considered a valid reason for treatment-limiting decisions.

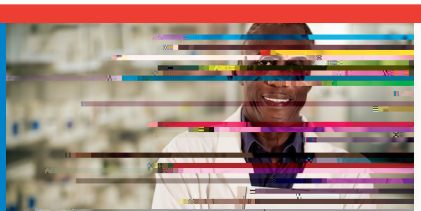
State law governs the criteria for the determination of brain death. However, standardized criteria for the determination of brain death have been developed and should be utilized. Speci□cally, patients must have no response to central pain, absent brainstem re□exes, and the inability to breathe independently. The clinical examination should be used rather than a con□rmatory test, such as electroencephalography or cerebral blood □ow assessment, unless prerequisites for using the clinical examination cannot be met. It is strongly encouraged that hospitals develop a de□ned brain death determination policy that derives from the accepted national standards.

OUTCOME ASSESSMENT AND QUALITY IMPROVEMENT IN TBI

Outcome assessment is essential to benchmarking the quality of care in TBI patients

A standardized and structured outcome assessment using the GOS-E at 6 months is recommended for TBI patients

TBI is a major cause of long-term change in functional, physical, emotional cognitive, and social domains. Assessment methods have di□erent strengths and weaknesses, and few can be applied across the complete TBI severity spectrum. For a global assessment of function, the Glasgow Outcome Scale (GOS) or its expanded version, the Glasgow Outcome Scale-Extended (GOS-E) is broadly used to assess outcome of TBI. While the GOS/GOS-E may be appropriate for rating outcome in the long term, it is not suited for assessing outcome upon discharge. This is particularly notable for patients at the more severe end of the TBI spectrum who have been admitted to the intensive care unit. These patients are often in poor condition on discharge from the intensive care unit but improve over the weeks and months thereafter. Observing these changes and evaluating long term outcomes may provide



reinforcing evidence for establishing best practices to treat patients aggressively in the first days post-injury.

Improvement after TBI may occur over months or even years. Conversely, a minority of patients may show deterioration over time. A standardized

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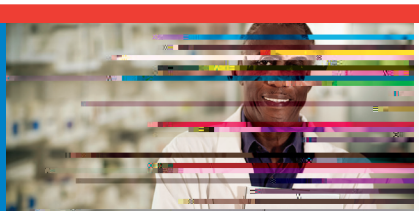

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