Stroke

AHA SCIENTIFIC STATEMENT

Addressing Systemic Complications of Acute Stroke: A Scientific Statement From the American Heart Association

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ABSTRACT: Systemic, nonneurological complications are common after ischemic and hemorrhagic strokes, affect different organ systems, and have a major impact on patient outcomes. Despite their obvious implications, this area in stroke management remains inadequately researched, and current literature offers fragmentary guidance for care. The purpose of this scientific statement is to elucidate the major systemic complications of strokes that occur during hospitalization, to synthesize evidence from current literature and existing guidelines, to address gaps in knowledge, and to provide a coherent set of suggestions for clinical care based on interpretation of existing evidence and expert opinion. This document advocates for improved interdisciplinary collaboration, team effort, and effective implementation strategies to reduce the burden of these events in clinical practice. It also calls for further research on strategies for preventing and managing systemic complications after stroke that improve outcomes in stroke survivors.

Key Words: AHA Scientific Statements ■ deglutition disorders ■ hemorrhagic stroke ■ ischemia ■ sleep apnea syndromes ■ stroke

The effects of a stroke can extend beyond the brain, involving various organ systems and giving rise to a range of systemic (nonneurological) complications.[1] These complications may stem from direct tissue injury caused by the stroke, its impact on different physiological systems, or adverse effects of specific diagnostics or therapies and carry significant implications for care, resource use, and patient outcomes. The reported frequency of poststroke systemic complications in the literature has varied between 13.9% and 95%, reflecting differences in study methodologies, cohort selection, ascertainment methods, and the analyses used (Supplemental Table 1A). A disproportionately high frequency of these events occurs shortly after a stroke, constitutes a leading cause of early stroke mortality, and is associated with long-term disability.[2]

Few well-designed randomized controlled clinical trials (RCTs) have examined specific interventions for preventing or treating poststroke systemic complications.[3,4] One RCT that specifically studied the impact of stroke unit compared with nonstroke unit care demonstrated that patients in stroke units were more likely to have established processes of care to manage complications, had fewer complications, and had lower odds of severe disability or death.[5] The large multicenter QASC RCT (Quality in Acute Stroke Care) examined implementation of protocol-based care targeting 3 main complications (fever, hyperglycemia, and swallowing assessment) across several stroke units and showed significantly better outcomes in the intervention compared with the routine care group, with an effect size comparable to that for intravenous thrombolysis.[6]

Although organized stroke unit care has been strongly recommend in guidelines,[7] stroke care remains fragmented in real-life practice, and many stroke survivors do not receive care in dedicated stroke units. Tailoring interventions to meet the unique needs of patients with stroke, considering their communication deficits,

Supplemental Material is available at https://www.ahajournals.org/journal/doi/suppl/10.1161/STR.0000000000000477.

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cognitive impairment, and complex physiology, remains a challenge. Furthermore, differences in clinical settings, access to resources, and quality of health services lead to variabilities in care and contribute to health care inequity.

To address the importance of this topic and its implications for care, the American Heart Association constituted a multidisciplinary writing group to review and curate existing literature on this topic, to identify knowledge gaps, and to provide suggestions for care whenever appropriate. This scientific statement exclusively addresses the major systemic stroke complications that occur during hospitalization and either are frequent or have major clinical relevance. For purposes of this document, we have adhered to the traditional topographic classification of diseases for categorizing these complications. Supplemental Table 2 summarizes key systemic complications reviewed in this scientific statement, clinical management considerations, and knowledge gaps/future directions.

METHODS

The American Heart Association Stroke Council's Stroke Scientific Statement Oversight Committee appointed a chair of the writing group on the basis of subject expertise who was entrusted with drafting an initial thematic framework for the scientific statement. This draft contained a topical list of clinically important systemic complications that occur during hospitalization after acute ischemic stroke (AIS), intracerebral hemorrhage (ICH), and subarachnoid hemorrhage but excluded those specific only to the intensive care unit milieu such as mechanical ventilation and hemodynamic support. In addition, specific neurological, cardiovascular, and hematological complications and electrolyte derangements were also excluded because they have been addressed in other guidelines and scientific statements.[7–10] The Statement Oversight Committee reviewed the manuscript outline and suggested changes to avoid redundancies and overlap with existing guidelines and scientific statements.

After initial approval of this outline, the American Heart Association Statement Oversight Committee nominated and convened a multidisciplinary expert writing group, including representatives from vascular neurology, neurocritical care, internal medicine, nursing care, gastroenterology, and urology. Members were assigned specific topics for content development and comprehensive literature review according to their expertise. The entire writing group subsequently reviewed the content, arrived at a consensus in an iterative fashion, and finalized the manuscript draft. The American Heart Association Science Advisory and Coordinating Committee reviewed the interim draft and suggested revisions, including incorporation of additional content, which was revised by the writing group and submitted for approval.

FEVER AND INFECTIONS

Fever

Hyperthermia and fever are common occurrences after a stroke and can develop from infectious and noninfectious causes. The preoptic nucleus of the anterior hypothalamus plays a central role in thermoregulation in both health and diseased states.[11] Fever occurs when the hypothalamic set point is elevated, whereas in hyperthermia, the hypothalamic set point is normal but heat generation exceeds dissipation.[11] Elevated body temperature is observed in 60% of patients with AIS to 90% of patients with ICH within 72 hours of stroke onset.[1] Fever and hyperthermia cannot be reliably differentiated on the basis of severity or patterns of temperature elevation alone. Although existing literature uses variable thresholds to define fever (37.7°C/99.5°F–38.3°C/100.9°F), epidemiological data indicate a consistent relationship between magnitude of body temperature elevation and poor outcomes in both ischemic and hemorrhagic strokes.[8,9,12] Patients with greater stroke severity and presence of ICH and intraventricular hemorrhage are at a higher risk for developing fever.[1,12,13] Stroke can also impair systemic immunity, rendering patients susceptible to systemic infections, particularly from bacterial pathogens.[14] Poststroke infections occur in almost 30% of all patients with acute stroke, two-thirds of them attributed to pneumonias and urinary tract infections (UTIs).[15] Fever may also precede a stroke, especially when stroke is a result of an underlying infection such as endocarditis or meningoencephalitis; strong evidence also implicates systemic infections as a trigger for a stroke.[16]

Noninfectious causes of poststroke fevers may include malignancy, autoimmune disorders, drug reaction, venous thromboembolism (VTE), or central fever[1,13,14] (Figure 1). Secondary aseptic cerebrospinal fluid inflammatory response from upregulation of cerebrospinal fluid proinflammatory pyrogenic cytokines could be a noninfectious cause of central fever, especially in hemorrhagic strokes (ICH, intraventricular hemorrhage, subarachnoid hemorrhage).[17] In addition, severe strokes have been associated with body temperature elevation within 4 to 6 hours of stroke onset but not mild or moderate strokes, suggesting that the extent of brain tissue damage may play a role in its genesis.[18]

Despite the clear association between temperature elevation and poor stroke outcomes, the utility of fever treatment in improving outcomes remains inconsistent. Although some cohort studies report that pharmacological treatment of temperature exceeding 37.5°C for >48 hours improved outcomes after ICH, a phase 3 RCT found no difference in functional outcomes between patients with AIS and ICH treated within 12 hours of symptom onset with paracetamol and those treated with placebo; however, a post hoc analysis of patients with a baseline body temperature of 37°C to 39°C showed that

 Stroke. 2024;55:00–00. DOI: 10.1161/STR.0000000000000477

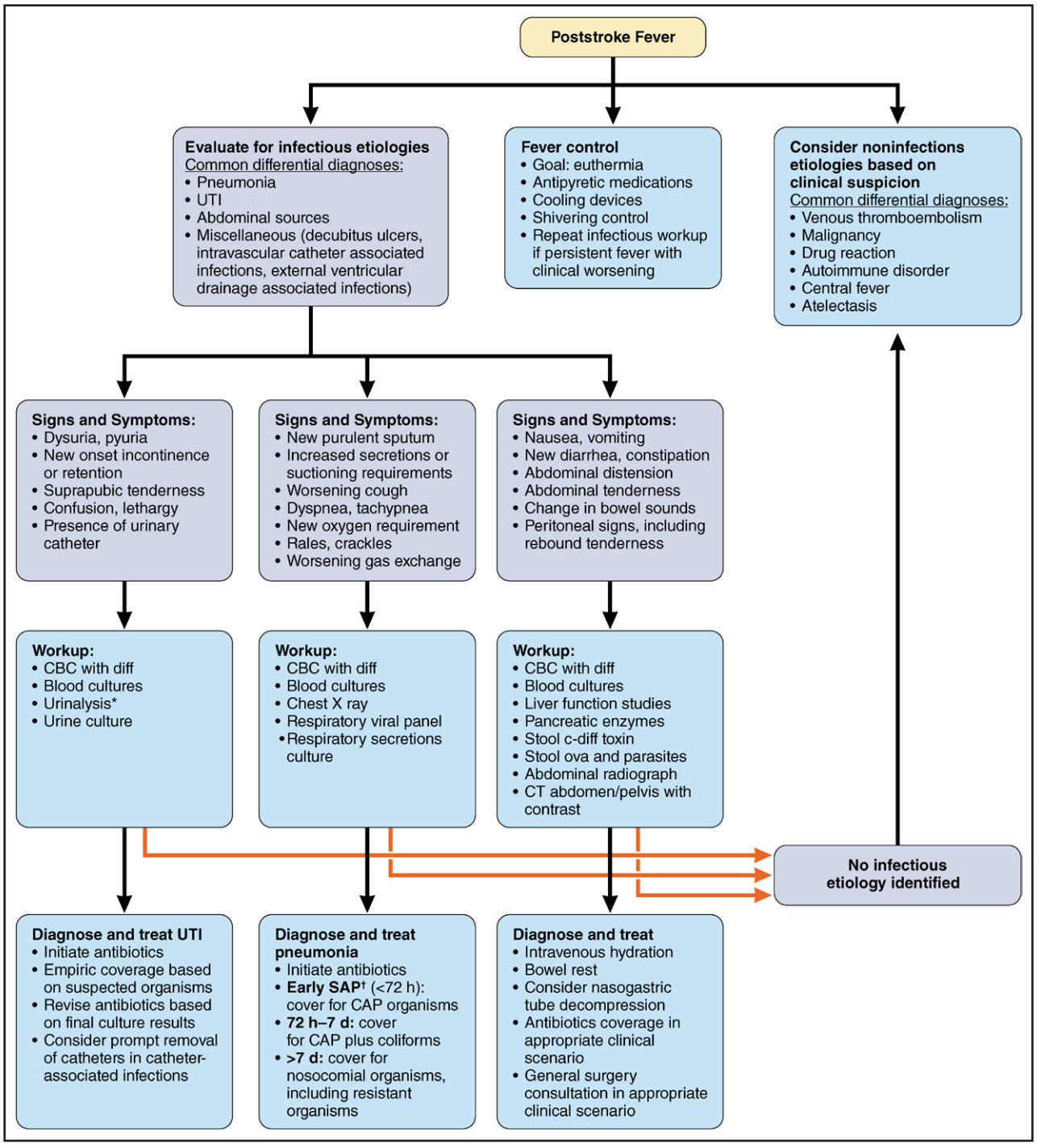

Figure 1. Suggested framework for diagnostic investigations and management of poststroke fever.
The figure outlines common sources of fever in hospitalized patients with stroke and a broad scheme for their diagnosis and management. CBC indicates complete blood count; and CT, computed tomography. *Stroke-associated pneumonia. †Community-acquired pneumonia.

paracetamol treatment led to better outcomes.[19] PRECIOUS (Prevention of Infections and Fever to Improve Outcome in Older Patients With Acute Stroke), a phase 3 RCT examining prophylactic antibiotics, fever control with paracetamol, and dysphagia treatment with metoclopramide in elderly patients with AIS and ICH, found no impact of intervention on stroke outcomes at 3 months.[20]

Managing poststroke fever can be challenging because it may be refractory to conventional antipyretics. Surface cooling devices and invasive endovascular cooling have been explored but have not led to clinical improvement despite greater success in achieving temperature control.[21] Although fever treatment alone has not demonstrated clear clinical benefit, protocol-based

monitoring and intervention for fever management was a major aspect of the QASC trial, revealing an overall improvement in outcomes.[6] A recent RCT, INTREPID (Impact of Fever Prevention in Brain Injured Patients), completed patient enrollment in May 2022 with results pending.[22] We recommend following the current American Heart Association guidelines on AIS, ICH, and subarachnoid hemorrhage in terms of fever control.[7–9]

Stroke-Associated Pneumonia

Almost 1 in 10 patients develop pneumonia after stroke during hospitalization.[15,23] Pneumonia is a leading cause of early stroke mortality, worsens stroke outcome, and increases risk of stroke recurrence.[15,23,24]

Stroke-associated pneumonia (SAP) is distinct from pneumonia in other contexts because of its unique clinical setting, pathophysiology, and microbiology.[25] Patients with stroke can develop reservoirs of infectious pathogens in their oral cavity, nasopharynx, and upper gastrointestinal tracts. Stroke-related deficits such as dysphagia, impaired cough, and reduced consciousness can facilitate migration of pathogens to the lungs, and stroke-induced immunosuppression impairs host immune responses, heightening susceptibility to infection.[14] Commonly isolated organisms in SAP include aerobic Gram-negative bacilli and Gram-positive cocci.[26]

Diagnosing SAP can be challenging because other causes of poststroke fever and dyspnea can cloud the clinical picture. The utility of chest x-ray in this context is limited, approaching an accuracy of only 68%.[27] Proposed standardized algorithms can enhance diagnostic accuracy of SAP; including plasma C-reactive protein in these algorithms may be advantageous but requires further study.[25,28] Several clinical instruments for predicting SAP have been developed, although their performance is moderate and not significantly improved with the addition of inflammatory blood biomarkers.[29,30]

RCTs of systemic preventive antibiotics in AIS have been ineffective in preventing pneumonia or improving clinical outcomes.[3] Metoclopramide, studied in patients with severe stroke with nasogastric feeding, showed promise in reducing pneumonia in a phase 2 trial but did not affect outcome in a phase 3 RCT; another multicenter trial of metoclopramide and oral decontamination is ongoing.[31,32] Consensus guidelines for antibiotic treatment for SAP have been proposed in the absence of high-quality data to inform therapy.[33]

Accurate prediction of SAP risk is essential for designing RCTs for effective prevention and treatment. Current predictive tools need refinement, and there is a need for improved diagnostic algorithms, accurate biomarkers, and reliable imaging techniques. Although routine preventive antibiotics are not recommended,[7,8] we advocate concerted efforts through prompt swallow assessment and oral health care (OHC) to avoid SAP.[7,34] Patient mobilization, head positioning, and chest physical therapy may play a role but require further investigation. In the absence of definitive evidence, aligning antibiotic treatment for SAP with consensus recommendations may be helpful[33] (Figure 1).

Oral Health Care

Patients with stroke have a higher prevalence of poor oral health, including gingivitis, periodontal disease, xerostomia, tooth loss, and dentures use, which increases risk of SAP from aspiration of oral biofilm and contributes to poor nutrition, pain, and reduced quality of life.[35,36] OHC remains a neglected aspect of stroke care.[36]

Providing OHC in hospitalized settings is challenging because stroke-related deficits can hinder self-care and there is insufficient evidence and emphasis to guide staff training and delivery of OHC.[36,37] Assessing oral health in patients with acute stroke is important for establishing a baseline, identifying hygiene requirements, devising care plans, and arranging dental referrals as necessary. There is a paucity of specific and validated oral health assessment tools for acute stroke settings.[36]

A Cochrane review of RCTs of OHC interventions in stroke care, conducted predominantly in rehabilitation settings, failed to demonstrate benefits of OHC intervention in improving gingival health, preventing pneumonia, enhancing patient satisfaction, and improving quality of life.[38] Questions persist about the best strategies for providing OHC in patients with acute stroke, including the role of oral decontamination with chlorhexidine and the impact of powered brushing on aspiration risk. Further trials of OHC interventions are needed that incorporate staff training and implementation strategies. We suggest using a standardized oral assessment tool on stroke admission, twice-daily brushing with nonfoaming toothpaste in an upright position, and targeted suctioning for patients with high-risk aspiration. Individualized approaches for issues such as dry mouth can be determined by nursing staff, with establishing pathways for staff education, training, and dental referrals.

Urinary Tract Infection

The pooled incidence of UTI in hospitalized patients with stroke is 10%, making it one of the most common poststroke infections.[1,15] UTIs after stroke increase the length of hospitalization and cost of care and delay rehabilitation.[39]

Several converging factors increase UTI risk after stroke, the most common being indwelling urinary catheters.[40] Higher stroke severity, advanced age, female sex, and urinary retention also augment UTI risk; preexisting

Stroke. 2024;55:00–00. DOI: 10.1161/STR.0000000000000477

comorbidities such as enlarged prostate, prolapse, or incontinence can further contribute.[39,40] Stroke-induced immunosuppression and use of urinary catheters may increase the severity of UTI, leading to prostatitis, pyelonephritis, and urosepsis.

Specific criteria for diagnosing symptomatic UTIs, catheter-associated UTI, and asymptomatic bacteriuria have been developed to avoid antibiotic overuse for treating UTI.[41] These criteria emphasize the presence of urinary symptoms combined with culture data for diagnosis. Diagnosis of UTI after stroke, however, poses unique challenges because of the difficulty of eliciting symptoms from patients who may have coexisting communication or other cognitive deficits.

A suggested framework for UTI diagnosis and management that borrows key elements from existing recommendations is outlined in Figure 1. A systematic review investigating the use of antibiotics for prophylaxis of poststroke infection did not show any benefit in improving functional outcome or decreasing mortality; however, lower rates of UTIs were observed in the antibiotic arm.[3] These results require cautious interpretation because they were based on secondary analysis with inherent risk of bias. Although current recommendations are not tailored to hospitalized patients with stroke, they include several suggestions for care that may help lower rates of catheter-associated UTI.[42] Adopting measures for managing urinary retention and incontinence, good perineal care, and good hand hygiene can help reduce the risk for poststroke UTI.[10]

RESPIRATORY COMPLICATIONS

Venous Thromboembolism

Deep venous thrombosis and pulmonary embolism (PE; VTE) are well-known stroke complication. The epidemiology, prevention, and treatment of deep venous thrombosis have been covered extensively in the existing stroke guidelines.[7–9] However, prospective epidemiological investigations on poststroke PE and good-quality RCTs evaluating the efficacy and risks of existing treatment strategies in this patient population are currently lacking.

The incidence of PE within the first month after stroke was estimated to be ≈1% in a large cohort study.[43] Despite its low incidence, it is a major cause of early mortality after stroke.[43,44] Diagnosing PE can be challenging in neurologically impaired patients with stroke, and a combination of signs, symptoms, and predisposing factors can help gauge its risk. These include symptoms such as dyspnea (especially of acute onset), chest pain, cough, hemoptysis, diaphoresis, dizziness, and syncope. Tachycardia, hypoxia, and hypotension are important clinical signs. Prestroke disability, high stroke severity, leg weakness, dehydration, and elevated D-dimer, C-reactive protein, and homocysteine levels are known risk factors for poststroke VTE.[45,46] Clinical decision rules (including D-dimer levels) have been developed to guide confirmatory testing for PE in clinical practice.[46] However, the accuracy of these tools has not been validated in hospitalized patients with stroke.

Pulmonary angiogram is the historical gold standard for confirmatory diagnosis but is invasive and not readily available.[47] The use of multidetector computed tomography angiography for diagnosing PE is more widespread, and a normal computed tomography angiography rules it out accurately.[48] Ventilation-perfusion single-photon-emission computed tomography is a low-radiation option, but its diagnostic performance is less well studied.[47]

Clinical support of acute PE in a patient with stroke includes supplemental oxygen to maintain normal arterial oxygen saturation, to avoid hypovolemia, and to maintain hemodynamic stability. It is strongly recommended that an early assessment of PE risk severity be made in all patients with suspected PE[46,49] (Figure 2). Patients with high-risk PE should be identified promptly because they face high mortality and require immediate critical care support and reperfusion therapy.[47,49] Such patients may have refractory hypoxia, are hemodynamically unstable, including having hypotension (systolic blood pressure of <90 mm Hg or a decrease in systolic blood pressure of >40 mm Hg), symptoms of shock, and end-organ hypoperfusion (cold, clammy skin; oliguria/anuria; rising serum lactate); and may progress to cardiac arrest.[47,50] Systemic thrombolysis, recommended by current guidelines on management of PE,[47,50] carries a high risk of intracranial hemorrhage in most patients with acute-subacute stroke and is rarely used in this patient population. Catheter-assisted pulmonary thrombectomy and surgical embolectomy are viable options but have not been systematically investigated in the stroke population. Patients with intermediate risk may have echocardiographic or computed tomography evidence of right-sided heart strain or elevated cardiac biomarkers (troponin levels) and are at high risk for hemodynamic decompensation. They require close monitoring and a plan for rescue reperfusion therapy if they decompensate[47,49] (Figure 2). Prompt consultation with a local critical care team for the management of intermediate- and high-risk stroke-associated PE should be considered.

Most patients with poststroke PE should be evaluated for systemic anticoagulation. However, the risk of bleeding complications requires careful consideration, and treatment decisions should be individualized. Validated tools to stratify bleeding risks in patients with acute stroke with VTE who are treated with therapeutic anticoagulation are currently unavailable. Use of short-acting, easily reversible anticoagulants is preferable, although the safety of this approach has not been evaluated in this patient population. Low-molecular-weight heparin has been recommended as the initial anticoagulant of

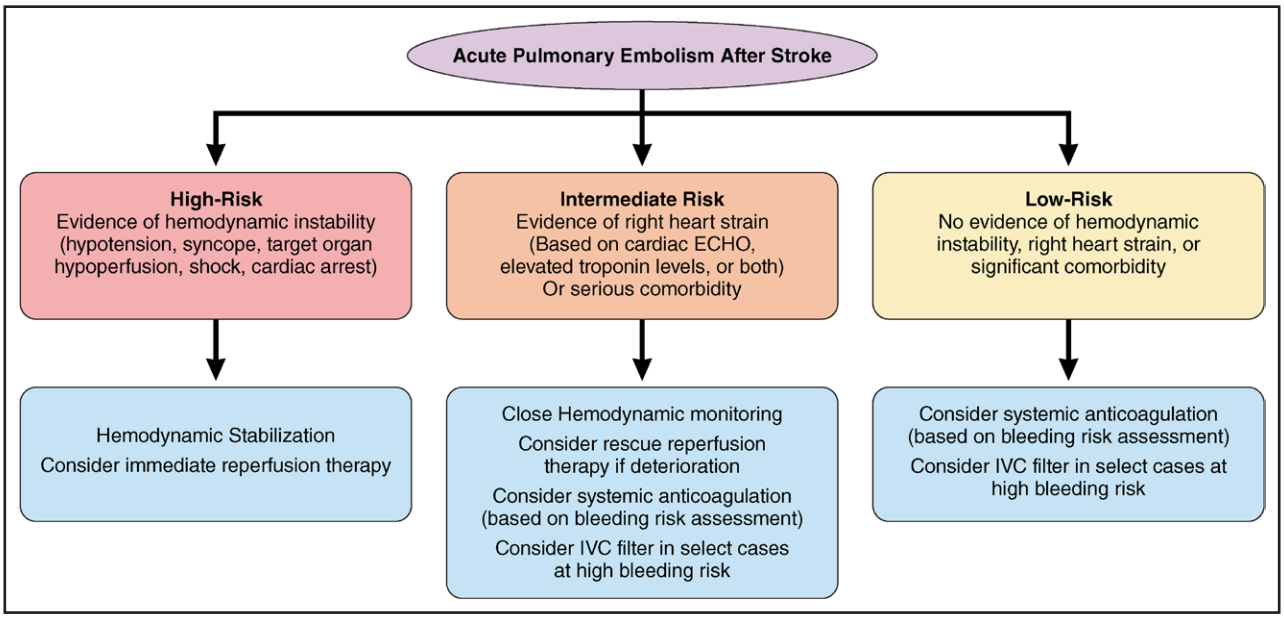

Figure 2. Suggested framework for management of acute pulmonary embolism after stroke based on risk stratification.
ECHO indicates echocardiogram; and IVC, inferior vena cava.

choice in current guidelines depending on renal function; other agents such as intravenous unfractionated heparin, intravenous thrombin inhibitors, fondaparinux, warfarin, and direct oral anticoagulants can be considered according to the individual risk profile.[47,49] Placement of an inferior vena cava filter has been recommended in patients with lower-extremity deep venous thromboses and those unable to receive systemic anticoagulation because of higher bleeding risks.[47,49] Most patients with stroke with PE require therapeutic anticoagulation for at least 3 months.[47,49] Reassessment for extended therapy beyond 3 months should be based on reassessing the risk for recurrent deep venous thrombosis (eg, prolonged immobility).[47,49] Moderate- or high-risk PE can require longer durations of anticoagulation.[47]

Breathing Disorders

Patients with stroke are at risk for respiratory complications such as pneumonia, aspiration pneumonitis, pulmonary edema, and PE. Stroke can also affect normal regulation of breathing, leading to a diverse array of breathing disorders Supplemental Table 3 which can impair pulmonary gas exchange and exacerbate brain injury.

Sleep-disordered breathing (SDB) has been well characterized after stroke, occurs during sleep or with drowsiness, and includes obstructive sleep apnea (OSA) and central apnea.[50,51] The most common SDB is OSA, which is a reduction of airflow during sleep from upper-airway collapse; in contrast, central sleep apnea is cessation of breathing due to loss of a central respiratory drive Supplemental Table 3.

A large meta-analysis reported that >70% of stroke survivors experienced SDB within a month of their stroke onset, with one-third having severe SDB.[51] OSA, a leading cause of SDB in patients with stroke, is a known risk factor for stroke but can develop anew or worsen after stroke. OSA is associated with worse functional outcomes, increased stroke recurrence rates, and higher mortality.[52] Pilot studies have suggested potential benefits of early OSA treatment using nasal continuous positive airway pressure (CPAP) and autotitrating CPAP, but this requires further confirmation.[53] However, CPAP use in patients with acute stroke poses risks, including increased intracranial pressure and aspiration. An ongoing multicenter RCT is investigating whether CPAP use for OSA in patients with AIS prevents stroke recurrence and improves outcomes.[54] Currently, routine screening for SDB in patients with acute stroke lacks conclusive evidence, and current AIS guidelines recommend against screening.[7]

Cheyne-Stokes respiration, characterized by cyclic breathing patterns, also occurs frequently in hospitalized patients with stroke but improves over time.[55] Stroke-related Cheyne-Stokes respiration is usually seen with large hemispheric infarcts or brainstem injury, particularly in patients with associated congestive heart failure or renal failure.[55,56] Although CPAP therapy has been recommended for treatment of Cheyne-Stokes respiration related to congestive heart failure, evidence to support its use in stroke-related Cheyne-Stokes respiration is lacking.[56]

Management of stroke-related breathing disorders requires a structured approach, with careful monitoring of respiratory status and gas exchange. Patients with acute hypoxemia should receive oxygen supplementation to maintain oxygen saturation >94%, taking care

to avoid oxygen toxicity and hypercapnia.[7] It is crucial to treat contributory causes of respiratory decompensation such as pulmonary edema, pneumonia, or atelectasis. Some patients may respond to proper positioning, including elevation of the head of the bed to enhance lung expansion and reduce aspiration risk. Chest physiotherapy may help mobilize secretions and prevent atelectasis. The efficacy of these measures in preventing poststroke pulmonary complications requires further study.

GASTROINTESTINAL COMPLICATIONS

Dysphagia

Swallowing impairments are common after stroke, increasing the risk of pneumonia and malnutrition while diminishing the quality of life for stroke survivors.[57] Effective therapy for stroke-related dysphagia is lacking, and current management revolves around early identification, dietary modification, or nutritional support through alternative means.[7–10,58]

Dysphagia screening (DS) is an initial assessment to determine the need for a comprehensive assessment by a speech-language pathologist before oral feeds are initiated.[10,59] Failing poststroke DS is associated with a significant risk of pneumonia, institutionalization, and mortality, although the effectiveness of DS in preventing these outcomes has not been established in a sufficiently powered RCT.[60,61] However, bundling DS with fever and glucose management in the QASC trial reduced death and dependency in hospitalized patients with stroke.[6] Current stroke guidelines recommend DS in all patients with stroke before the initiation of oral feeds or medications.[7–9]

A reliable DS tool should be easy to use by frontline clinicians and align with clinical practice in real-world context.[59] Various DS instruments have been developed and tested in stroke population, but no single tool stands out as superior.[62] Factors such as diagnostic accuracy, ease of use, and available resources should guide the choice of the screening instrument. Use of an internally developed, nonvalidated DS tool is discouraged. DS should be conducted as soon as possible after hospital admission, with rescreening if there is a change in patient's neurological status.[10] In most hospital settings, DS is performed by a nurse, although other clinicians can also conduct these assessments. All personnel involved in DS should receive adequate training in tool use and recognizing dysphagia signs. Patients who fail DS require a formal dysphagia assessment by a speech-language pathologist, who uses their clinical discretion to pursue a bedside clinical evaluation or an instrumental assessment. Patient and family education about the risks of stroke-related dysphagia and need for dietary modification or nothing-by-mouth status can help manage expectations and alleviate distress associated with dietary restriction.

Nutritional Support

The current AIS guidelines on nutritional support after stroke are informed largely by the multicenter FOOD trial (Feed or Ordinary Diet) and recommend initiating a diet for all patients with stroke within 7 days of hospital admission.[7,63] They also recommend starting nasogastric feeds within a week after stroke for patients unable to safely swallow, with a transition to percutaneous gastrostomy (PEG) tubes for those anticipated to have prolonged dysphagia (>2–3 weeks).[7] Nutritional supplementation is advisable for patients with stroke who are at risk of malnutrition, especially those with swallowing impairment.[7] However, there is a need for further research to develop personalized nutritional support strategies after stroke.

Dysphagia in many patients with stroke improves by hospital discharge, although approximately one-third may have dysphagia at 1 month.[64] Predictive instruments have been developed to help estimate individual recovery trajectories for dysphagia after AIS, but their role in guiding management decisions such as PEG placement remains untested.[64] Many patients with stroke who undergo PEG placement have severe strokes with enduring disabilities, and it is uncertain whether PEG feeding improves their overall outcomes. In the FOOD trial, PEG placement was associated with increased risk of poor outcomes, and other observational data show significantly higher rates of institutionalization, pneumonia, sepsis, and death in these patients.[63,65]

We suggest a consensus-driven approach for decisions on PEG placement because of its profound impact on the patient's quality of life and uncertain clinical benefit. Shared decision-making, involving patients or their surrogates and clinicians, is essential, prioritizing the patient's preferences and values to preserve their dignity and control. A recently developed prognosticating tool can supplement clinical judgment in assessing overall clinical recovery and mortality after PEG placement, assisting in more informed decision-making.[66] Acknowledging cognitive biases and uncertainties in prognostication is important for clinicians, and further research is needed to improve shared decision-making on PEG feeding after stroke.

Gastrointestinal Bleeding

Gastrointestinal bleeding (GIB) is relatively uncommon after an acute stroke, with a reported incidence of 1.3% to 5% (Supplemental Table 1B), but exerts a significant impact on stroke care and outcomes.[67,68] It can lead to hemodynamic changes or withdrawal of antiplatelet or anticoagulant therapy or induce a procoagulant state, increasing the risk of infarct expansion, stroke recurrence, or VTE.

Retrospective analysis from a large cohort showed that GIB in patients with AIS was associated with more

than tripling of the risk for recurrent in-hospital stroke, death, and severe disability.[67] In another sizeable prospective study, GIB increased the risk of a recurrent stroke by 1.5-fold at 3 months.[68] Potential causes for poststroke GIB include peptic ulcers, nasogastric tube trauma, gastrointestinal malignancy, angiodysplasia, ischemic colitis, or Mallory-Weiss tear from recurrent vomiting.[63,67,68] Preexisting peptic ulcer disease poses the strongest risk for GIB in these patients.[67,68]

Nurses play a crucial role in early GIB detection, which can present with overt hematemesis, melena, or hematochezia with hypotension or may be occult. Ensuring hemodynamic stability with volume resuscitation is the first priority.[69] Transfusion decisions are individualized and can be guided by hemodynamic parameters and the presence of ongoing bleeding.[69] In hemodynamically stable patients with stroke, a hemoglobin target between 7 and 8 g/dL may be appropriate but requires further study.[70] Proton pump inhibitors are used in confirmed or suspected cases of upper GIB.[71]

Determining whether to discontinue anticoagulant therapy or reverse its effects needs to be tailored according to the risk for stroke recurrence and GIB. The American College of Gastroenterology provides conditional recommendations against withholding aspirin monotherapy in patients with acute GIB and temporary discontinuation of P2Y12 receptor inhibitors in patients on dual antiplatelets[72]; however, the risks of withholding dual antiplatelets should be carefully considered in patients at high risk for thrombosis such as those with recent arterial stent placement. The timing for safe resumption of anticoagulant therapy in patients with AIS with GIB is currently unclear.

Management of poststroke GIB requires a multidisciplinary approach. A gastroenterology evaluation is important to determine the need and timing for additional investigations. The American College of Gastroenterology recommends early endoscopy (within 24 hours) for most patients with acute upper GIB and a colonoscopy within 24 to 36 hours for suspected lower GIB.[72] Despite some procedural risks, endoscopy appears safe in the early aftermath of a stroke and may reduce mortality in patients with stroke with acute GIB.[73]

Constipation and Diarrhea

Constipation and diarrhea are common after a stroke. Constipation is more frequent, likely because of immobility, diet changes, medications, and impaired neuronal modulation of colonic motility after stroke.[74] Nurses play a key role in management by monitoring stool consistency and frequency.[10] Encouraging mobility, avoiding medications with anticholinergic properties, and using stool softeners are some commonly advocated strategies.[10] Structured bowel programs and nurse-led interventions in bowel care can be effective in improving bowel evacuation in these patients.[75,76] Hospitalized patients with stroke are also susceptible to nosocomial diarrhea. Causes include certain medications, infections, and enteral feeding. Presence of fever, lower abdominal pain/cramping, and leukocytosis are suggestive of an infectious colitis. Hospitalized patients exposed to antibiotics are predisposed to *Clostridioides difficile* colitis. Severe diarrhea may lead to volume depletion and electrolyte abnormalities, but its impact on clinical stroke recovery is unknown.

Fecal Incontinence

Fecal incontinence (FI) is a common, distressing symptom for patients with stroke and often coexists with urinary incontinence.[77,78] In a prospective observational cohort, the prevalence of new-onset poststroke FI was 30% at 7 to 10 days and 11% at 3 months and 1 year.[78] Factors such as difficulty accessing the toilet, anticholinergic medications, cognitive impairment, diarrhea, and constipation can contribute to FI after stroke.[78]

Currently, there is no evidence to support routine testing with anorectal manometry or ultrasound in managing poststroke FI. Instead, a structured review of fluid, diet, and medications to identify contributory factors of FI may be effective.[75] Treating concomitant diarrhea, constipation, and fecal impaction is important, along with maintaining perianal skin hygiene and using barrier creams to prevent skin breakdown. Biofeedback training and pelvic floor rehabilitation in hospitalized patients with stroke require further exploration. FI is often an unseen problem in stroke survivors. Raising awareness about FI in caregivers and screening all patients with stroke for FI constitute important aspects of stroke care (Figure 3).

RENAL AND URINARY COMPLICATIONS

Acute Kidney Injury

Kidney disease and stroke share common risk factors, and many patients with acute stroke have known or newly detected kidney disease.[79] Acute kidney injury (AKI) has a reported pooled incidence of almost 20% after stroke.[80] Although only a minority of patients with stroke with AKI require renal replacement therapy, poststroke AKI is independently associated with increased mortality and disability, cardiac complications, and extended hospitalization.[80,81] Risk factors for AKI include preexisting renal disease, older age, higher stroke severity, congestive heart failure, and ischemic heart disease.[80]

The use of intravenous iodinated contrast for stroke imaging (computed tomography angiograms and perfusion studies) has a low incidence of causing contrast-associated AKI.[79,82] However, preexisting renal impairment can increase the risk of developing contrast-associated AKI after endovascular thrombectomy.[83]

 Stroke. 2024;55:00–00. DOI: 10.1161/STR.0000000000000477

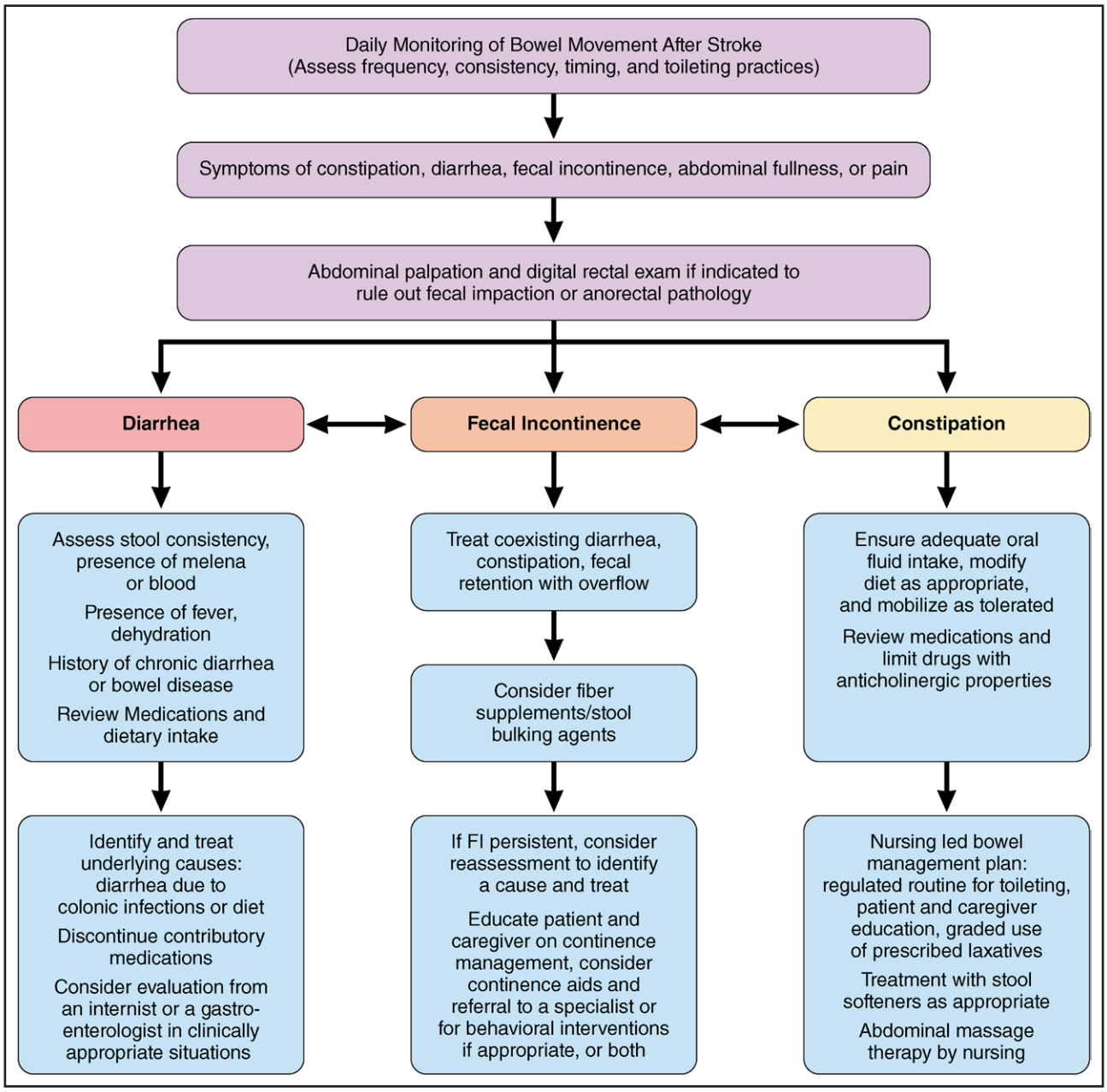

Figure 3. A suggested approach to dysfunction of bowel motility and FI in hospitalized patients with stroke.
FI indicates fecal incontinence.

AKI may also lead to a contrast-associated encephalopathy after dye administration.[84] The risks of contrast-associated AKI should be decided on a case-by-case basis to support critical decision-making. Volume expansion with either isotonic sodium chloride or sodium bicarbonate is recommended in patients at increased risk for contrast-associated AKI.[85] Other potential risks for developing poststroke AKI include the use of nephrotoxic drugs and osmotherapeutic agents (hypertonic saline and mannitol).[82,83,85]

Dedicated studies that can guide management of AKI after stroke are lacking. Existing guidelines for AKI management recommend monitoring serum creatinine and urine output, identifying underlying causes, avoiding nephrotoxic agents, and using isotonic crystalloids for intravascular volume expansion in at-risk patients or patient with AKI.[82,85] Caution is advised with blood pressure reduction in patients with acute ICH because rapid lowering can precipitate acute renal failure.[86] A suggested approach to managing AKI is outlined in Figure 4.

Urinary Incontinence and Retention

Urinary incontinence can emerge as a new symptom after a stroke or exacerbate preexisting difficulties with urinary control, affecting nearly half of all patients with

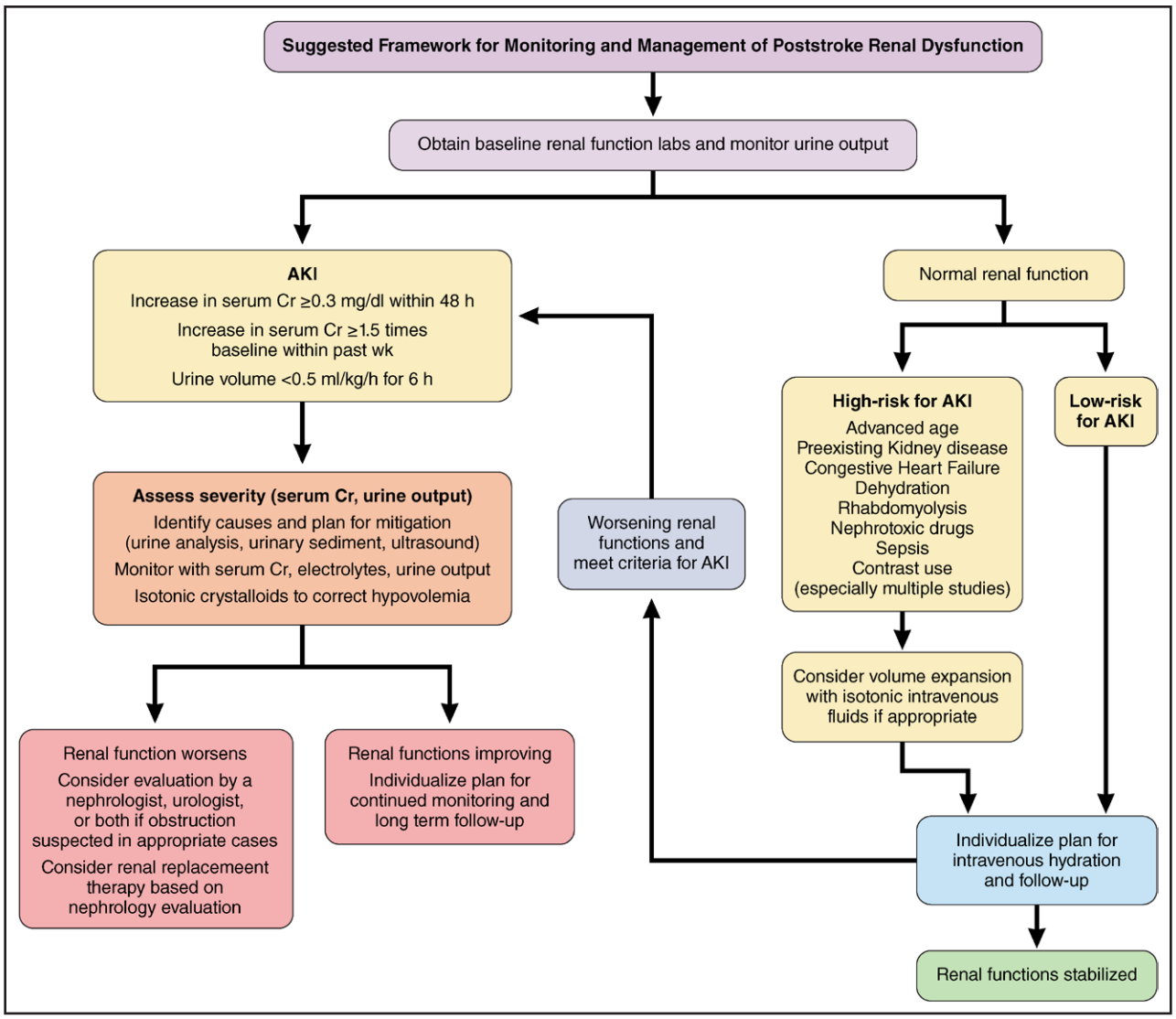

Figure 4. Suggested framework for screening, diagnosis, and management of AKI after stroke.
AKI indicates acute kidney injury; and Cr, creatinine.

acute stroke.[77,87] Although poststroke urinary incontinence improves over time, some patients have enduring difficulties with bladder control.[77,87]

Impaired sensorium or cognitive deficits from stroke may diminish bladder sensations or control and hinder independent toileting. Medications (anticholinergics, opioids), concurrent autonomic neuropathy or lumbosacral polyradiculopathy, and urinary infections can also promote urinary retention.

We suggest screening every hospitalized patient with stroke for urinary incontinence and retention. A bladder scan revealing a postvoid residual >150 cm^3 indicates incomplete emptying. Detailed assessment should explore cognitive awareness, communication abilities, and access to toileting facilities to individualize management plans.[10] Indwelling catheters are best avoided to prevent catheter-associated UTI.[10,42] When needed, catheter insertion must be aseptic and involve proper catheter care. Urodynamic studies and medications such as antimuscarinic agents or α-blockers offer limited benefit in these patients.[88]

Behavioral techniques such as bladder training and nurse-driven interventions such as scheduled voiding with postvoid residual monitoring and intermittent catheterization can be effective.[10,42] Strategies to enhance cognitive awareness and early mobilization, improve access to a toilet, and enhance patient/caregiver education may also be helpful.

TRANSITIONS OF CARE

The importance of care transitions for stroke survivors has been highlighted in previous statements.[10,89] Many poststroke systemic complications require ongoing care

to ensure their appropriate management and resolution that is based on close coordination among clinicians, patients, and caregivers. Interdisciplinary teams that may include internists, neurologists, nurses, trained rehabilitation specialists, and social workers with access to other specialists and health care infrastructure are prerequisites for these efforts. However, major fissures in real-world clinical care make it uniquely challenging for stroke survivors, especially those with enduring disabilities, who are more likely to have developed complications, to access timely care. Poststroke disability, communication and cognitive difficulties, and transportation issues are some obvious hurdles for these patients. Similarly, variations in discharge destinations (home, acute rehabilitation facility, skilled nursing facility, nursing home), availability of trained staff and care infrastructure, caregiver support, access to specialists, food and housing insecurities, and financial resources can be major impediments to receiving appropriate care.

Only a few studies have systematically evaluated posthospitalization stroke care coordination models in real-life practice.[90,91] Further research is needed to develop efficient models for care transitions, taking into consideration patient needs, social contexts, and their unique vulnerabilities. The role of pragmatic innovative strategies using interdisciplinary teams, home visits, and telemedicine requires further evaluation for mitigating the effects of poststroke systemic complications and improving clinical outcomes and patients' sense of well-being.

CONCLUSIONS AND FUTURE DIRECTIONS

Systemic complications that emerge in the early aftermath of a stroke contribute disproportionately to poorer patient outcomes and strain health care resources. This document has been composed with the intent of promoting coherent, system-based assessment and care of poststroke complications. Although it is not a comprehensive compilation of all possible poststroke systemic complications, it identifies areas for further research and offers suggestions for care. There is a clear need to develop new interventions and strategies for mitigating these events that require further evaluation in well-designed studies. Interdisciplinary teams including physicians and nonphysician practitioners with experience in stroke, rehabilitation, and hospital medicine; neuroscience nurses; and trained rehabilitation specialists are critically important in the identification and management of poststroke complications. Formation of such teams is necessary to bridge knowledge gaps and fragmentation of care and to inform real-life practice. These endeavors should be coupled with implementation strategies that facilitate their uptake in diverse clinical settings. Collectively, these efforts can be channeled to design better systems of care and development of standard-of-care metrics for hospitalized patients with stroke.

ARTICLE INFORMATION

The American Heart Association makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest.

This statement was approved by the American Heart Association Science Advisory and Coordinating Committee on August 27, 2024, and the American Heart Association Executive Committee on September 23, 2024. A copy of the document is available at https://professional.heart.org/statements by using either "Search for Guidelines & Statements" or the "Browse by Topic" area. To purchase additional reprints, call 215-356-2721 or email Meredith.Edelman@wolterskluwer.com

The American Heart Association requests that this document be cited as follows: Kumar S, Chou SH-Y, Smith CJ, Nallaparaju A, Laurido-Soto OJ, Leonard AD, Singla AK, Leonhardt-Caprio A, Stein DJ; on behalf of the American Heart Association Stroke Council; Council on Cardiovascular and Stroke Nursing; and Council on Hypertension. Addressing systemic complications of acute stroke: a scientific statement from the American Heart Association. *Stroke.* 2024;55:e•••–e•••. doi: 10.1161/STR.0000000000000477

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Disclosures

Writing Group Disclosures

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This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $5000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $5000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

†Significant.

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This table represents the relationships of reviewers that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all reviewers are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $5000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $5000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Significant.

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