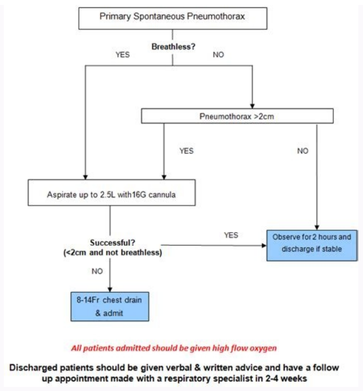
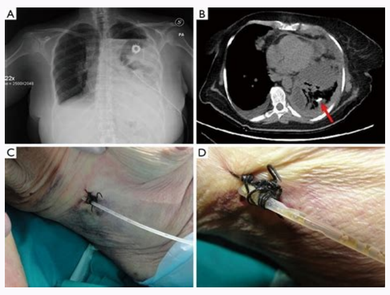
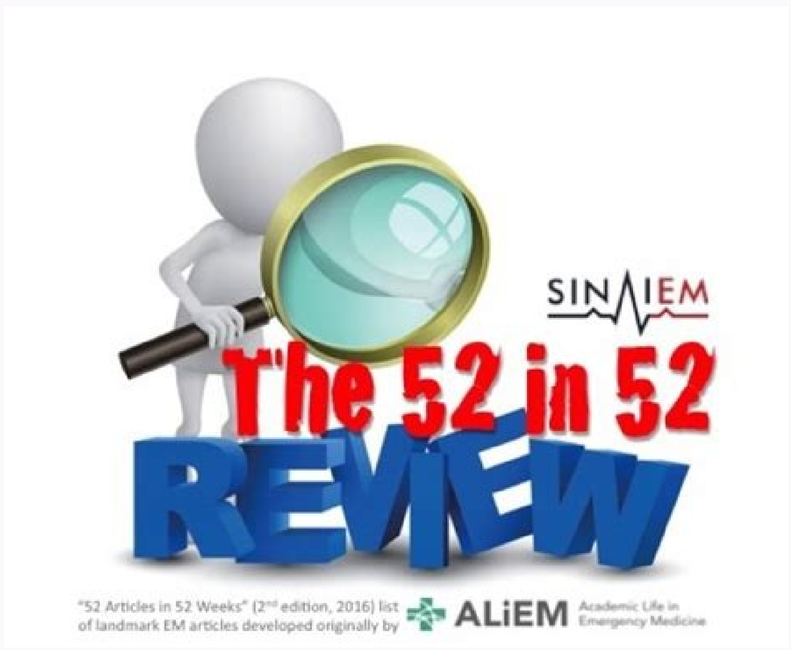
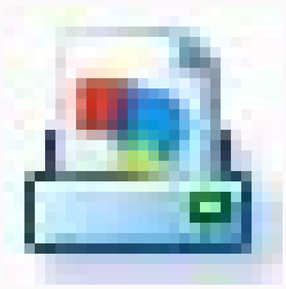
Nice guidelines chest drain management









Nice guidelines on fluid management. Chest drain nice guidelines.

The organization of health care systems is increasingly recognized as a key component of optimal stroke care. This guideline recommends development of regional systems that provide initial intracerebral hemorrhage (ICH) care and the capacity, when appropriate, for rapid transfer to facilities with neurocritical care and neurosurgical capabilities.Hematoma expansion is associated with worse ICH outcome. There is now a range of neuroimaging markers that, along with clinical markers such as time since stroke onset and use of antithrombotic agents, help to predict the risk of hematoma expansion. These neuroimaging markers include signs detectable by noncontrast computed tomography, the most widely used neuroimaging modality for ICH.ICHs, like other forms of stroke, occur as the consequence of a defined set of vascular pathologies. This guideline emphasizes the importance of, and approaches to, identifying markers of both microvascular and macrovascular hemorrhage pathogeneses.When implementing acute blood pressure lowering after mild to moderate ICH, treatment regimens that limit blood pressure variability and achieve smooth, sustained blood pressure control appear to reduce hematoma expansion and yield better functional outcome.ICH while anticoagulated has extremely high mortality and morbidity. This guideline provides updated recommendations for acute reversal of anticoagulation after ICH, highlighting use of protein complex concentrate for reversal of vitamin K antagonists such as warfarin, idarucizumab for reversal of the thrombin inhibitor dabigatran, and andexanet alfa for reversal of factor Xa inhibitors such as rivaroxaban, apixaban, and edoxaban.Several in-hospital therapies that have historically been used to treat patients with ICH appear to confer either no benefit or harm. For emergency or critical care treatment of ICH, prophylactic corticosteroids or continuous hyperosmolar therapy appears to have no benefit for outcome, whereas the use of platelet transfusions outside the setting of emergency surgery or severe thrombocytopenia appears to worsen outcome. Similar considerations apply to some prophylactic treatments historically used to prevent medical complications after ICH. Use of graduated knee- or thigh-high compression stockings alone is not an effective prophylactic therapy for prevention of deep vein thrombosis, and prophylactic antiseizure medications in the absence of evidence for seizures do not improve long-term seizure control or functional outcome.Minimally invasive approaches for evacuation of supratentorial ICHs and intraventricular hemorrhages, compared with medical management alone, have demonstrated reductions in mortality. The clinical trial evidence for improvement of functional outcome with these procedures is neutral, however. For patients with cerebellar hemorrhage, indications for immediate surgical evacuation with or without an external ventricular drain to reduce mortality now include larger volume (>15 mL) in addition to previously recommended indications of neurological deterioration, brainstem compression, and hydrocephalus.The decision of when and how to limit life-sustaining treatments after ICH remains complex and highly dependent on individual preference. This guideline emphasizes that the decision to assign do not attempt resuscitation status is entirely distinct from the decision to limit other medical and surgical interventions and should not be used to do so. On the other hand, the decision to implement an intervention should be shared between the physician and patient or surrogate and should reflect the patient's wishes as best as can be discerned. Baseline severity scales can be useful to provide an overall measure of hemorrhage severity but should not be used as the sole basis for limiting life-sustaining treatments.Rehabilitation and recovery are important determinants of ICH outcome and quality of life. This guideline recommends use of coordinated multidisciplinary inpatient team care with early assessment of discharge planning and a goal of early supported discharge for mild to moderate ICH. Implementation of rehabilitation activities such as stretching and functional task training may be considered 24 to 48 hours after moderate ICH; however, early aggressive mobilization within the first 24 hours after ICH appears to worsen 14-day mortality. Multiple randomized trials did not confirm an earlier suggestion that fluoxetine might improve functional recovery after ICH. Fluoxetine reduced depression in these trials but also increased the incidence of fractures.A key and sometimes overlooked member of the ICH care team is the patient's home caregiver. This guideline recommends psychosocial education, practical support, and training for the caregiver to improve the patient's balance, activity level, and overall quality of life.Since 1990, the American Heart Association (AHA)/American Stroke Association (ASA) has translated scientific evidence into clinical practice guidelines with recommendations to improve cerebrovascular health. These guidelines, which are based on systematic methods to evaluate and classify evidence, provide a foundation for the delivery of quality cerebrovascular care. The AHA/ASA sponsors the development and publication of clinical practice guidelines without commercial support, and members volunteer their time to the writing and review efforts.Clinical practice guidelines for stroke provide recommendations applicable to patients with or at risk of developing cerebrovascular disease. The focus is on medical practice in the United States, but many aspects are relevant to patients throughout the world. Although it must be acknowledged that guidelines may be used to inform regulatory or payer decisions, the core intent is to improve quality of care and align with patients' interests. Guidelines are intended to define practices meeting the needs of patients in most, but not all, circumstances and should not replace clinical judgment; furthermore, the recommendations set forth should be considered in the context of individual patient values, preferences, and associated conditions.The AHA/ASA strives to ensure that guideline writing groups contain requisite expertise and are representative of the broader medical community by selecting experts from a broad array of backgrounds, representing different sexes, races, ethnicities, intellectual perspectives, geographic regions, and scopes of clinical practice and by inviting organizations and professional societies with related interests and expertise to participate as endorsers. The AHA/ASA has rigorous policies and methods for development of guidelines that limit bias and prevent improper influence. The complete policy on relationships with industry and other entities (RWI) can be found at in 2017, numerous modifications to AHA/ASA guidelines have been implemented to make guidelines shorter and enhance user-friendliness. Guidelines are written and presented in a modular knowledge chunk format; each chunk includes a table of recommendations, a brief synopsis, recommendation-specific supportive text, and, when appropriate, flow diagrams or additional tables. Hyperlinked references are provided to facilitate quick access and review. Other modifications to the guidelines include the addition of Knowledge Gaps and Future Research segments in some sections and a web guideline supplement (Online Data Supplement) for useful but noncritical tables and figures.Joseph P. Broderick, MD, FAHAChair, AHA Stroke Council Scientific Statement Oversight Committee1. IntroductionApproximately 10% of the 795 000 strokes per year in the United States are intracerebral hemorrhages (ICHs),1 defined by brain injury attributable to acute blood extravasation into the brain parenchyma from a ruptured cerebral blood vessel. The clinical impact of ICH appears disproportionately high among lower-resource populations both in the United States and internationally. In US-based studies, ICH incidence has been reported to be ≈1.6-fold greater among Black than White people2 and 1.6-fold greater among Mexican American than non-Hispanic White people.3 Internationally, ICH incidence is substantially higher in low- and middle-income versus high-income countries, both as a proportion of all strokes and in absolute incidence rates.4,5Several additional features of ICH make it a greater public health threat than conveyed by incidence numbers alone. ICH is arguably the deadliest form of acute stroke, with early-term mortality about 30% to 40% and no or minimal trend toward improvement over more recent time epochs.6–9 Incidence of ICH increases sharply with age and is therefore expected to remain substantial as the population ages, even with counterbalancing public health improvements in blood pressure (BP) control.8 Another growing source of ICH is more widespread use of anticoagulants,10 a trend likely to counterbalance the reduced ICH risk associated with increasing prescription of direct oral anticoagulants (DOACs) relative to vitamin K antagonists (VKAs).11ICH thus remains in need of novel treatments and improved application of established approaches for every aspect of the disease: primary and secondary prevention, acute inpatient care, and poststroke rehabilitation and recovery. This guideline seeks to synthesize data in the ICH field into practical recommendations for clinical practice.1.1. Methodology and Evidence ReviewThe recommendations listed in this guideline are, whenever possible, evidence based and supported by extensive evidence review. A search for literature derived from research principally involving human subjects, published in English, and indexed in MEDLINE, PubMed, Cochrane Library, and other selected databases relevant to this guideline was conducted between October 2020 and March 2021. Additional trials published between March 2021 and November 2021 that affected the content, Class of Recommendation (COR), or Level of Evidence (LOE) of a recommendation were included when appropriate. For specific search terms used, readers are referred to the Online Data Supplement, which contains the final evidence tables summarizing the evidence used by the guideline writing group to formulate recommendations. In addition, the guideline writing group reviewed documents related to subject matter previously published by the AHA/ASA. References selected and published in the present document are representative and not all inclusive.Each topic area was assigned a primary writer and a primary and sometimes secondary reviewer. Author assignments were based on the areas of expertise of the members of the guideline writing group and their lack of any RWI related to the section material. All recommendations were fully reviewed and discussed among the full guideline writing group to allow diverse perspectives and considerations for this guideline. Recommendations were then voted on, and a modified Delphi process was used to reach consensus. Guideline writing group members who had RWI that were relevant to certain recommendations were recused from voting on those particular recommendations. All recommendations in this guideline were agreed to by between 88.9% and 100% of the voting guideline writing group members.1.2. Organization of the Writing GroupThe guideline writing group consisted of vascular neurologists, neurocritical care specialists, neurological surgeons, an emergency physician, a hematologist, a rehabilitation medicine physician, a board-certified acute care nurse practitioner, a fellow-in-training, and a lay/patient representative. The writing group included representatives from the AHA/ASA, the American Association of Neurological Surgeons/Congress of Neurological Surgeons, and the American Academy of Neurology. Appendix 1 of this document lists guideline writing group members' relevant RWI and other entities. For the purposes of full transparency, the guideline writing group members' comprehensive disclosure information is available online.1.3. Document Review and ApprovalThis document was reviewed by the AHA Stroke Council Scientific Statement Oversight Committee, the AHA Science Advisory and Coordinating Committee, and the AHA Executive Committee; reviewers from the American Academy of Neurology, the Society of Vascular and Interventional Neurology, and the American Association of Neurological Surgeons/Congress of Neurological Surgeons; and 53 individual content reviewers. Appendix 2 lists reviewers' comprehensive disclosure information.1.4. Scope of the GuidelineThis guideline addresses the diagnosis, treatment, and prevention of ICH in adults and is intended to update and replace the AHA/ASA 2015 ICH guideline.12 This 2022 guideline is limited explicitly to spontaneous ICHs that are not caused by head trauma and do not have a visualized structural cause such as vascular malformation, saccular aneurysm, or hemorrhage-prone neoplasm. These hemorrhages without a demonstrated structural or traumatic cause are often referred to as primary ICH (see further comment on this terminology in Section 2.1, Small Vessel Disease Types). This guideline thus does not overlap with AHA/ASA guidelines or scientific statements on the treatment of arteriovenous malformations,13 aneurysmal subarachnoid hemorrhage,14 or unruptured saccular aneurysms.13,15 This guideline does, however, address imaging approaches to ICH that help differentiate primary ICH from these secondary causes.This guideline aims to cover the full course of primary ICH (Figure 1), from the location and organization of emergency care (Section 3), initial diagnosis and assessment (Section 4), and acute medical and surgical interventions (Sections 5.1, 5.2, and 6) to further inpatient care of post-ICH complications (Sections 5.3–5.5), goals of care assessment (Section 7), rehabilitation and recovery (Section 8), and secondary prevention of recurrent ICH (Section 9). Because of the substantial differences in pathogenesis and course between ICH and ischemic stroke, the writing group sought, when possible, to base its recommendations on data derived specifically from ICH patient groups. Some aspects of inpatient medical care and post-ICH rehabilitation are likely to be similar between patients with ICH and patients with ischemic stroke, however. Readers are therefore referred to relevant AHA/ASA guidelines and scientific statements for ischemic stroke in these overlapping areas.16,17Table 1 is a list of associated AHA/ASA guidelines and scientific statements that may be of interest to the reader.Table 1. Associated AHA/ASA Guidelines and StatementsTitleOrganizationPublication yearAHA/ASA guidelines 2021 Guideline for the Prevention of Stroke in Patients With Stroke and Transient Ischemic Attack: A Guideline From the American Heart Association/American Stroke AssociationAHA/ASA2021 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice GuidelinesACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA2017 Guidelines for Adult Stroke Rehabilitation and Recovery: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke AssociationAHA/ASA2016 Guidelines for the Management of Patients With Unruptured Intracranial Aneurysms: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke AssociationAHA/ASA2015 Guidelines for the Management of Spontaneous Intracerebral Hemorrhage: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke AssociationAHA/ASA2015 Guidelines for the Primary Prevention of Stroke: A Statement for Healthcare Professionals From the American Heart Association/American Stroke AssociationAHA/ASA2014 Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage: A Guideline for Healthcare Professionals From the American Heart Association/American Stroke AssociationAHA/ASA2012AHA/ASA scientific statements Care of the Patient With Acute Ischemic Stroke (Prehospital and Acute Phase of Care): Update to the 2009 Comprehensive Nursing Care Scientific Statement: A Scientific Statement From the American Heart AssociationAHA/ASA2021 Management of Brain Arteriovenous Malformations: A Scientific Statement for Healthcare Professionals From the American Heart Association/American Stroke AssociationAHA/ASA2017 Prevention of Stroke in Patients With Silent Cerebrovascular Disease: A Scientific Statement for Healthcare Professionals From the American Heart Association/American Stroke AssociationAHA/ASA2017 Palliative and End-of-Life Care in Stroke: A Statement for Healthcare Professionals From the American Heart Association/American Stroke AssociationAHA/ASA2014Figure 1. Guideline overview for primary ICH. ICH indicates intracerebral hemorrhage. Recommendations on the topics above can be found in the guideline in the sections indicated: *Sections 3 and 5. †Section 4. ‡Sections 5 and 6. §Section 7. ‖Section 5. #Section 8. **Section 9.Another area where this ICH guideline interfaces with prior ischemic stroke guidelines is the challenging area of antithrombotic agent use in patients after ICH who are at risk for both recurrent ICH and ischemic stroke (Section 9.1.3, Management of Antithrombotic Agents). This guideline does not attempt to reassess the extensive literature on assessment of future ischemic stroke risk and instead refers the reader to existing AHA guidelines on primary and secondary ischemic stroke prevention.18,19This ICH guideline has a new section on assessment of ICH risk in individuals with no prior ICH but with neuroimaging findings such as cerebral microbleeds or cortical superficial siderosis suggestive of a hemorrhage-prone microvasculopathy. This topic, which was also previously discussed in an AHA scientific statement on the wider area of silent cerebrovascular disease,20 does not fall strictly under the heading of ICH management. This guideline writing group nonetheless included the section (9.2, Primary ICH Prevention in Individuals With High-Risk Imaging Findings) because of its close relationship to the considerations used for secondary prevention of recurrent ICH (Section 9.1, Secondary Prevention) and the high frequency with which these small hemorrhagic lesions are detected as incidental findings on magnetic resonance imaging (MRI) performed for other indications. Evidence on how to interpret and act on incidental hemorrhagic lesions remains limited but is likely to grow with the widespread incorporation of blood-sensitive MRI methods into research studies and clinical practice.1.5. COR and LOERecommendations are designated with both a COR and an LOE. The COR indicates the strength of recommendation, encompassing the estimated magnitude and certainty of benefit in proportion to risk. The LOE rates the quality of scientific evidence supporting the intervention on the basis of the type, quantity, and consistency of data from clinical trials and other sources (Table 2).Table 2. Applying Class of Recommendation and Level of Evidence to Clinical Strategies, Interventions, Treatments, or Diagnostic Testing in Patient Care (Updated May 2019)*AbbreviationsAbbreviationMeaning/PhraseADLactivities of daily livingAFatrial fibrillationAHAAmerican Heart AssociationaPCCactivated prothrombin complex concentrateASAAmerican Stroke AssociationATACH-2Antihypertensive Treatment of Acute Cerebral Hemorrhage IIAVERTA Very Early Rehabilitation TrialBPblood pressureCAAcerebral amyloid angiopathyCLEAR IIIClot Lysis: Evaluating Accelerated Resolution of Intraventricular Hemorrhage Phase IIICLOTSClots in Legs or Stockings After StrokeCORClass of RecommendationCPPcerebral perfusion pressureCTcomputed tomographyCTAcomputed tomography angiographyDBPdiastolic blood pressureDIAGRAMDiagnostic Angiography to Find Vascular MalformationsDNARdo not attempt resuscitationDOACdirect oral anticoagulantDSAdigital subtraction angiographyDVTdeep vein thrombosisEDemergency departmentEIBPLearly intensive blood pressure loweringEMSemergency medical servicesERICHEthnic/Racial Variations of Intracerebral HemorrhageEVDexternal ventricular drain/drainageFFPfresh-frozen plasma4-F PCC4-factor prothrombin complex concentrateGCSGlasgow Coma ScaleHEhematoma expansionHRhazard ratioICHintracerebral hemorrhageICPintracranial pressureICUintensive care unitINCHInternational Normalized Ratio (INR) Normalization in Coumadin Associated Intracerebral HemorrhageINRinternational normalized ratioINTERACT2The Second Intensive Blood Pressure Reduction in Acute Cerebral Hemorrhage TrialIPCintermittent pneumatic compressionIVCinferior vena cavaIVHintraventricular hemorrhageIVTintraventricular thrombolysisLMWHlow-molecular-weight heparinLOELevel of EvidenceLOSlength of stayLVADleft ventricular assist deviceMISminimally invasive surgeryMISTIE IIIMinimally Invasive Surgery Plus rt-PA for Intracerebral Hemorrhage EvacuationMRAmagnetic resonance angiographyMRImagnetic resonance imagingmRSmodified Rankin ScaleMSUmobile stroke unitNCCTnoncontrast computed tomographyNDneurological deteriorationNICE-SUGARNormoglycemia in Intensive Care Evaluation and Survival Using Glucose Algorithm RegulationNIHSSNational Institutes of Health Stroke ScaleNSAIDnonsteroidal anti-inflammatory drugORodds ratioPCCprothrombin complex concentratePEpulmonary embolismPREVAILEvaluation of the WATCHMAN Left Atrial Appendage [LAA] Closure Device in Patients With Atrial Fibrillation Versus Long Term Warfarin TherapyPRoFESSPrevention Regimen for Effectively Avoiding Second StrokesPROGRESSPerindopril Protection Against Recurrent Stroke StudyPROTECT-AFWATCHMAN Left Atrial Appendage System for Embolic Protection in Patients With Atrial FibrillationQASCQuality in Acute Stroke CareRCTrandomized controlled trialRRTrenal replacement therapyRWIrelationships with industry and other entitiesSAEserious adverse eventSBPsystolic blood pressureSPARCLStroke Prevention by Aggressive Reduction in Cholesterol LevelsSSRIsselective serotonin reuptake inhibitorsSTICHSurgical Trial in Intracerebral HemorrhageTBItraumatic brain injuryTXAtranexamic acidUFHunfractionated heparinVKAvitamin K antagonistVTEvenous thromboembolism2. General Concepts2.1. Small Vessel Disease TypesDespite our use of the term primary ICH to distinguish from ICH with a demonstrated structural cause (Section 1.4, Scope of the Guideline), these seemingly spontaneous hemorrhages are not truly primary but rather represent the consequence of defined underlying (and often co-occurring) vascular pathologies. The 2 common cerebral small vessel pathologies that account for the overwhelming majority of primary ICH are arteriolosclerosis and cerebral amyloid angiopathy (CAA). Each is a common age-related pathology, appearing at autopsy at moderate to severe extents in 30% to 35% of individuals enrolled in a longitudinal study of aging.21 Arteriolosclerosis (also referred to as lipohyalinosis) is detected as concentric hyalinized vascular wall thickening favoring the penetrating arterioles of the basal ganglia, thalamus, brainstem, and deep cerebellar nuclei (collectively referred to as deep territories). Its major associated risk factors are hypertension, diabetes, and age. CAA is defined by deposition primarily of the β-amyloid peptide in the walls of arterioles and capillaries in the leptomeninges, cerebral cortex, and cerebellar hemispheres (lobar territories). The primary risk factors for CAA are age and apolipoprotein E genotypes containing the ε2 or ε4 alleles.ICH occurs in a relatively small subset of those brains with advanced arteriolosclerosis or CAA, typically in deep territories for arteriolosclerosis and lobar territories for CAA, the brain locations favored by the underlying pathologies. Small, often asymptomatic cerebral microbleeds in these compartments are substantially more common, occurring in >20% of population-based individuals >60 years of age scanned with sensitive T2*-weighted MRI methods.22,23 The presence of multiple strictly lobar ICHs, microbleeds, or cortical superficial siderosis (chronic blood products over the cerebral subpial surface) has been pathologically validated as part of the Boston criteria to detect CAA-related hemorrhage with reasonably high specificity and sensitivity.24 Microbleeds associated with arteriolosclerosis tend to occur in deep territories but can appear in lobar territories as well.The underlying small vessel types of ICH have several practical implications for the formulation of ICH guidelines. They establish a hemorrhage-prone environment in which use of antithrombotic agents creates increased risk of ICH.25 It is important to note, however, that the small vessel pathologies that underlie ICH are also associated with increased risk of ischemic stroke,26 highlighting the complexity and importance of balancing the risks versus benefits of antithrombotic treatment. Among the cerebral small vessel diseases, CAA inferred by the Boston criteria appears to confer substantially greater risk for recurrent hemorrhage than arteriolosclerosis (recurrent ICH rates in a pooled analysis of 7.39%/y after CAA-related ICH versus 1.11%/y after non–CAA-related ICH).272.2. Mechanisms for ICH-Related Brain InjuryICH is understood to injure surrounding brain tissue through the direct pressure effects of an acutely expanding mass lesion and through secondary physiological and cellular pathways triggered by the hematoma and its metabolized blood products.28 Direct pressure effects can include both local compression of immediately surrounding brain tissue and more widespread mechanical injury caused by increased intracranial pressure (ICP), hydrocephalus, or herniation. Early HE, possibly driven by mechanical shearing of surrounding vessels by the initial hematoma,29 is common and a consistent predictor of worse ICH outcome.30Secondary physiological and cellular injury mechanisms postulated to be triggered by ICH include cerebral edema, inflammation, and biochemical toxicity of blood products such as hemoglobin, iron, and thrombin.28 Although it is plausible that the underlying small vessel disease type may affect the mechanism and severity of ICH-related brain injury, there is currently no strong evidence for substantial differences between the acute course of arteriolosclerosis-related and CAA-related ICH other than differences attributable to ICH location.Several of the major medical therapies for ICH such as BP lowering and reversal of anticoagulation are aimed at limiting HE. The search for effective medical treatments for protecting tissue from secondary post-ICH injury, like the search for effective neuroprotectants for ischemic stroke, has to date been unsuccessful. Surgical hematoma evacuation through craniotomy, minimally invasive approaches, or ventriculostomy is aimed at both preventing further pressure-related injury and protecting against secondary physiological and cellular injury. One complexity that arises in the interpretation of results of surgical ICH trials is the possibility that mortality might be prevented without improvement in functional outcome, an issue addressed explicitly in the current guidelines.2.3. Limits to GeneralizabilityA key limitation that runs through all sections of this guideline is that much of the data come from high-resource countries and from more affluent demographic groups within those countries. The potential limitations of generalizability to lower-resource settings and populations noted to be disproportionately at risk of ICH (Section 1, Introduction), highlight the need for future guidelines based explicitly on data from these underserved and underrepresented groups.3. Organization of Prehospital and Initial Systems of CareRecommendations for Organization of Prehospital and Initial Systems of CareReferenced studies that support recommendations are summarized in Data Supplements 1 through 12.SynopsisMuch of the data for prehospital care and stroke systems of care are derived from studies of stroke of all types (including ICH). Furthermore, it is generally not possible for prehospital clinicians to distinguish between patients with ICH and those with other types of stroke. As a result, the recommendations for prehospital care of patients with hemorrhagic stroke are essentially identical to those recommended for any patient with stroke: early recognition, expedient transport to the most appropriate facility, and prenotification before hospital arrival to expedite the in-hospital stroke response. Although it can be difficult to measure the precise time to onset of ICH treatment, it is reasonable to infer that earlier diagnosis will be closely linked to earlier treatment. To facilitate rapid diagnosis and treatment of ICH, we recommend public health measures to educate the public, build and maintain organized systems of care, and ensure appropriate training of first responders.Recommendation-Specific Supportive TextEarly symptom recognition is essential for timely ICH care. In the United States, ≈67% of adults know the signs and symptoms of stroke and the need to call EMS; stroke knowledge increased almost 15 percentage points between 2009 and 2017.33 Public education campaigns can improve stroke knowledge,35,50 increase the use of EMS for stroke,31 and use of EMS is associated with shorter time to diagnosis.32 In the largest cluster randomized controlled study of >75 000 subjects, an educational intervention reduced time to hospital arrival in women (median, 328 minutes versus 559 minutes) but not men.34 Although some smaller studies have demonstrated modest benefits, others have shown no or only transient benefits.51–54 Knowledge of stroke warning signs varies by race, sex, ethnicity, age, education, and urbanicity,33 which may contribute to disparities in outcomes. Public education campaigns should make every attempt to address underserved groups and those with the largest opportunities to improve awareness.No existing clinical decision scale can differentiate ICH from other diseases with high sensitivity or specificity in the absence of neuroimaging. Prehospital scales such as FAST (Face, Arm, Speech, Time to call 911), LAPSS (Los Angeles Prehospital Stroke Scale), CPSS (Cincinnati Prehospital Stroke Scale), and ROSIER (Recognition of Stroke in the Emergency Room) are available and typically are validated in all stroke rather than ICH specifically.41 Differences include whether they focus on sensitivity or specificity and whether they screen for stroke severity as well as presence. For dispatch, a group found that a specific dispatch stroke assessment tool was associated with shorter time to diagnosis,37 and a clinical trial found that a dispatch stroke screen reduced time to both hospital arrival and stroke unit admission (although only 5% had ICH).36 One group analyzed ICH specifically39 and found an association between documented stroke scale use and ICH recognition. The sensitivity for ICH was 84%, and stroke scale documentation was independently associated with ICH recognition and shorter door–to–computed tomography (CT) times (20 minutes versus 47 minutes). Most studies of stroke scale use in practice inadequately account for false-negative cases, thereby likely artificially boosting performance. One group developed a clinical prediction rule to classify stroke subtypes, including ICH, in the prehospital setting; however, neither the sensitivity nor positive predictive value was published.40One group found that in a large national cohort of patients with stroke, EMS use compared with arrival to hospital by other means is independently associated with earlier emergency department (ED) arrival (adjusted odds ratio [OR], 2.00 [95% CI, 1.93–2.08]), quicker ED evaluation (adjusted OR, 1.89 [95% CI, 1.78–2.00]), and more rapid treatment for ischemic stroke (adjusted OR, 1.44 [95% CI, 1.28–1.63]).32,42 For ICH specifically, a large multicenter cohort study found that time from symptom onset to ED was 63 minutes versus 227 minutes in patients who used EMS versus those who did not use EMS, and time to hospital admission was 167 minutes versus 537 minutes.55 Thus, persistent efforts to ensure activation of the 9-1-1 or a similar emergency system by patients or other members of the public for suspected stroke are warranted.Many observational studies in patients with stroke (including both ischemic and ICH) have found that use of prehospital notification to the destination ED is associated with faster time to neuroimaging and shorter time to alteplase in ischemic stroke.56–59 For example, a large registry found that after adjustment for covariates, EMS use (with prenotification) was associated with faster door-to-CT times than both private transport and EMS without prenotification.44 In the AHA Get With The Guidelines–Stroke registry, EMS personnel provided prearrival notification to the destination ED for 67% of transported patients with stroke.43 EMS prenotification was associated with shorter door-to-imaging times and shorter symptom onset–to–needle times. One group found that for ICH, early stroke team activation was associated with faster door-to-CT times (24 minutes versus 48 minutes) and faster time to hemostatic medication when used (63 minutes versus 99 minutes).60Many regions have developed stroke systems of care and stratify hospitals according to their ability to deliver intravenous thrombolytics or endovascular therapy for ischemic stroke. Triage algorithms suggest routing patients on the basis of the results of prehospital stroke severity scales. These scales often indicate high severity in the case of ICH, which would direct patients with potential ICH preferentially to advanced stroke centers such as a comprehensive stroke center. Whether patients with ICH benefit from the higher level of care versus earlier temporizing at regional facilities remains to be seen and should be studied. One observational study found that Canadian provinces that had implemented stroke systems of care had reduced mortality for the entire cohort (including ICH, ≈10% of the cohort; adjusted incidence rate ratios, 0.85 [95% CI, 0.79–0.92]).45Most studies of MSUs have focused on time to thrombolysis for stroke, and subgroup analyses of those diagnosed with ICH are small and underpowered. One group randomized their geographic region to weeks on/off for MSU availability and found that those patients treated in MSUs had faster times from symptom onset to laboratory results and to CT.47 No MSU diagnosis of ICH (or lack of ICH) required revision during follow-up. Another study in 2 regions of Germany found similar reductions in time to CT.46 The MSU reduced the use of interfacility transfer to zero for ICH because those with ICH were taken to a comprehensive stroke center as the initial hospital. Forty-one percent of the MSU patient group and none of the standard care group received BP management in the field after diagnosis, suggesting that MSU led to earlier initiation of treatment. Issues of logistics, feasibility, and cost currently appear to restrict MSU use to certain regions and facilities, and all studies are currently underpowered to evaluate any association with clinical outcome after ICH.No clinical trials of different EMS response strategies were found to have been conducted in ICH. Some have

been published in traumatic brain injury (TBI). One large clinical trial of TBI found that in patients with Glasgow Coma Scale (GCS) score 11 000 patients with acute stroke (15% ICH) compared lying-flat position to a sitting-up position with the head elevated to at least 30° for the first 24 hours. Lying flat to improve cerebral perfusion was not associated with benefit for the primary outcome, mRS score at 90 days.The concept of enhancing brain plasticity through use of selective serotonin reuptake inhibitors (SSRIs) has been suggested by animal model studies.495 However, multiple studies of fluoxetine, in either patients with ICH or patients with stroke in general, have not shown beneficial effects on functional outcome.493–497 Patients allocated fluoxetine were less likely to develop new depression by 6 months than patients on placebo but were more prone to fractures.A trial of very early mobilization (AVERT [A Very Early Rehabilitation Trial) compared frequent, higher-dose, and very early mobilization with usual care in 2104 patients with stroke, of whom 258 (12%) had ICH.499 The intervention was defined as a standardized treatment beginning within 24 hours of stroke onset, focusing on sitting, standing, and walking and resulting in at least 3 additional out-of-bed sessions compared with usual care (increase intensity). The study included >2100 patients in 5 countries and showed that the intervention increased the risk of poor outcome at 3 months. A prespecified subanalysis in patients with ICH showed that this early and intense intervention led to an increased risk of mortality at 14 days after stroke.498Knowledge Gaps and Future ResearchAn area for future study is patients' return to work, driving, and participation in other meaningful social activities. The current literature in this area is based largely on epidemiological studies. Greater independence in ADLs, fewer neurological deficits, and better cognitive ability were the most common predictors of return to work. More studies are needed to investigate how vocational rehabilitation should be performed and the role of occupational/vocational therapy in this process.There is a knowledge gap from the professionals' side concerning sexual life after ICH, contributing to the infrequency of this topic being addressed in the conversation with patients. Many people fear returning to sexual activity after stroke. However, it seems as though intercourse increases BP only slightly (up to ≈140 mm Hg) for a short time, and then it recovers to baseline level soon after sexual activity in healthy adults.There is a lack of knowledge about physical training after ICH. For example, it is unclear how to guide people after ICH in terms of weight lifting (lifts using large muscle groups versus small, heavy lifts versus repetitive lifts) and how much and how long to raise their BP. Furthermore, it is unclear what to advise about any potential bleeding risk related to exertion when BP gets >300 mm Hg.There are insufficient data on medications to improve post-ICH functional outcome. Neurostimulants, for example, have not been studied extensively for recovery of consciousness or other recovery steps after ICH.Another emerging recovery modality that should be studied after ICH is remote video administration of rehabilitation activities (telerehabilitation).8.2. Neurobehavioral ComplicationsRecommendations for Neurobehavioral ComplicationsReferenced studies that support recommendations are summarized in Data Supplements 72 and 73.SynopsisMood disturbances and cognitive dysfunction are common consequences after ICH. Poststroke depression occurs in 20% to 25% of patients with ICH within the first year after stroke,522 and this persists over time.523 Thirty-three percent of patients with ICH experience dementia either before or after their ICH,524 and the incidence of post-ICH dementia increases over time, with 1 study showing an incidence of new-onset dementia of 14.2% at 1 year, increasing to 28.3% at 4 years.525 Another study noted 32% prevalence of cognitive impairment at 3 years after stroke.526 Analysis of neuroimaging features of patients who develop post-ICH dementia suggests underlying CAA as a contributing factor.525 Neurobehavioral complications after ICH are underrecognized by clinicians, leading to worsened long-term patient-centered outcomes such as independence and community reintegration.527 Poststroke depression is associated with increased short- and long-term mortality528–532 and poor functional outcomes532–534 and leads to greater physical limitations, which can impair rehabilitative efforts.535 Poststroke depression also can lead to suicide, which is twice as high in the first 2 years after stroke compared with the general population.536 Similarly, cognitive impairment predicts poststroke disability526,535,537 and mortality.537–539 There is also an interaction between the two: Cognitive symptoms can be caused by depression, and depression can interfere with cognitive function. Recognition and treatment of these stroke complications can have a large impact on stroke recovery.Recommendation-Specific Supportive TextPatients with poststroke depression and anxiety should be referred to a mental health professional for consideration of psychotherapy or talking-based therapy because several meta-analyses have shown a significant improvement in depression scores540,541 and remission of poststroke depression540,541 in patients who underwent psychotherapy with or without pharmacotherapy. Psychotherapy also significantly reduces poststroke anxiety.542 Pharmacological therapy is beneficial in reducing poststroke depression and anxiety prevalence and symptoms.540,542–548 Three of the randomized trials evaluating fluoxetine for motor recovery after stroke showed reductions in poststroke depression when fluoxetine was started 2 to 15 days after ischemic stroke or hemorrhagic stroke.493,496,549 Several studies suggest that transcranial magnetic stimulation also reduces symptoms of poststroke depression.544,550Validated screening tools to evaluate for depression and anxiety can lead to improved patient outcomes. One prospective RCT found a significant improvement in depression symptoms for patients with acute ischemic stroke when screening was paired with an Activate-Initiate-Monitor intervention, where Activate represents patient recognition of depression, Initiate represents antidepressant medication, and Monitor represents treatment.551 In a meta-analysis, Meader and colleagues509 evaluated the Center for Epidemiological Studies Depression Scale, Hamilton Depression Rating Scale, and Patient Health Questionnaire-9. All had optimal receiver-operating characteristics curves to detect poststroke depression and anxiety. Therefore, any of these screening tools can be used to assess for post-ICH mood disorders. Although many studies report poststroke depression during hospitalization and rehabilitation, mood disorders recur over time. For patients who developed poststroke depression, recurrence increased from 28% in year 2 to 100% by year 15.529 Although the optimal timing and frequency of depression screening are uncertain, screening should occur not only at transition points across the continuum of care (eg, hospitalization to inpatient rehabilitation) but also in the outpatient setting, especially for patients with a history of poststroke depression within the first year after ICH.529Multiple tests are available to screen for cognitive impairment. A meta-analysis compared studies evaluating the Mini-Mental State Examination, Montreal Cognitive Assessment, Rotterdam–Cambridge Cognition Examination, and Addenbrooke's Cognitive Examination–Revised and showed that all demonstrated similar accuracy to detect cognitive impairment and dementia.510 The Montreal Cognitive Assessment has a high specificity and was shown in 1 study to be the most valid and clinically feasible tool across a wide range of cognitive impairment,507 but it has a lower specificity for screening.510,552 The Depression, Obstructive Sleep Apnea, and Cognitive Impairment screening tool takes 4 microbleeds),572 and the presence of disseminated cortical siderosis (HR, 4.69).576,577 The presence of microbleeds and cortical siderosis can be determined during the etiological workup of ICH (Section 4.1, Diagnostic Assessment of Acute ICH Course). Carriers of apolipoprotein E genotypes associated with amyloid angiopathy are similarly at higher risk of ICH recurrence compared with those with the more common ε3/ε3 genotype; those with the ε2 or ε4 allele have an HR of 3.3 and 2.5 for recurrence, respectively.578 Recurrence risk also increases with higher measured outpatient BP563 and age570,579 (HR, 2.8 in age >65 years) and is higher in those of Black race (HR, 1.22) or Asian race (HR, 1.29) compared with White race (race defined by self-designation, clinicians, or administrative personnel while in hospital).568 Association of ICH recurrence with Hispanic ethnicity has been inconsistent.568,580Knowledge Gaps and Future ResearchThere is insufficient evidence to estimate ICH recurrence risk on an individual-patient basis. Deriving and validating a prediction rule incorporating clinical, radiological, and genotype biomarkers and determining the most informative thresholds for categorizing these factors would be helpful to estimate the risk of recurrence.The mechanism by which race is associated with ICH recurrence, including the likely crucial role of social determinants of health, is unclear. More research into this association is required.MRI findings suggestive of small vessel disease may reflect an increased risk for ICH recurrence. More research is needed into the recurrence risks associated with T2 hyperintensities, enlarged perivascular spaces, microangiopathic changes, intragyral hemorrhage, and lobar versus nonlobar microbleeds.9.1.2. BP ManagementRecommendations for BP ManagementReferenced studies that support recommendations are summarized in Data Supplements 75 and 76.SynopsisHypertension has a strong causal association with ICH and is a major modifiable risk factor for all stroke subtypes. Uncontrolled hypertension accounts for 73.6% of the global population-attributable risk for ICH.93 Despite this, a significant proportion of ICH survivors continue to have poorly controlled BP.563,583 Moreover, patients with ICH are also at risk of future ischemic stroke and cardiovascular disease because of overlapping risk factors. Treating hypertension after ICH is a safe and effective way to mitigate future ICH risk and reduce events across the spectrum of vascular disease.581 It is therefore critical to measure and identify uncontrolled hypertension after ICH and aggressively manage BP to prevent recurrence.Recommendation-Specific Supportive TextIn a large prospective cohort study of 1145 patients with primary ICH and a median follow-up of 36.8 months, inadequate BP control was associated with increased risk of both lobar (HR, 3.53 [95% CI, 1.65–7.54]) and nonlobar (HR, 4.23 [95% CI, 1.02–17.52]) ICH recurrence.563 In PROGRESS (Perindopril ProtectionAgainst Recurrent Stroke Study), treatment with perindopril and indapamide reduced mean BP by 10.8/4.4 mm Hg in patients enrolled with ICH and resulted in a relative risk reduction of 42% (95% CI, 14–60) in major vascular events and a number needed to treat of 18 to prevent ICH recurrence over 5 years.581 The optimal timing for BP lowering after ICH is not known, and a decision to initiate antihypertensive therapy in the acute setting should be in accordance with the recommendations discussed in Section 5.1, Acute BP Lowering.In the PRoFESS trial (Prevention Regimen for Effectively Avoiding Second Strokes), the risk of ICH during follow-up was higher in subjects with SBP ≥160 mm Hg compared with those with SBP of 130 to 139 mm Hg (HR, 2.07 [95% CI, 1.22–3.51]), with a nonsignificant trend toward lower rates of ICH with SBP

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