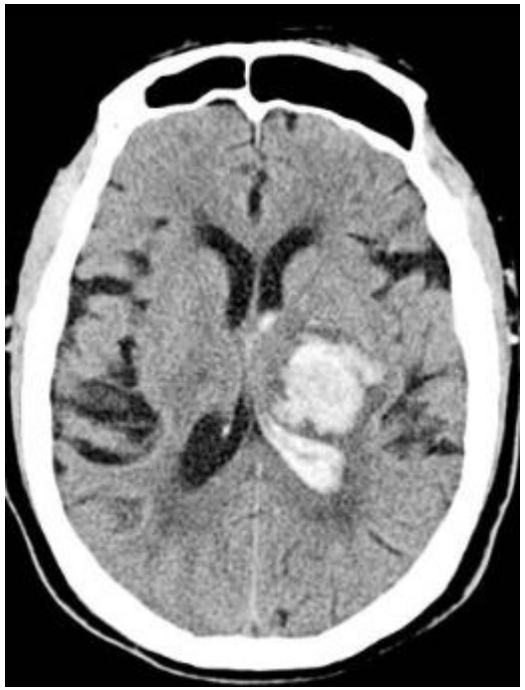
P.O. Ospedale del Mare ASL Napoli 1 Centro

EMORRAGIA INTRACEREBRALE: GESTIONE MEDICA E LIMITI DELLA TERAPIA NEI PAZIENTI COMPLESSI



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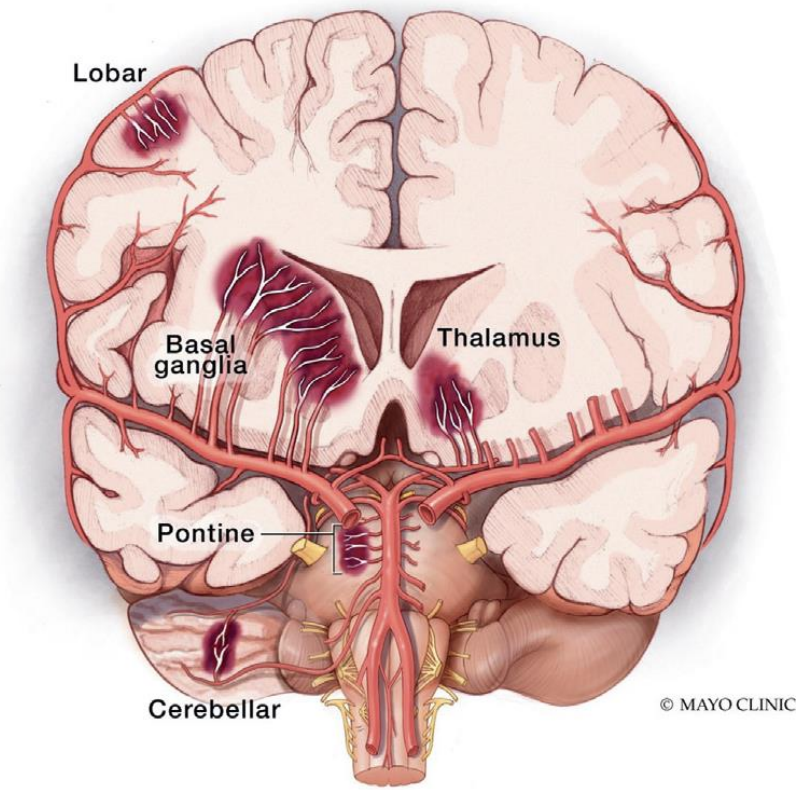
P.O. Ospedale del Mare
28/10/2023

Definition

- "Spontaneous intracerebral hemorrhage" (s-ICH) results from spontaneous rupture of blood vessels in the brain.
- It is the second most common cause of stroke after ischemic stroke.



O'Carroll CB et al. Intracerebral Hemorrhage: A Common yet Disproportionately Deadly Stroke Subtype Mayo Clin Proc. 2021;96(6):1639-1654

Keypoints

1. History, Physical examination, laboratory assessment and General Inpatient Care.
2. Brain Imaging.
3. Blood Pressure Management.
4. Seizures and Antiseizure Drugs.
5. Management of Bleeding under NOAC therapy.
6. Management of Cerebral Edema, Brain Compression and Intracranial Pressure.
7. Prevention of Venous Thromboembolism.
8. (Re-)initiation of Anticoagulation post intracranial bleeding.
9. Secondary Prevention

General Inpatient Care

- Focused **history**.
- **Physical examination**.
- **Routine laboratory** (complete blood count, prothrombin time/ international normalized ratio [INR]/partial thromboplastin time, creatinine/estimated glomerular filtration rate, glucose, cardiac troponin, toxicology screen, and infiammatory markers).
- **Dysphagia screening** protocol should be implemented before initiation of oral intake to reduce disability and the risk of pneumonia.
- ECG and continuous **cardiac monitoring** for the first 24 to 72 hours of admission is reasonable to monitor for cardiac arrhythmias and new cardiac ischemia.
- Diagnostic laboratory and radiographic testing for **infection**.
- Treating hypoglycemia **(<40–60 mg/dL**, <2.2–3.3 mmol/L) and hyperglycemia **(>180– 200 mg/dL**, >10.0–11.1 mmol/L).
- Pharmacologically treating an **elevated temperature.** Do not use prophylactic antibiotics.
- Gastrointestinal stress ulcer prophylaxis

Greenberg SM et al - 2022 Guideline for the Management of Patients With Spontaneous Intracerebral Hemorrhage: A Guideline From the American Heart Association/American Stroke Association Stroke. 2022

Table 3. Initial History, Physical Examination and Laboratory Workup in Patients With ICH

Assessment type	Comments
History	
Time of symptom onset (or time patient was last normal)	
Symptoms	Headache Thunderclap: Aneurysm, RCVS, some instances of CVST Slower onset: Mass lesion, some instances of CVST, ischemic stroke with hemorrhagic transformation Focal neurologic deficits Seizures Decreased level of consciousness
Vascular risk factors	Ischemic stroke Prior ICH Hypertension (Section 9.1.2) Hyperlipidemia Diabetes Metabolic syndrome Imaging biomarkers (eg, cerebral microbleeds; Section 9.1.1)
Medications	Antithrombotics: Anticoagulants (Section 5.2.1), thrombolytics, antiplatelet agents (Section 5.2.2), NSAIDs (9.1.4), dose and time of last ingestion Vasoconstrictive agents (associated with RCVS): Triptans, SSRIs (Section 8.2), decongestants, stimulants, phentermine, sympathomimetic drugs Antihypertensives (as a marker of chronic hypertension) Estrogen-containing oral contraceptives (hemorrhage attributable to CVST)
Cognitive impairment or dementia	Associated with (but not specific for) amyloid angiopathy
Substance use (Section 9.1.5)	Smoking Alcohol use Marijuana (associated with RCVS) Sympathomimetic drugs (amphetamines, methamphetamines, cocaine)
Liver disease, uremia, malignancy, and hematologic disorders	May be associated with coagulopathy

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Physical examination	
Vital signs	Including assessment of airway, breathing, circulation
A general physical examination focusing on the head, heart, lungs, abdomen, and extremities	
A focused neurological examination	A structured examination (such as the NIHSS) can be completed in minutes and provides a quantification that allows easy communication of the severity of the event to other caregivers. GCS is relevant to patients with impaired level of consciousness.
Serum and urine tests	
Complete blood count, blood urea nitrogen and creatinine, liver function tests, glucose, inflammatory markers (ESR and/or CRP)	Anemia is associated with poor outcomes and hemorrhagic expansion.[73,74] Thrombocytopenia is associated with increased mortality.[75] Acute kidney injury and hyperglycemia are associated with worse outcomes and mortality.[68–71,76–81] Inflammatory markers are associated with infective endocarditis.[82] GFR influences clearance of DOACs.[83]
Prothrombin time (with INR) and an activated partial thromboplastin time, specific tests for DOACs when appropriate	Anticoagulant-related hemorrhages are associated with an increased hematoma volume, greater volume and time interval of expansion, and increased morbidity and mortality.[84–86] Specific tests for DOACs (including dilute thrombin time, anti-Xa activity) may be useful for considering reanticoagulation.[87]
Cardiac-specific troponin and ECG	Elevated troponin levels are associated with increased mortality. Signs of left ventricular hypertrophy and other abnormalities on ECG can identify chronic hypertension, myocardial ischemia, or prior cardiac injury.
Urine toxicology screen	Cocaine and other sympathomimetic drugs are associated with ICH.
Pregnancy test in a woman of childbearing age	Peripartum angiopathy, eclampsia, HELLP syndrome, and sinus venous thrombosis can cause ICH in pregnant women.

Greenberg SM et al - 2022 Guideline for the Management of Patients With Spontaneous Intracerebral Hemorrhage: A Guideline From the American Heart Association/American Stroke Association Stroke. 2022

Brain Imaging

ACUTE PHASE

In case of ICH, the use of cerebral **CT or CTA** is suggested to search for any structural abnormalities underlying the bleeding.

- In patients with lobar spontaneous ICH and age <70 years, deep/posterior fossa spontaneous ICH and age <45 years, or deep/ posterior fossa and age 45 to 70 years without history of hypertension, **acute CTA** plus consideration of **venography** is recommended to exclude macrovascular causes or cerebral venous thrombosis.

- In patients with spontaneous ICH with a negative CTA/venography, it is reasonable to perform **MRI and MRA** to establish a nonmacrovascular cause of ICH (such as CAA, small vessel disease, cavernous malformation or malignancy).

SUBACUTE/CHRONIC PHASE

- **MRI with the gradient echo technique** is suggested to highlight the presence of small and previous microbleeds (microbleeds), a non-specific finding even if more frequently observed in patients with risk factors for cerebrovascular diseases. It is able to objective the hemosiderin outcomes in the subacute and chronic phase even years after the event, as the hemosiderin remains for an indefinite time in the tissue, so it is a stable marker of previous hemorrhage.

Greenberg SM et al - 2022 Guideline for the Management of Patients With Spontaneous Intracerebral Hemorrhage: A Guideline From the American Heart Association/American Stroke Association Stroke. 2022

ICH score to predict 30-day mortality after spontaneous ICH

Component	ICH score points
GCS score* at presentation	
3 to 4	2
5 to 12	1
13 to 15	0
ICH volume on initial imaging	
≥30 cm^3	1
<30 cm^3	0
Intraventricular extension of ICH	
Present	1
Absent	0
Infratentorial origin of ICH	
Yes	1
No	0
Age (years)	
≥80	1
<80	0
Total	**0 to 6**

The predicted 30-day mortality is 13% for an ICH score of 1, 26% for score of 2, 72% for a score of 3, 97% for a score of 4, and 100% for a score of 5.

Blood Pressure Management

AHA/ASA GUIDELINE

2022 Guideline for the Management of Patients With Spontaneous Intracerebral Hemorrhage: A Guideline From the American Heart Association/American Stroke Association

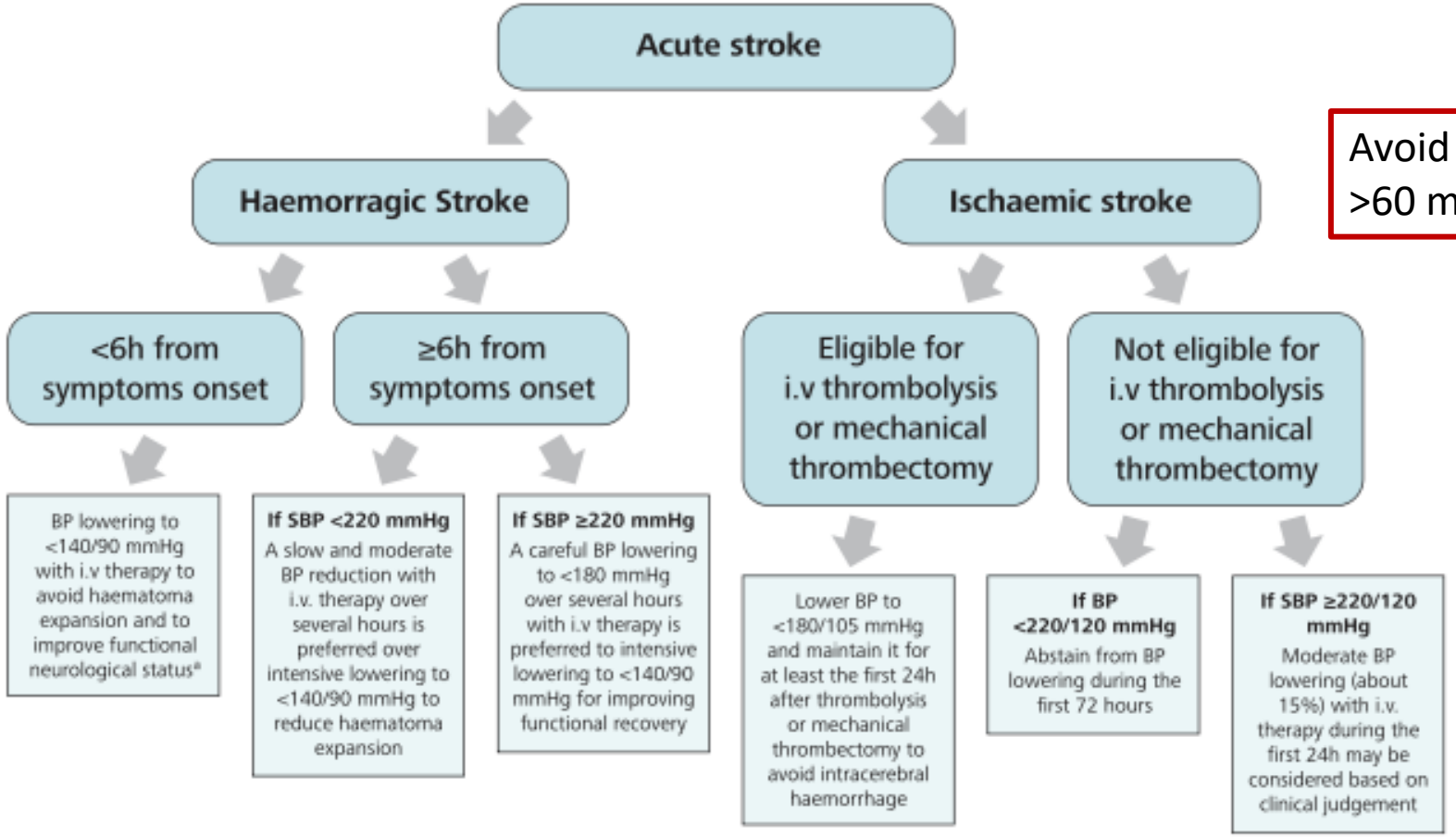
Recommendations for Acute BP Lowering
Referenced studies that support recommendations are summarized in Data Supplements 16 and 17.

COR	LOE	Recommendation
2a	B-NR	1. In patients with spontaneous ICH requiring acute BP lowering, careful titration to ensure continuous smooth and sustained control of BP, avoiding peaks and large variability in SBP, can be beneficial for improving functional outcomes.[138]
2a	C-LD	2. In patients with spontaneous ICH in whom acute BP lowering is considered, initiating treatment within 2 hours of ICH onset and reaching target within 1 hour can be beneficial to reduce the risk of HE and improve functional outcome.[139,140]
2b	B-R	3. In patients with spontaneous ICH of mild to moderate severity presenting with SBP between 150 and 220 mm Hg, acute lowering of SBP to a target of 140 mm Hg with the goal of maintaining in the range of 130 to 150 mm Hg is safe and may be reasonable for improving functional outcomes.[138,141–147]
2b	C-LD	4. In patients with spontaneous ICH presenting with large or severe ICH or those requiring surgical decompression, the safety and efficacy of intensive BP lowering are not well established.[148]
3: Harm	B-R	5. In patients with spontaneous ICH of mild to moderate severity presenting with SBP >150 mm Hg, acute lowering of SBP to <130 mm Hg is potentially harmful.[146,149,150]

ISA-AII
Italian Stroke Association - Associazione Italiana Ictus

PA sistolica <140 mmHg, possibilmente entro un'ora e di mantenere per almeno 24 ore e preferibilmente per i primi 7 giorni.

Blood Pressure Management



Avoid absolute reductions of SBP >60 mmHg from initial SBP

Recommendations and statements	CoR	LoE
In patients with haemorraghic stroke and < 6h after symptom onset, a BP <140/90 mmHg is recommended to avoid haematoma expansion.	II	B
In patients with haemorraghic stroke >6h after symptom onset, an SBP ≥220 mmHg may be carefully lowered with i.v. therapy to <180 mmHg. If SBP < 220 mmHg, slow and moderate BP reductions are preferable over intensive BP to <140/90 mmHg.	II	B

2023 ESH Guidelines for the management of arterial hypertension.

Blood Pressure Management

Recommendation

In patients with acute (<24 hours) intracerebral haemorrhage there is continued uncertainty over the benefits and risks (advantages/disadvantages) of intensive blood pressure lowering on functional outcome.
Quality of evidence: Moderate⊕⊕⊕
Strength of recommendation: -

In patients with hyperacute (<6 hours) intracerebral haemorrhage, we suggest lowering blood pressure to below 140 mm Hg (and to keep it above 110 mm Hg) to reduce haematoma expansion.
Quality of evidence: Moderate⊕⊕⊕
Strength of recommendation: Weak ↑

Expert consensus statement

In patients with acute intracerebral haemorrhage, we suggest initiating antihypertensive treatment as early as possible and ideally within 2 hours of symptom onset. The decrease of systolic blood pressure should not exceed 90 mm Hg from baseline values. Vote 10 of 10.

In patients with acute intracerebral haemorrhage, we suggest lowering blood pressure according to recommended levels beyond 6 hours after onset of treatment for at least 24 hours and up to 72 hours to reduce haematoma expansion. Vote 10 of 10.

Guideline

EUROPEAN STROKE JOURNAL

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2021, Vol. 6(2) XLVIII–LXXXIX
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SAGE

European Stroke Organisation (ESO) guidelines on blood pressure management in acute ischaemic stroke and intracerebral haemorrhage

Else Charlotte Sandset[1,2], Craig S Anderson[3,4], Philip M Bath[5], Hanne Christensen[6], Urs Fischer[7], Dariusz Gąsecki[8], Avtar Lal[9], Lisa S Manning[10], Simona Sacco[11], Thorsten Steiner[12,13] and Georgios Tsivgoulis[14,15]

Recommendation

In patients with acute intracerebral haemorrhage there is continued uncertainty over the benefits and risks (advantages/disadvantages) of continuing versus temporarily stopping previous blood pressure lowering therapy.
Quality of evidence: Moderate⊕⊕⊕
Strength of recommendation: -

Expert consensus statement

In patients acute intracerebral haemorrhage who need blood pressure lowering therapy to maintain blood pressure within the recommended range and who do not have swallowing problems, we suggest continuation of prior oral antihypertensive agents. Vote 10 of 10.

In patients with acute intracerebral haemorrhage who need blood pressure lowering therapy to maintain blood pressure within the recommended range and who have dysphagia or decreased level of consciousness, we suggest temporarily stopping previous oral hypertensive therapy and using intravenous antihypertensive agents until swallowing is restored or a nasogastric tube is in place. Vote 10 of 10.

Associazione Neurologia Emergenza Urgenza

Blood Pressure Management

Labetalolo (Trandate)

Meccanismo d'azione: simpaticolitico ad azione solo periferica, antagonista dei recettori alfa-1 e beta-1 e minimo agonista beta-2; riduce le resistenze periferiche.

Via e modalità di somministrazione:
- in bolo *ev*: 50 mg in 1 m, seguiti da dosi di 50 mg ogni 5-10 m, max. 200 mg;
- in infusione: 200 mg in 200 cc di fisiol. (1 mg = 1 ml) a circa 2 ml/m.

Controindicazioni: asma, scompenso cardiaco, bradicardia, blocchi A-V, acidosi metabolica, insufficienza renale.

Clonidina (Catapresan)

Meccanismo d'azione: simpaticolitico ad azione solo centrale, agonista alfa-2; inibisce la produzione di catecolamine riducendo il tono simpatico.

Via e modalità di somministrazione:
- *im*: 150 mcg;
- *ev*: 150 mcg diluiti in 10 ml di fisiol. in 10 m;
- in infusione: 0.2 mcg/kg/m.

Controindicazioni: bradicardia, blocchi A-V, PRES.

Urapidil (Ebrantil)

Meccanismo d'azione: simpaticolitico ad azione sia periferica che centrale, antagonista dei recettori alfa-1; riduce sia le resistenze periferiche che il tono simpatico centrale.

Via e modalità di somministrazione:
- in bolo *ev*: 25 mg in 1 m, seguiti da dosi di 25 mg ogni 2 m, max. 100 mg;
- in infusione: 250 mg in 500 cc di fisiol. (1 mg = 2 ml) a circa 4 ml/m

Controindicazioni: stenosi aortica.

Nitroglicerina (Venitrin), Nitroprussiato sodico

Meccanismo d'azione: profarmaci che liberano ossido nitrico (NO) a livello della muscolatura liscia vasale, provocando vasodilatazione.

Via e modalità di somministrazione:
- nitroglicerina: in infusione: 10 mg in 500 cc di fisiol. (1 mg = 50 ml) a 0,75-1,5-3 mg/h.
- nitroprussiato sodico: in infusione: 1 fiala ricostituita diluita in 1000 cc di gluc. (100 mcg = 1 ml) a 3 mcg/kg/m.

Controindicazioni: ipertensione endocranica, ICH, PRES.

Seizures and Antiseizure Drugs

8-15% but may be up to 30% when including patients with nonconvulsive seizures.

- ✓ In patients with spontaneous ICH, impaired consciousness, and confirmed electrographic seizures, antiseizure drugs should be administered to reduce morbidity.
- ✓ In patients with spontaneous ICH and clinical seizures, antiseizure drugs are recommended to improve functional outcomes and prevent brain injury from prolonged recurrent seizures.

- In patients with spontaneous ICH without evidence of seizures, prophylactic antiseizure medication is not beneficial to improve functional outcomes, long-term seizure control, or mortality.

Treatment → Intravenous antiseizure medication

Patients with ICH and a seizure	Optimal duration
Early seizure (<14 days from ICH onset)	Continue treatment for several days and then wean when patients are clinically stable if seizures do not recur.
Late seizure (>14 days from ICH onset)	Continue long-term seizure therapy

Greenberg SM et al - 2022 Guideline for the Management of Patients With Spontaneous Intracerebral Hemorrhage: A Guideline From the American Heart Association/American Stroke Association Stroke. 2022

Management of Bleeding under NOAC therapy.

Guideline

EUROPEAN STROKE JOURNAL

European Stroke Journal
2019, Vol. 4(4) 294–306

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journals.sagepub.com/home/eso
SAGE

European Stroke Organisation Guideline on Reversal of Oral Anticoagulants in Acute Intracerebral Haemorrhage

Hanne Christensen[1], Charlotte Cordonnier[2], Janika Kõrv[3], Avtar Lal[4], Christian Ovesen[1], Jan C Purrucker[5], Danilo Toni[6] and Thorsten Steiner[5,7]

VKA

- Ultrafast INR testing
- INR: reverse if >1.3
- 1. line
  - INR >1.3-1.9: PCC 10 IU/kg
  - INR >1.9: PCC 30 IU/kg
- 2.line
  - FFP 20 mL/kg
- Always Vit K 10 mg IV
- Repeat INR at 12-24 hours

fXa-inhibitors

- In some centres specific testing is available. To be used if results are ready in <20 min
- 1. line
  - andexanet alfa* (for apixaban, rivaroxaban)
  - Last dose>7 hours: bolus 400 mg, infusion 480 mg over 120 minutes
  - Last dose <7 hours:bolus 800 mg, infusion 960 mg over 120 minutes
- Repeat specific testing after 3, 6, 12 and 24 hours because rebound might occur
- 2. line
  - PCC 50 IU/kg
  - (if andexanet is not available or for edoxaban-ICH)

dabigatran

- In some centres specific testing is available. To be used if results are ready in <20 min
- Idarucizumab
  - 2.5 g x 2 IV
- Repat specific testing after 12 and 24h because rebound might occur

LMWH and UFH

- Protamina solfato→ 1 mg ev ogni 100 U.I. di eparina fino a 50 mg in 10 minuti

Management of Cerebral Edema, Brain Compression and Intracranial Pressure

Tier	Therapies
Zero, standard measures for all patients at risk of intracranial pressure elevation	Supportive medical care (airway, breathing, circulation)
	Analgesia for comfort
	Sedation to tolerate medical interventions (Richmond Agitation and Sedation Scale score 0 to -2)[37]
	Avoid fever (normothermia 36 °C to 37 °C [96.8 °F to 98.6 °F])
	Avoid constipation/abdominal distension
	Head at 30- to 45-degree elevation
	Head midline; avoid jugular vein compression
	Isotonic or hyperosmolar fluids targeting normal serum sodium (>135 mmol/L)
	Steroids for select conditions[b]
One	Mannitol or hypertonic saline for symptom-directed or osmolality/sodium level goal
	CSF diversion, drain 5-10 mL if external ventricular drain in place
	Selective consideration of surgical decompression or lesion resection
	Mild hyperventilation[c]
Two	Hypertonic saline if refractory to mannitol; consider higher osmolality goal
	Sedation and analgesia for deeper Richmond Agitation and Sedation Scale goal
	Reconsider surgical decompression as lifesaving measure
	Mild hyperventilation[c]
Three	Patient determined not to be a surgical candidate
	Sedation/barbiturate titrated to intracranial pressure goal or EEG burst suppression
	Moderate hypothermia (core temperature 32 °C to 34 °C [89.6 °F to 93.2 °F])
	Moderate hyperventilation[d]

Recommendations for Neuroinvasive Monitoring, ICP, and Edema Treatment
Referenced studies that support recommendations are summarized in Data Supplements 49 through 54.

COR	LOE	Recommendations
1	B-NR	1. In patients with spontaneous ICH or IVH and hydrocephalus that is contributing to decreased level of consciousness, ventricular drainage should be performed to reduce mortality.[347–350]
2b	B-NR	2. In patients with moderate to severe spontaneous ICH or IVH with a reduced level of consciousness, ICP monitoring and treatment might be considered to reduce mortality and improve outcomes.[159,351–356]
2b	B-NR	3. In patients with spontaneous ICH, the efficacy of early prophylactic hyperosmolar therapy for improving outcomes is not well established.[357–361]
2b	C-LD	4. In patients with spontaneous ICH, bolus hyperosmolar therapy may be considered for transiently reducing ICP.[362–364]
3: No Benefit	B-R	5. In patients with spontaneous ICH, corticosteroids should not be administered for treatment of elevated ICP.[365–369]

2021 American Academy of Neurology Management of Cerebral Edema, Brain Compression, and Intracranial Pressure By Eric M. Liotta.

Management of Cerebral Edema, Brain Compression and Intracranial Pressure

Hypertonic saline is available in concentrations ranging from 2% to 23.4% and may be given by bolus or continuous infusion.

For bolus dosing, **150 mL to 500 mL of 3% saline over 15 to 30 minutes or 30 mL of 23.4% saline over 10 minutes** is common.

Mannitol is an osmotic diuretic that is delivered by a filtered peripheral IV catheter as a 20% solution at a **bolus dose of 0.5 g/kg to 2 g/kg,** depending on the severity of the indication. Mannitol is typically redosed as boluses **every 4 to 6 hours.**

Steroids are **not used** in the management of cerebral edema from hemorrhagic or ischemic stroke because current evidence suggests no benefit and **potential harm**.

Hypotonic fluids are **contraindicated** as they may exacerbate cerebral edema and intracranial pressure.

2021 American Academy of Neurology
Management of Cerebral Edema, Brain Compression, and Intracranial Pressure By Eric M. Liotta.

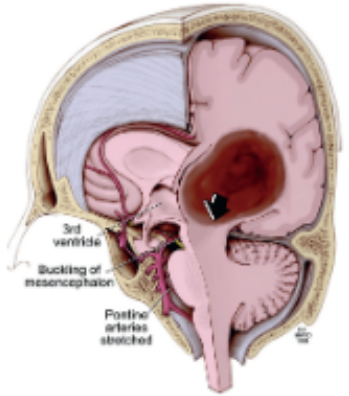
Indication for emergent surgery

Indication for emergent surgery on onset
Cerebellar hemorrhage > 3 cm in diameter or associated with acute neurological deterioration, brainstem compression, or hydrocephalus due to ventricular obstruction.
Intraventricular hemorrhage with ventricular enlargement associated with acute neurologic deterioration.
Supratentorial (hemispheric) hemorrhage associated with acute neurological deterioration and life-threatening brain compression or hydrocephalus

Not all patients will benefit from surgery.

Imaging findings (CT or MRI)
Increasing shift of brain tissue beyond midline
Ventricular or brainstem compression
Obstructive hydrocephalus
Herniation (transtentorial, parafalcine, uncal, central, tonsillar) of brain structures

Clinical exam findings
Pupillary changes, including impaired reactivity to light
Abducens nerve (cranial nerve VI) palsy; alert patients may report horizontal diplopia
Progressive drowsiness
Cushing triad consisting of bradycardia, respiratory depression, and hypertension
Focal symptoms related to **herniation syndromes**



- Headache
- Altered level of consciousness
- Dilation of ipsilateral pupil
- Cranial nerve III palsy
  - Ptosis
  - Loss of medial gaze
- Decerebrate posturing
- Hemiparesis
- Dilation of opposite pupil
- Alteration of respiration
- Bradycardia
- Hypertension
- Respiratory arrest

Greenberg SM et al - 2022 Guideline for the Management of Patients With Spontaneous Intracerebral Hemorrhage: A Guideline From the American Heart Association/American Stroke Association Stroke. 2022

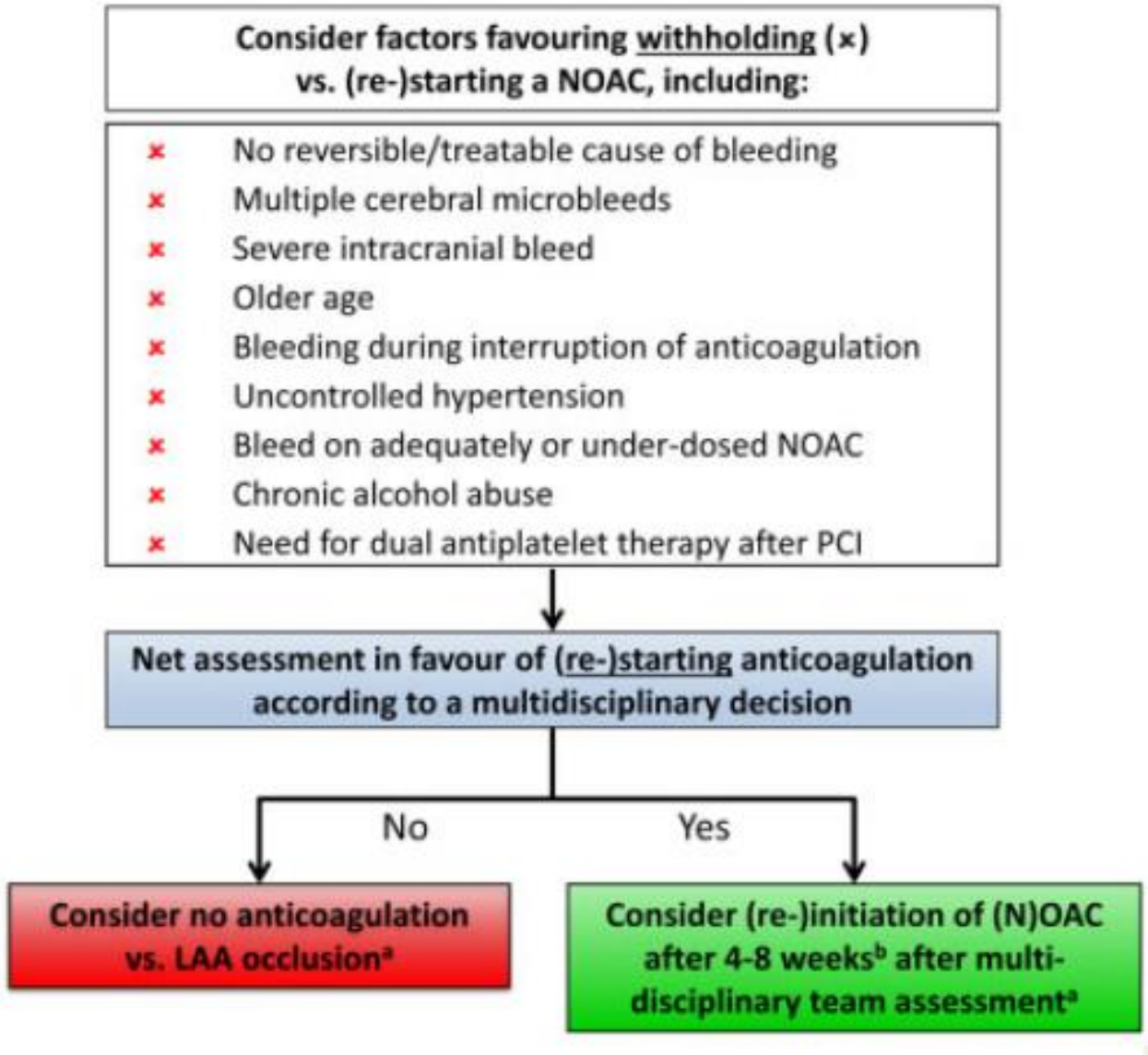
(Re-)initiation of Anticoagulation post intracranial bleeding.

ESC European Society of Cardiology
Europace (2021) 23, 1612–1676
doi:10.1093/europace/euab065

POSITION PAPER
EHRA Practical Guide

2021 European Heart Rhythm Association Practical Guide on the Use of Non-Vitamin K Antagonist Oral Anticoagulants in Patients with Atrial Fibrillation

Jan Steffel[1]*, Ronan Collins[2], Matthias Antz[3], Pieter Cornu[4], Lien Desteghe[5,6], Karl Georg Haeusler[7], Jonas Oldgren[8], Holger Reinecke[9], Vanessa Roldan-Schilling[10], Nigel Rowell[11], Peter Sinnaeve[12], Thomas Vanassche[12], Tatjana Potpara[13], A. John Camm[14], and Hein Heidbüchel[5,6]



(Re-)initiation of Anticoagulation post intracranial bleeding.

AHA/ASA GUIDELINE

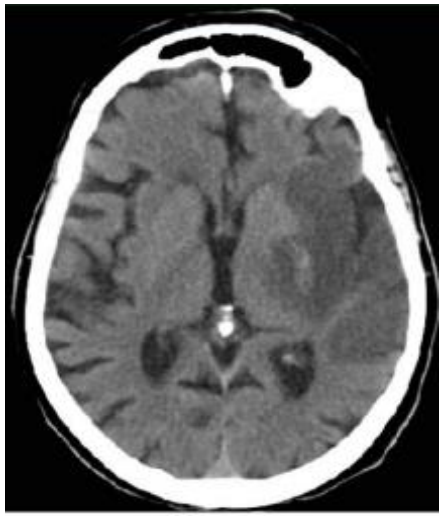
2022 Guideline for the Management of Patients With Spontaneous Intracerebral Hemorrhage: A Guideline From the American Heart Association/American Stroke Association

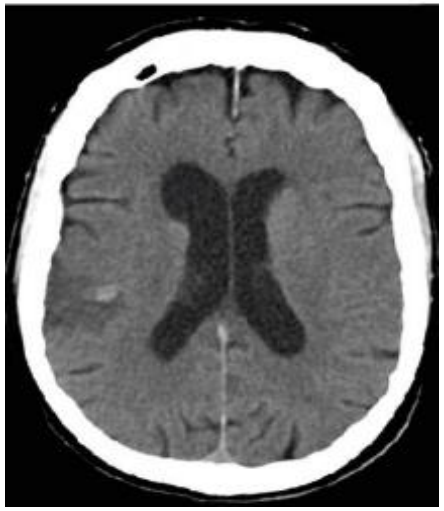
9.1.3. Management of Antithrombotic Agents

Recommendations for Management of Antithrombotic Agents
Referenced studies that support recommendations are summarized in Data Supplements 77 through 79.

COR	LOE	Recommendations
2a	C-LD	1. In patients with spontaneous ICH and conditions placing them at high risk of thromboembolic events, for example, a mechanical valve or LVAD, early resumption of anticoagulation to prevent thromboembolic complications is reasonable.[586,587]
2b	B-R	2. In patients with spontaneous ICH with an indication for antiplatelet therapy, resumption of antiplatelet therapy may be reasonable for the prevention of thromboembolic events based on consideration of benefit and risk.[588,589]
2b	B-NR	3. In patients with nonvalvular atrial fibrillation (AF) and spontaneous ICH, the resumption of anticoagulation to prevent thromboembolic events and reduce all-cause mortality may be considered based on weighing benefit and risk.[590–595]
2b	C-LD	4. In patients with AF and spontaneous ICH in whom the decision is made to restart anticoagulation, initiation of anticoagulation ≈7 to 8 weeks after ICH may be considered after weighing specific patient characteristics to optimize the balance of risks and benefits.[596,597]
2b	C-LD	5. In patients with AF and spontaneous ICH deemed ineligible for anticoagulation, left atrial appendage closure may be considered to reduce the risk of thromboembolic events.[598–602]

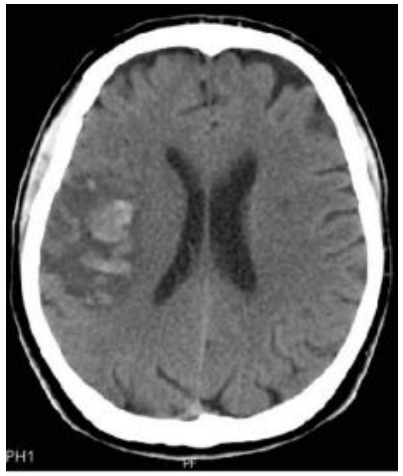
- Infarto emorragico 1 (HI 1) petecchie non confluenti
- Infarto emorragico 2 (HI 2) petecchie confluenti

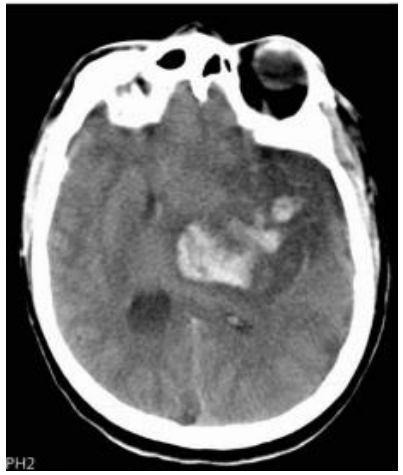




La sospensione del trattamento antitrombotico deve essere valutata caso per caso. È consigliabile l'interruzione di un trattamento anticoagulante, mentre un trattamento con antiaggreganti piastrinici potrebbe, in alcuni casi, essere continuato (ad es. nel paziente con stroke secondario a stenosi carotidea significativa o fibrillazione atriale, condizioni nelle quali il rischio di recidiva precoce è molto alto, con attento monitoraggio clinico e neuro-radiologico).

- Ematoma parenchimale 1 (PH 1), interessamento emorragico < 30% dell'area ischemica con lieve/moderato effetto massa
- Ematoma parenchimale 2 (PH 2), interessamento emorragico > 30% con significativo effetto massa





Interrompere qualsiasi trattamento antitrombotico, almeno fino alla dimostrazione della cessazione del sanguinamento.

Paciaroni et al. Hemorrhagic transformation in ischemic stroke and its treatment during thrombolysis, Reviews in Health Care 2011

Prevention of Venous Thromboembolism

Profilassi del tromboembolismo venoso in pazienti con emorragia cerebrale spontanea

Prophylaxis of venous thrombosis in patients with spontaneous intracerebral bleeding

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Ad alto rischio

- età avanzata
- sesso femminile
- obesità
- immobilizzazione prolungata
- paralisi degli arti
- localizzazione lobare
- ampio diametro dell'ECS
- NIHSS ≥ 12

Tutte le linee guida sostanzialmente concordano sul ruolo della profilassi meccanica con CPI nei pazienti con ECS per prevenire il TEV, e suggeriscono di iniziare la loro applicazione il prima possibile.

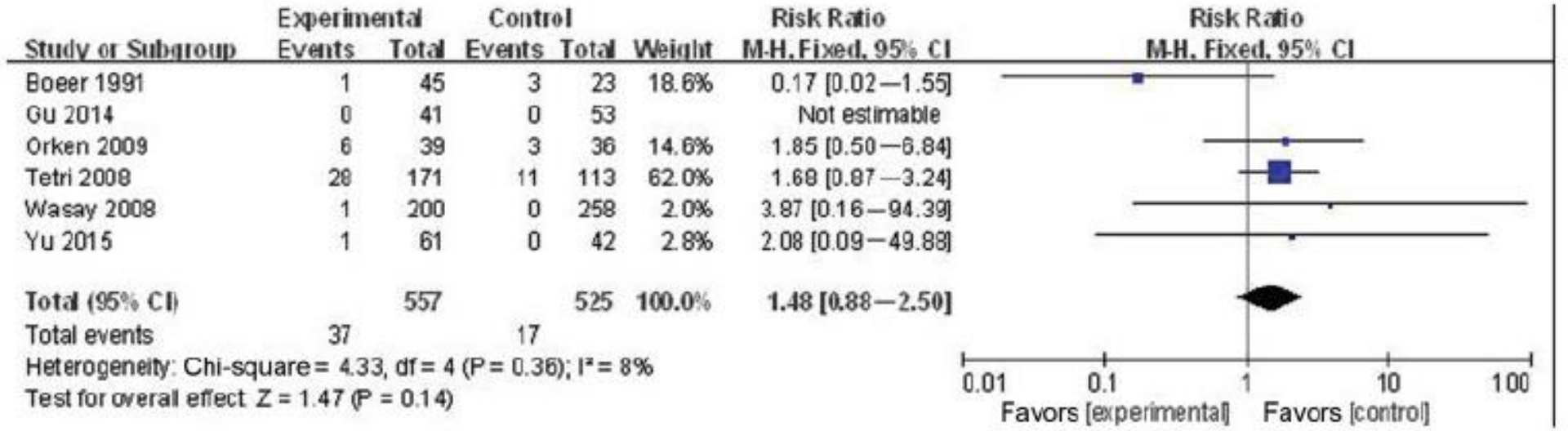
Strategie farmacologiche solo dopo che la stabilizzazione del paziente sia documentata, ovvero dopo la dimostrazione della cessazione del sanguinamento valutata sia in base alla stabilità clinica sia per mezzo di una TC di controllo.

Prevention of Venous Thromboembolism

Safety of Prophylactic Heparin in the Prevention of Venous Thromboembolism After Spontaneous Intracerebral Hemorrhage: A Meta-analysis

Xi Pan[1,*] Jihui Li[2,*] Lan Xu[3] Shengming Deng[2] Zhi Wang[1]

Heparin initiation on day 2 led to a statistically lower rate of PE than did initiation on day 4 or day 10.



Study or Subgroup	Experimental Events	Total	Control Events	Total	Weight	Risk Ratio M-H, Fixed, 95% CI
Boeer 1991	1	45	3	23	18.6%	0.17 [0.02—1.55]
Gu 2014	0	41	0	53		Not estimable
Orken 2009	6	39	3	36	14.6%	1.85 [0.50—6.84]
Tetri 2008	28	171	11	113	62.0%	1.68 [0.87—3.24]
Wasay 2008	1	200	0	258	2.0%	3.87 [0.16—94.39]
Yu 2015	1	61	0	42	2.8%	2.08 [0.09—49.88]
Total (95% CI)		**557**		**525**	**100.0%**	**1.48 [0.88—2.50]**
Total events	37		17			

Heterogeneity: Chi-square = 4.33, df = 4 (P = 0.36); I^2 = 8%

Test for overall effect: Z = 1.47 (P = 0.14)

Fig. 2 Any hematoma enlargement in studies comparing low-molecular-weight heparin or unfractionated heparin with non-heparin treatments (elastic compression stockings, intermittent pneumatic compression, or placebo) for the prevention of venous thromboembolism.

Prophylactic heparin was associated with:
-a nonsignificant increase in any hematoma enlargement and mortality
-a nonsignificant reduction in extracranial hemorrhage,
-a nonsignificant increase in the incidence of major disability

Prevention of Venous Thromboembolism

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CLINICAL SCIENCES

Is Prophylactic Anticoagulation for Deep Venous Thrombosis Common Practice After Intracerebral Hemorrhage?

Shyam Prabhakaran, MD, MS, Patricia Herbers, MS, Jane Khoury, PhD, Opeolu Adeoye, MD, Pooja Khatri, MD, Simona Ferioli, MD, and Dawn O. Kleindorfer, MD

Stroke
Volume 42, Issue 3, March 2011; Pages 705-709
https://doi.org/10.1161/STROKEAHA.110.600593

ORIGINAL CONTRIBUTIONS; CLINICAL SCIENCES

Pharmacological Deep Vein Thrombosis Prophylaxis Does Not Lead to Hematoma Expansion in Intracerebral Hemorrhage With Intraventricular Extension

Tzu-Ching Wu, MD, Mallik Kasam, PhD, Nusrat Harun, MS, Hen Hallevi, MD, Hesna Bektas, MD, Indrani Acosta, MD, Vivek Misra, MD, Andrew D. Barreto, MD, Nicole R. Gonzales, MD, George A. Lopez, MD, James C. Grotta, MD, and Sean I. Savitz, MD

LMWH and UFH are effective in reducing the risk of VTE, and both are safe against enlarged hematoma, administered both within 48 hours and within four days.

Secondary Prevention

COR	LOE	Recommendations
2b	B-NR	1. In patients with spontaneous ICH and an established indication for statin pharmacotherapy, the risks and benefits of statin therapy on ICH outcomes and recurrence relative to overall prevention of cardiovascular events are uncertain.[605–609]
3: Harm	B-NR	2. In patients with spontaneous ICH, regular long-term use of nonsteroidal anti-inflammatory drugs (NSAIDs) is potentially harmful because of the increased risk of ICH.[610,611]

COR	LOE	Recommendations
1	B-R	1. In patients with spontaneous ICH, BP control is recommended to prevent hemorrhage recurrence.[563,581]
2a	B-NR	2. In patients with spontaneous ICH, it is reasonable to lower BP to an SBP of 130 mm Hg and diastolic BP (DBP) of 80 mm Hg for long-term management to prevent hemorrhage recurrence.[581,582]

Association Between Statin Use and Intracerebral Hemorrhage Location

A Nested Case-Control Registry Study

Nils Jensen Boe, MD, Stine Munk Hald, MD, Mie Micheelsen Jensen, MD, Jonas Asgaard Bojsen, MD, Mohammad Talal Elhakim, MD, Sandra Florisson, MD, Alisa Saleh, Anne Clausen, MSc, Sören Möller, PhD, Frederik Severin Gråe Harbo, MD, Ole Graumann, PhD, Jesper Hallas, DrMedSci, Luis Alberto García Rodríguez, MD, Rustam Al-Shahi Salman, MD, PhD, Larry B. Goldstein, MD, and David Gaist, PhD

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"We found that statin use was associated with a lower risk of ICH, particularly with longer treatment duration. This association did not vary by hematoma location"

GRAZIE PER L'ATTENZIONE