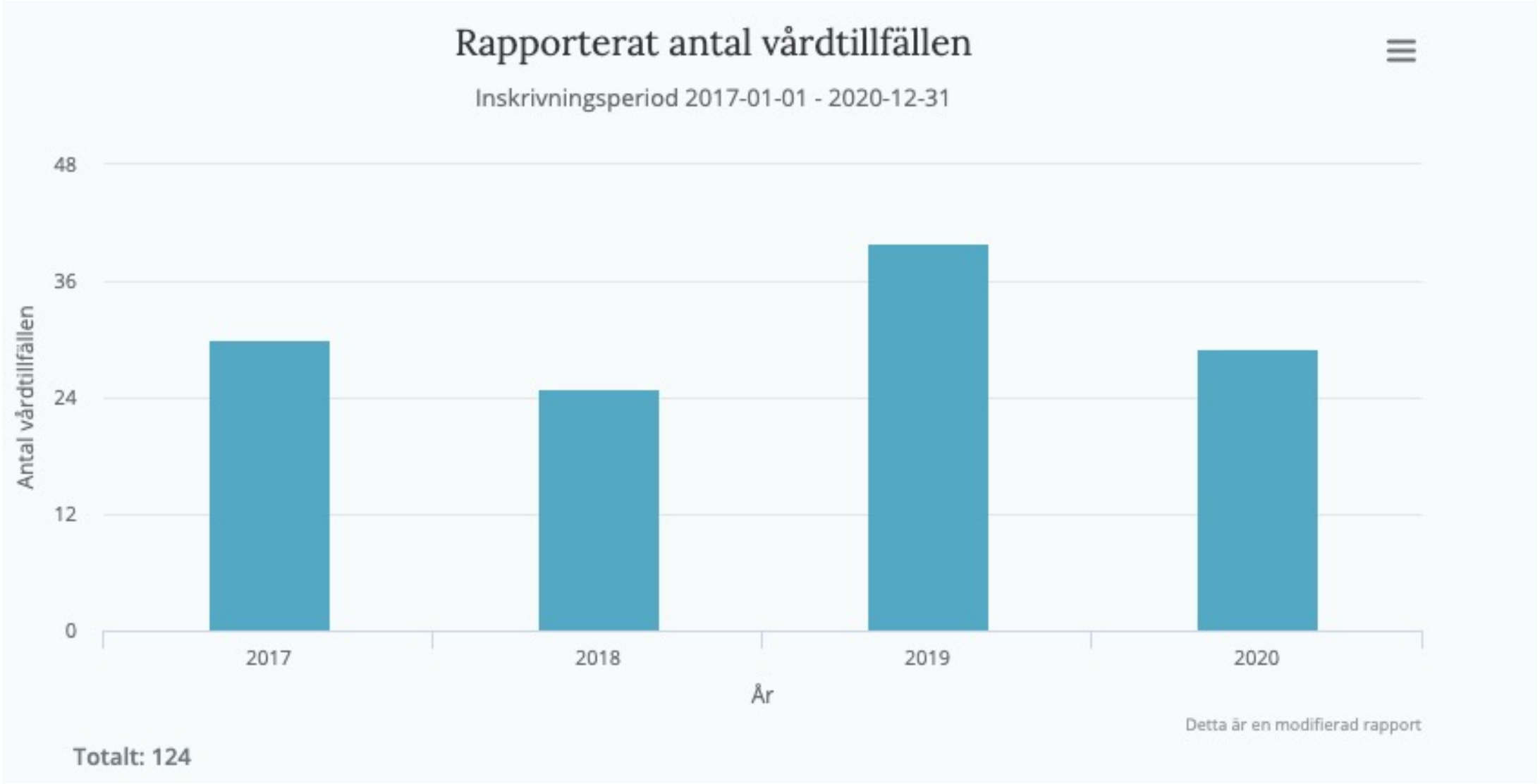
Subarachnoidalblödning

- Intensivistens perspektiv

- Camilla Brorsson

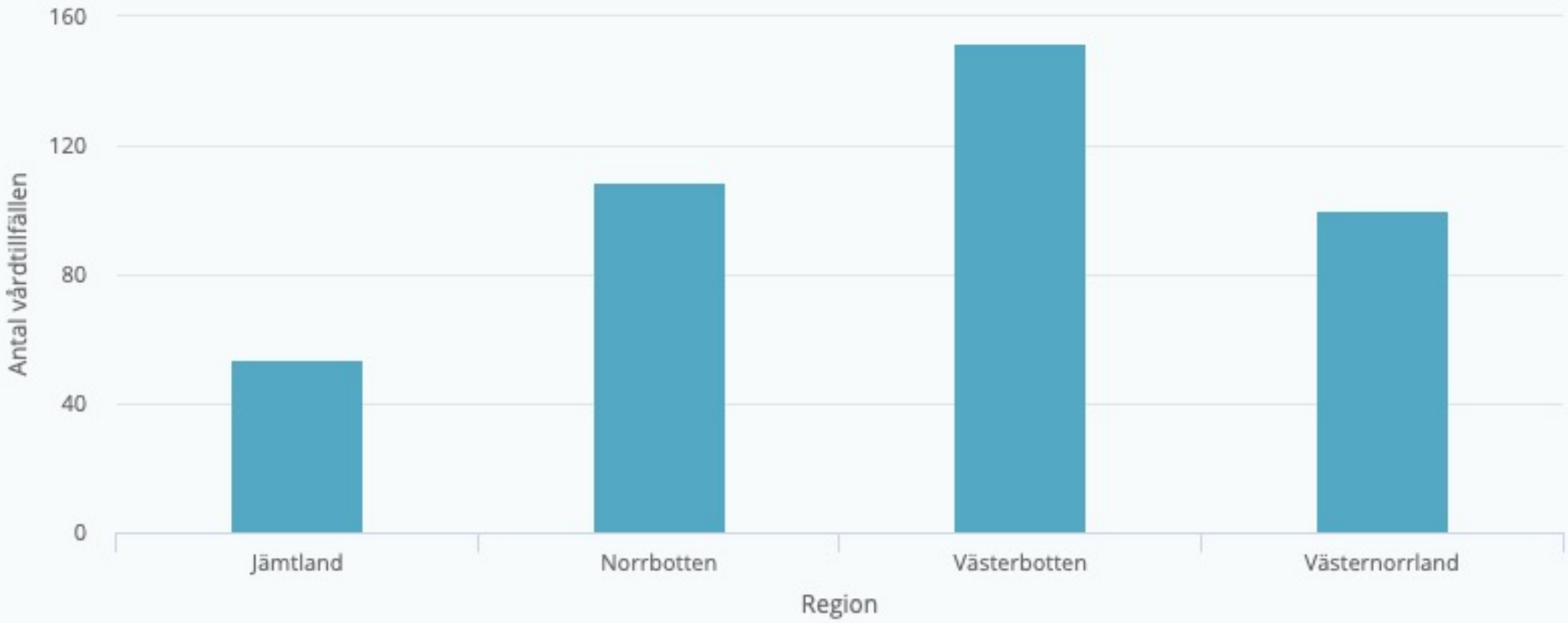
- Maj 2021


Rapporterat antal vårdtillfällen
Inskrivningsperiod 2017-01-01 - 2020-12-31
48
36
24
12
0
Antal vårdtillfällen
2017
2018
2019
2020
År
Detta är en modifierad rapport
Totalt: 124

SAH IVA, NUS

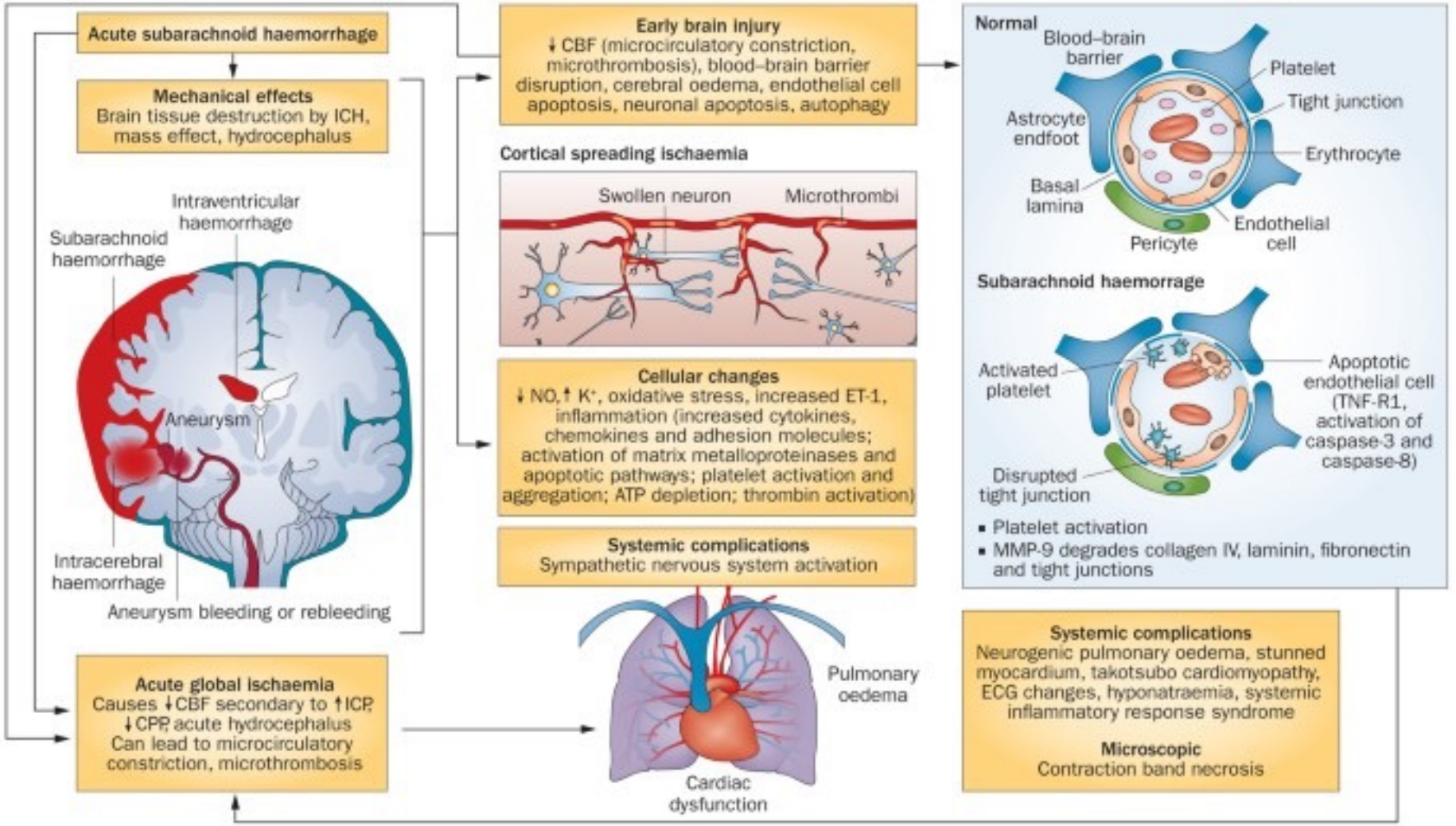
Rapporterat antal vårdtillfällen

Inskrivningsperiod 2017-01-01 - 2020-12-31

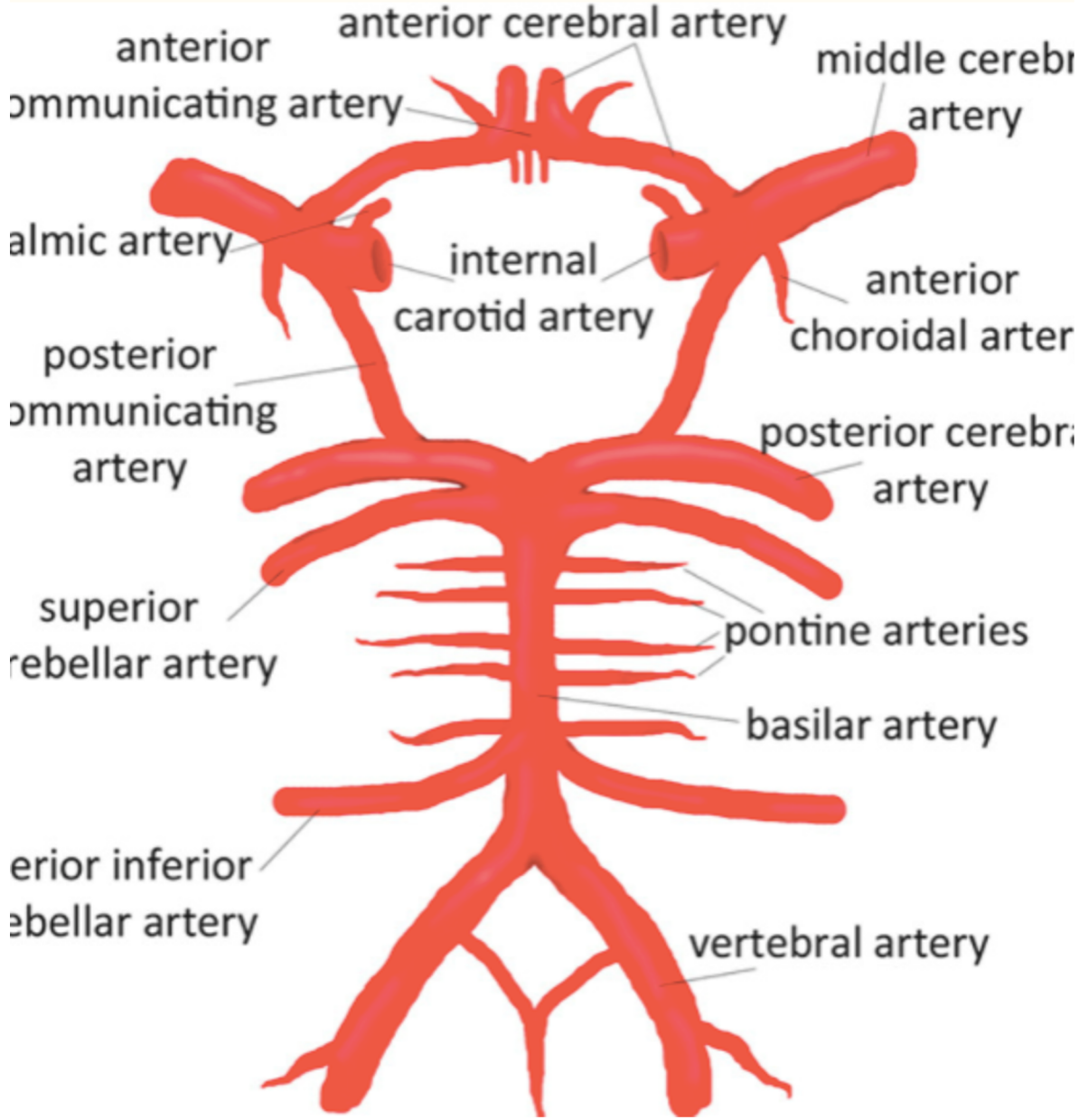


Detta är en modifierad rapport

Totalt: 415

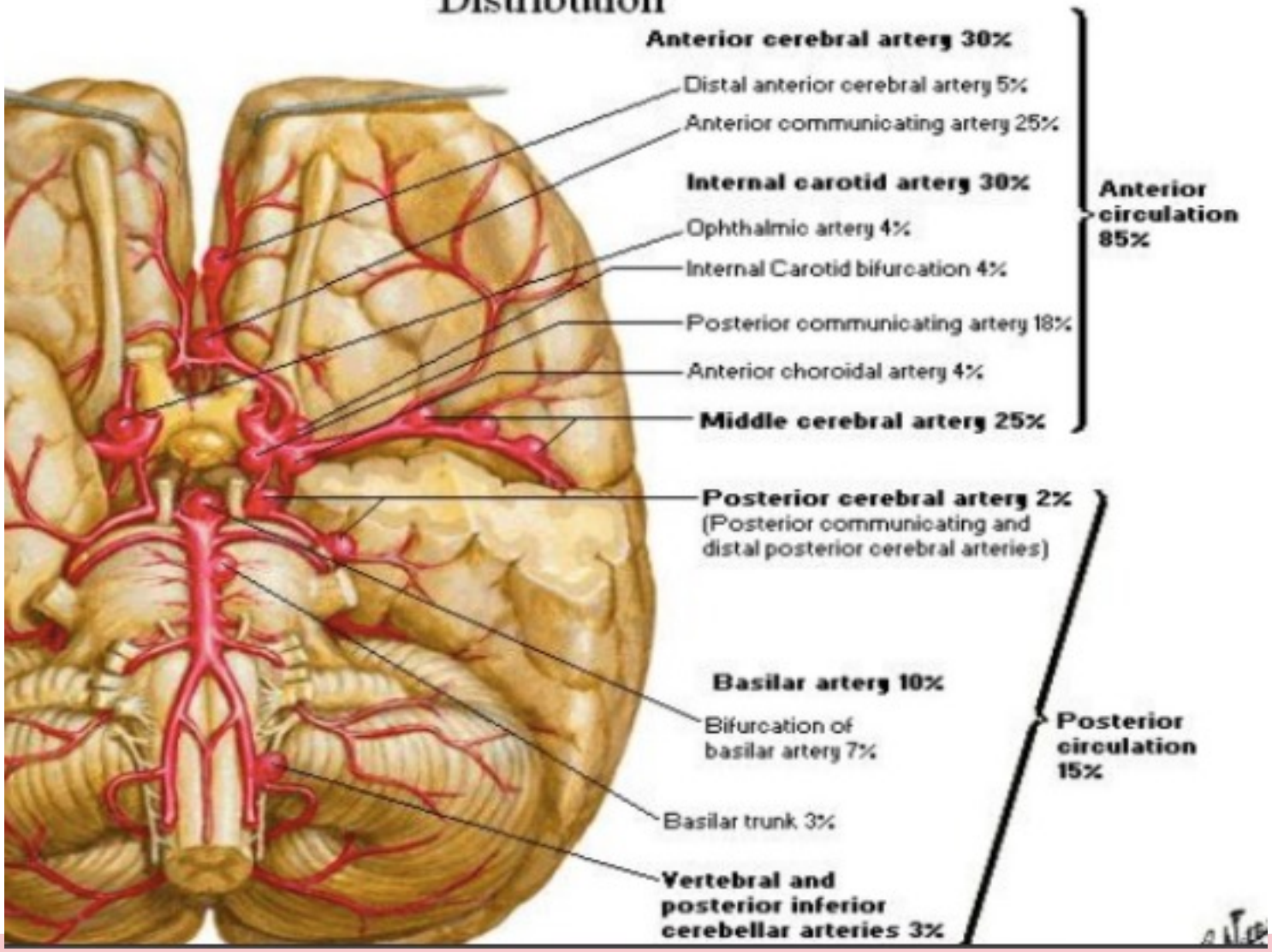

Acute subarachnoid haemorrhage
Mechanical effects
Brain tissue destruction by ICH, mass effect, hydrocephalus
Intraventricular haemorrhage
Subarachnoid haemorrhage
Aneurysm
Intracerebral haemorrhage
Aneurysm bleeding or rebleeding
Acute global ischaemia
Causes ↓CBF secondary to ↑ICP, ↓CPP, acute hydrocephalus
Can lead to microcirculatory constriction, microthrombosis
Early brain injury
↓ CBF (microcirculatory constriction, microthrombosis), blood–brain barrier disruption, cerebral oedema, endothelial cell apoptosis, neuronal apoptosis, autophagy
Cortical spreading ischaemia
Swollen neuron
Microthrombi
Cellular changes
↓ NO, ↑ K+, oxidative stress, increased ET-1, inflammation (increased cytokines, chemokines and adhesion molecules; activation of matrix metalloproteinases and apoptotic pathways; platelet activation and aggregation; ATP depletion; thrombin activation)
Systemic complications
Sympathetic nervous system activation
Pulmonary oedema
Cardiac dysfunction
Normal
Blood–brain barrier
Platelet
Tight junction
Astrocyte endfoot
Erythrocyte
Basal lamina
Endothelial cell
Pericyte
Subarachnoid haemorrage
Activated platelet
Apoptotic endothelial cell (TNF-R1, activation of caspase-3 and caspase-8)
Disrupted tight junction
▪ Platelet activation
▪ MMP-9 degrades collagen IV, laminin, fibronectin and tight junctions
Systemic complications
Neurogenic pulmonary oedema, stunned myocardium, takotsubo cardiomyopathy, ECG changes, hyponatraemia, systemic inflammatory response syndrome
Microscopic
Contraction band necrosis

- De flesta aneurysmen sitter i främre cirkulationen i Circulus Willisi (ISUIA study)
- Aneurysm I posteriora cirkulationen rupturerar oftare
- Sacculära aneurysm vanligare än fusiforma



Congenital Intracranial Aneurysms

Distribution



Bråttom - bråttom

NIVA - IVA har tät kontakt runt dessa patienter

AHA/ASA Guideline

Guidelines for the Management of Aneurysmal Subarachnoid Hemorrhage

A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association

The American Academy of Neurology affirms the value of this statement as an educational tool for neurologists.

Endorsed by the American Association of Neurological Surgeons and Congress of Neurological Surgeons; and by the Society of NeuroInterventional Surgery

Natural History and Outcome of aSAH: Recommendations

1. **The initial clinical severity of aSAH should be determined rapidly by use of simple validated scales (eg, Hunt and Hess, World Federation of Neurological Surgeons), because it is the most useful indicator of outcome after aSAH *(Class I; Level of Evidence B).***
2. **The risk of early aneurysm rebleeding is high, and rebleeding is associated with very poor outcomes. Therefore, urgent evaluation and treatment of patients with suspected aSAH is recommended *(Class I; Level of Evidence B).***
3. **After discharge, it is reasonable to refer patients with aSAH for a comprehensive evaluation, including cognitive, behavioral, and psychosocial assessments *(Class IIa; Level of Evidence B).*** (New recommendation)

neurologstatus

Risk för reblödning

Table Grading scales for subarachnoid hemorrhage

Hunt and Hess[10]	Modified Fisher[11]	World Federation of Neurological Surgeons[12]
1: No symptoms or mild headache	0: No SAH or IVH	Grade 1: GCS 15 without focal deficit
2: Moderate to severe headache, stiff neck	1: Focal or diffuse SAH <1 mm thick; no IVH	Grade 2: GCS 13-14 without focal deficit
3: Drowsy or confused, mild focal neurological deficits	2: Focal or diffuse SAH <1 mm thick; IVH present	Grade 3: GCS 13-14 with focal deficit
4: Stupor, hemiparesis	3: Focal or diffuse SAH >1 mm thick; no IVH	Grade 4: GCS 7-12 with or without focal deficit
5: Coma, decerebrate posturing	4: Focal or diffuse SAH >1 mm thick; IVH present	Grade 5: GCS <7 with or without focal deficit

Abbreviations: GCS, Glasgow Coma Scale; IVH, intraventricular hemorrhage; SAH, subarachnoid hemorrhage.

Vilka kommer till IVA, NUS?

- … och när läggs man istället på NIVA, NUS?

- The Hunt & Hess and the WFNS grade
  - Correlate with patient outcome
- Fischer classification
  - Predict the likelihood of symptomatic cerebral vasospasm

Prediktorer

- **Hur påverkad man är vid insjuknandet predikterar starkt outcome!**

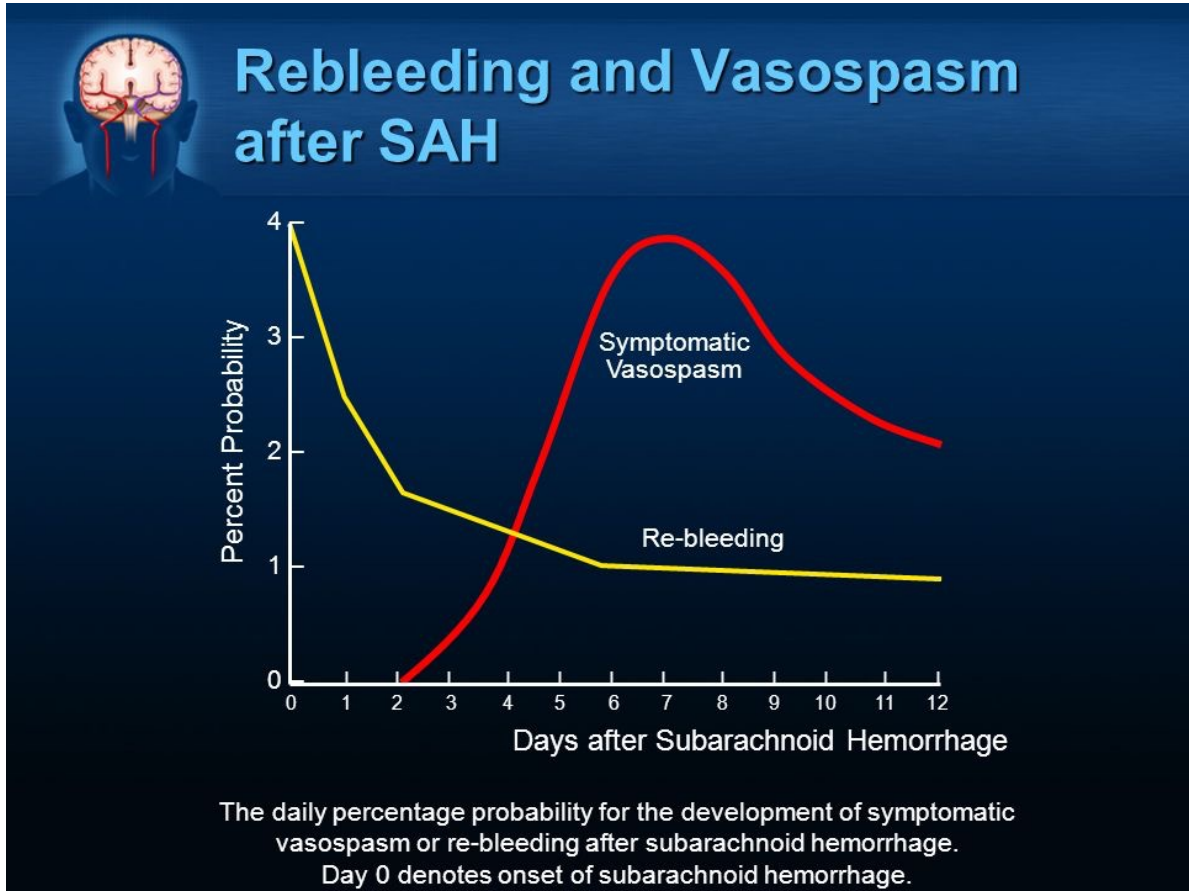
- Preexisting severe medical illness
- Global cerebral edema on CT scan
- IVH & ICH
- Symptomatic vasospasm, delayed cerebral infarction
- Hyperglycemia, fever, anemia

Medical Measures to Prevent Rebleeding After aSAH: Recommendations

1. **Between the time of aSAH symptom onset and aneurysm obliteration, blood pressure should be controlled with a titratable agent to balance the risk of stroke, hypertension-related rebleeding, and maintenance of cerebral perfusion pressure *(Class I; Level of Evidence B)*.** (New recommendation)
2. **The magnitude of blood pressure control to reduce the risk of rebleeding has not been established, but a decrease in systolic blood pressure to <160 mm Hg is reasonable *(Class IIa; Level of Evidence C)*.** (New recommendation)
3. **For patients with an unavoidable delay in obliteration of aneurysm, a significant risk of rebleeding, and no compelling medical contraindications, short-term (<72 hours) therapy with tranexamic acid or aminocaproic acid is reasonable to reduce the risk of early aneurysm rebleeding *(Class IIa; Level of Evidence B)*.** (Revised recommendation from previous guidelines)

Reblödning sker hos 1 av 6 patienter med SAH

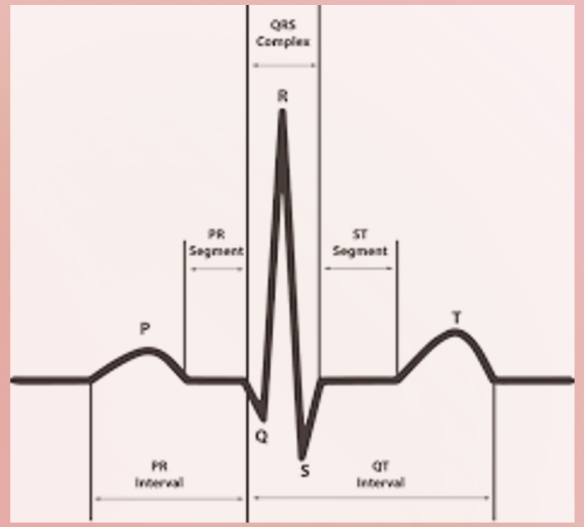
Vanligtvis inom de första 24 timmarna



Tidig organpåverkan

- Katekolaminstorm kan leda till myokardischemi/infarct

- Förekommer I 20-30% av SAH-patienter



Kardiell påverkan…

- Hypertension (27%)
- Hypotension (18%)
- Life-threatening arrhythmia (8%)
- Myocardial ischemia (6%)
- Successful resuscitation from cardiac arrest (4%)
- Troponin I elevation (20%-68%)
- Regional wall motion abnormalities (26%)

ECG Changes

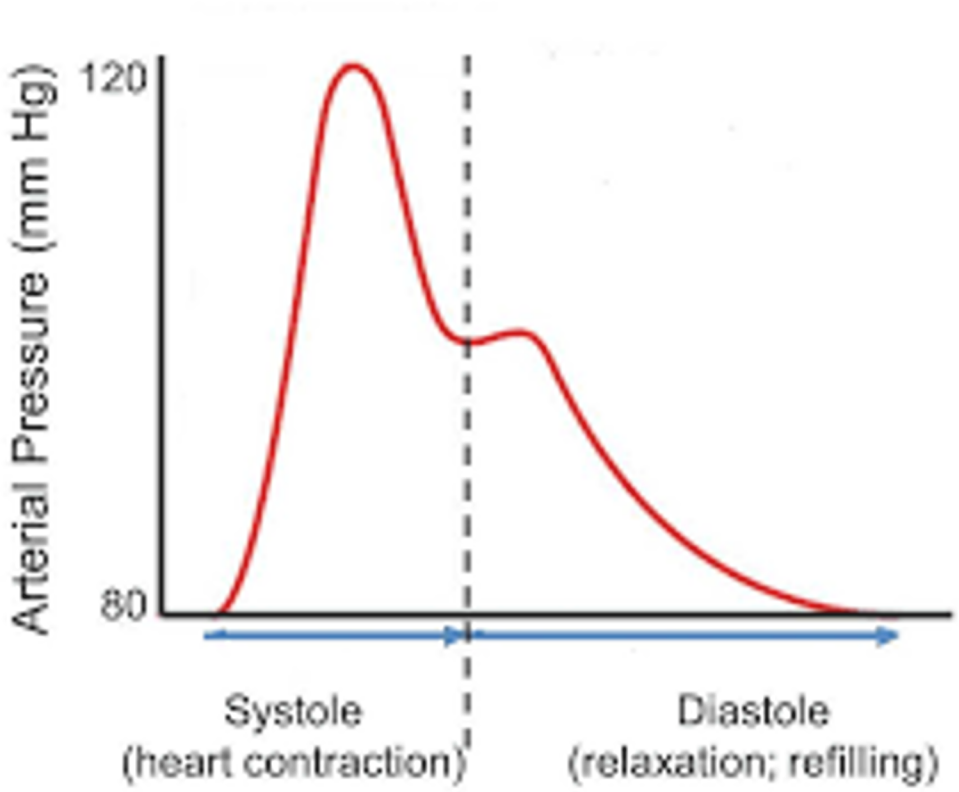
- ST segment changes (15%–67%)
- T-wave changes (12%–92%)
- Prominent U waves (4%–52%)
- QT prolongation (11%–66%)
- Conduction abnormalities (7.5%)
- Sinus bradycardia (16%)
- Sinus tachycardia (8.5%)

Hjärtstunning…

- Most severe form of cardiac injury
- Transient lactic acidosis
- Cardiogenic shock
- Pulmonary edema
- Widespread T-wave inversions
- Prolonged QT interval
- Reversible left ventricular wall motion abnormalities

ALI vanligt ”neurogent lungödem”

- Normovolemi!
- Dobutamin förstahandsval
- Noradrenalin vid behov



- Tidalvolym 6-8 ml/kg
- PaO2>12, PaCO2 5

Kom ihåg!
Vid insjuknande – risk för:

Reblödning

Cirkulations/respirationspåverkan

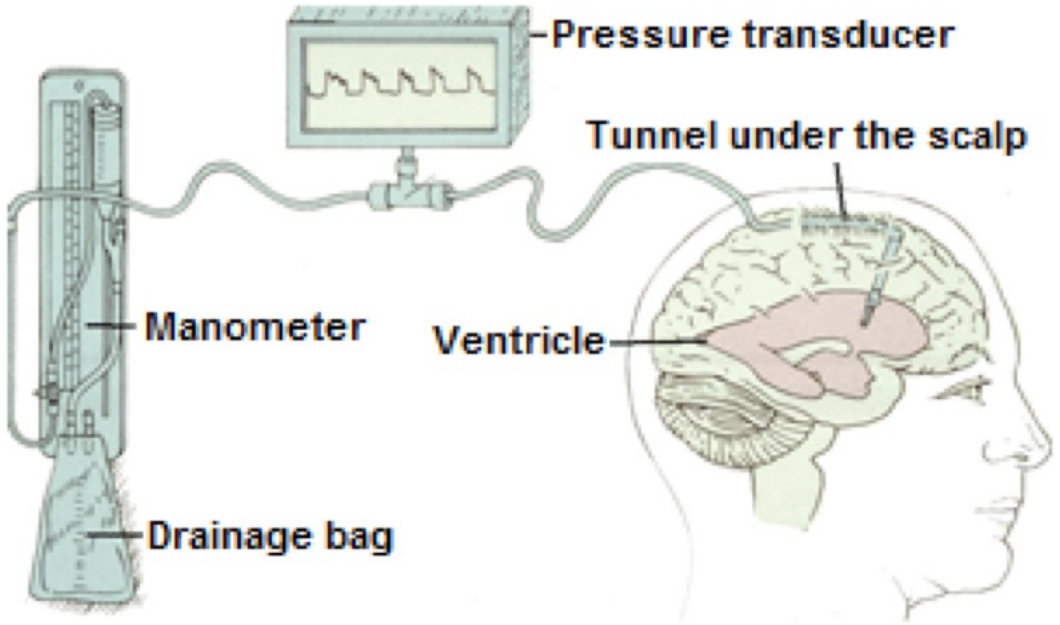
- Initial management of patients with SAH is stabilization. Assess the level of consciousness, airway, breathing and circulation (ABCs).
- Endotracheal intubation should be performed for patients presenting with coma/depressed consciousness/inability to protect airway/ increased ICP.
- Vascular access should be obtained, including central and arterial lines.
- Monitoring
  - Cardiac monitoring
  - Pulse oximetry
  - BP monitoring (arterial BP monitoring is indicated in high-grade SAH)
  - End-tidal carbon dioxide.
  - Urine output measurement via placement of a Foley's catheter.

Kramper?

- Early - 6% to 18%
- Delayed - 3% to 7%
- Risk factors
  - MCA aneurysm
  - Thickness of SAH
  - ICH
  - Re-bleeding
  - Infarction
  - Poor neurological grade
  - History of hypertension
- Routine use of anticonvulsants is not recommended

1. **Low-volume hospitals (eg, <10 aSAH cases per year) should consider early transfer of patients with aSAH to high-volume centers (eg, >35 aSAH cases per year) with experienced cerebrovascular surgeons, endovascular specialists, and multidisciplinary neuro-intensive care services *(Class I; Level of Evidence B).*** (Revised recommendation from previous guidelines)
2. **Annual monitoring of complication rates for surgical and interventional procedures is reasonable *(Class IIa; Level of Evidence C).*** (New recommendation)
3. **A hospital credentialing process to ensure that proper training standards have been met by individual physicians treating brain aneurysms is reasonable *(Class IIa; Level of Evidence C).*** (New recommendation)

Hydrocephalus kan uppstå tämligen snabbt efter SAH



Subarachnoidalt blod förhindrar/förlångsammar normal återresorption av CSF

Complications of SAH

Delayed Neurological Deteriorations, Delayed Cerebral Ischemia, and Vasospasm

Hyponatremia

Hydrocephalus

Anemia

Fever

Pain

Seizures

Poor Glucose Control

Changes in Intravascular Volume

EKG
Artärnål
Troponin dagligen
HjärtEKO
ev PiCCO

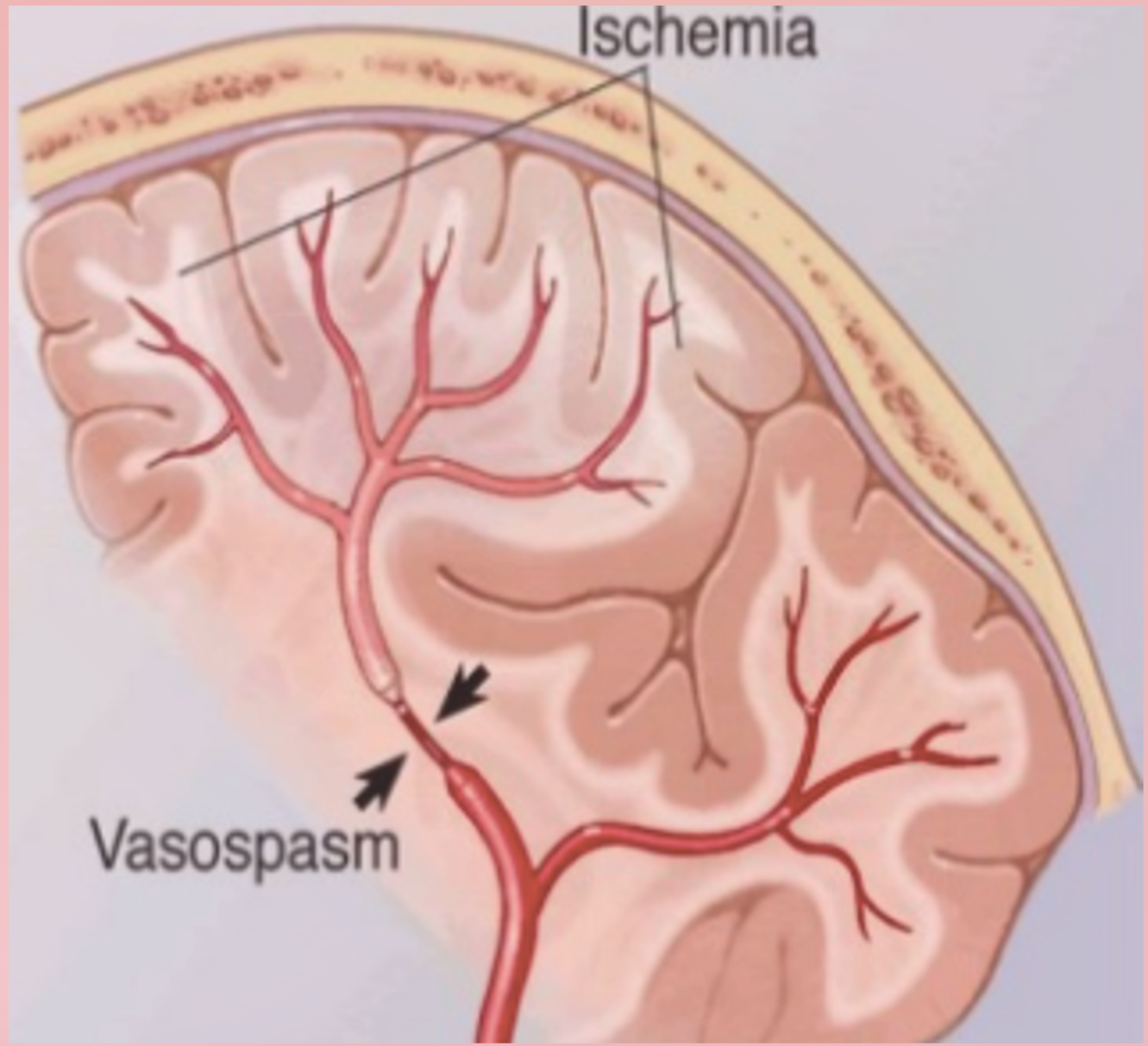
2. **Monitoring volume status in certain patients with recent aSAH by some combination of central venous pressure, pulmonary wedge pressure, and fluid balance is reasonable, as is treatment of volume contraction with crystalloid or colloid fluids *(Class IIa; Level of Evidence B).***

Vasospasm

Vanligen efter dygn 3

Peak dag 6-8

Vanligtvis över efter 3 veckor

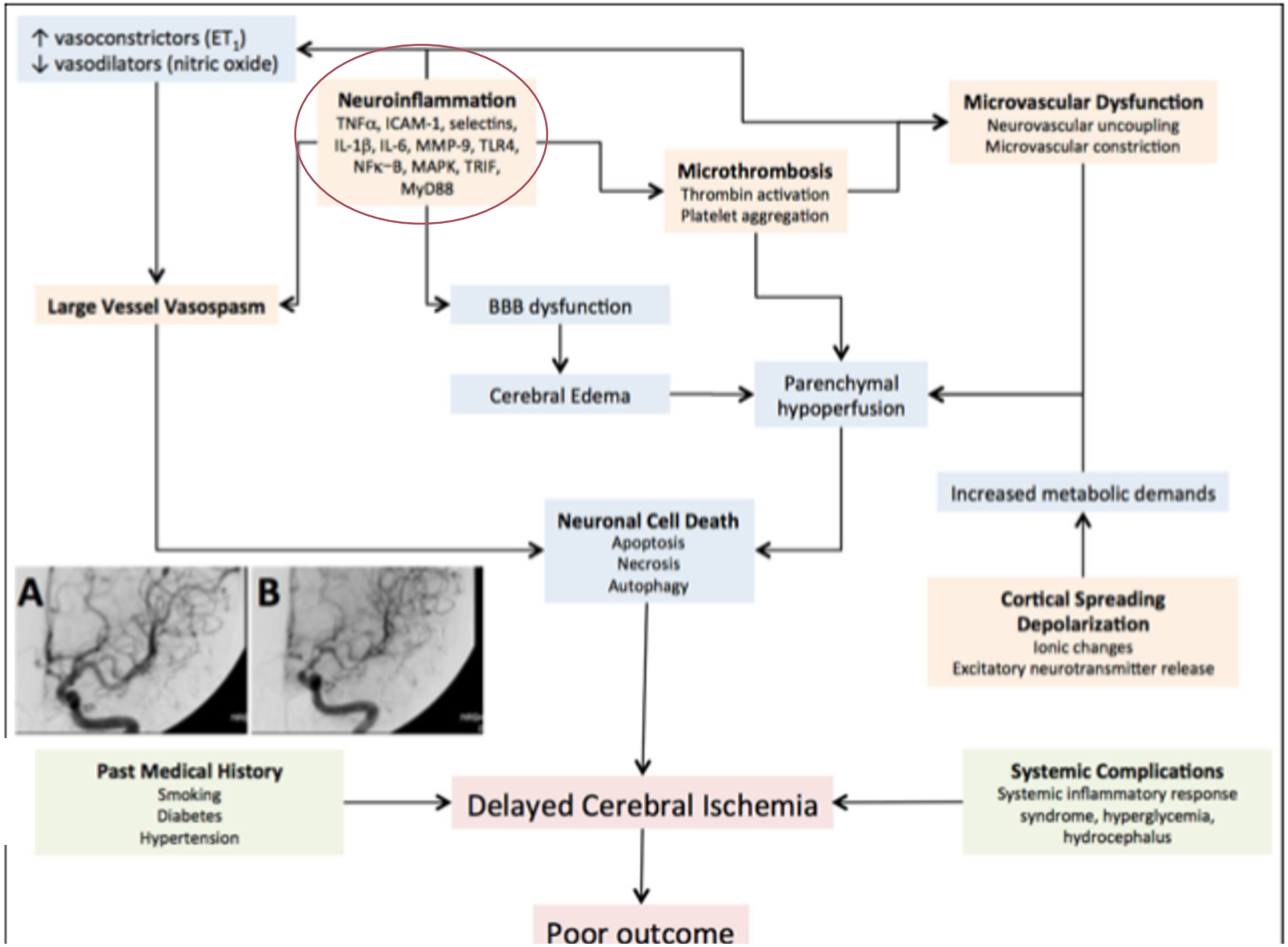


- Aggressiv volymterapi -
- normovolemi!
- (plasma, albumin, blod)
- Högt blodtryck kan behövas!
- Diskutera med NKK

Management of Cerebral Vasospasm and DCI After aSAH: Recommendations

1. **Oral nimodipine should be administered to all patients with aSAH *(Class I; Level of Evidence A)*.** (It should be noted that this agent has been shown to improve neurological outcomes but not cerebral vasospasm. The value of other calcium antagonists, whether administered orally or intravenously, remains uncertain.)
2. **Maintenance of euvolemia and normal circulating blood volume is recommended to prevent DCI *(Class***

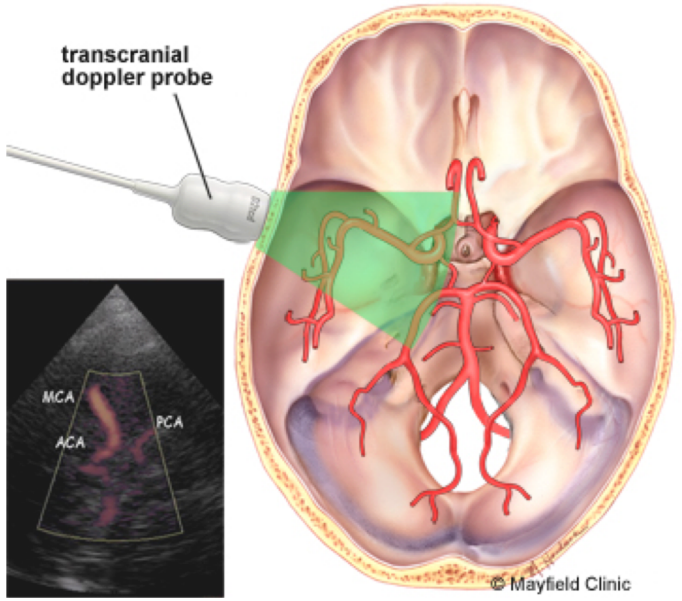
Fig. 2 Pathogenesis of delayed cerebral ischemia after SAH. The main mechanisms under investigation include large vessel vasospasm, microcirculatory dysfunction, microthrombosis, cortical spreading depolarizations, and neuroinflammation. Catheter angiography showing baseline (A) and severe vasospasm (B) at day 5 after SAH



Curr Atheroscler Rep (2017) 19: 50
https://doi.org/10.1007/s11883-017-0690-x

- …förebyggs/behandlas med Nimodipin iv
- risk för shuntning
- risk för hypotension

- daglig transkraniell doppler (ökad flödeshastighet indikerar spasm)



Mean MCA velocity	MCA : ICA (Lindegaard Ratio)	Interpretation
< 120 cm/sec	< 3	Normal
120- 200 cm/sec	3 -6	Mild vasospasm
> 200 cm/sec	> 6	Severe vasospasm

I; Level of Evidence B). (Revised recommendation from previous guidelines)

3. **Prophylactic hypervolemia or balloon angioplasty before the development of angiographic spasm is not recommended *(Class III; Level of Evidence B).*** (New recommendation)
4. **Transcranial Doppler is reasonable to monitor for the development of arterial vasospasm *(Class IIa; Level of Evidence B).*** (New recommendation)
5. **Perfusion imaging with CT or magnetic resonance can be useful to identify regions of potential brain ischemia *(Class IIa; Level of Evidence B).*** (New recommendation)
6. **Induction of hypertension is recommended for patients with DCI unless blood pressure is elevated at baseline or cardiac status precludes it *(Class I; Level of Evidence B).*** (Revised recommendation from previous guidelines)
7. **Cerebral angioplasty and/or selective intra-arterial vasodilator therapy is reasonable in patients with symptomatic cerebral vasospasm, particularly those who are not rapidly responding to hypertensive therapy *(Class IIa; Level of Evidence B).*** (Revised recommendation from previous guidelines)

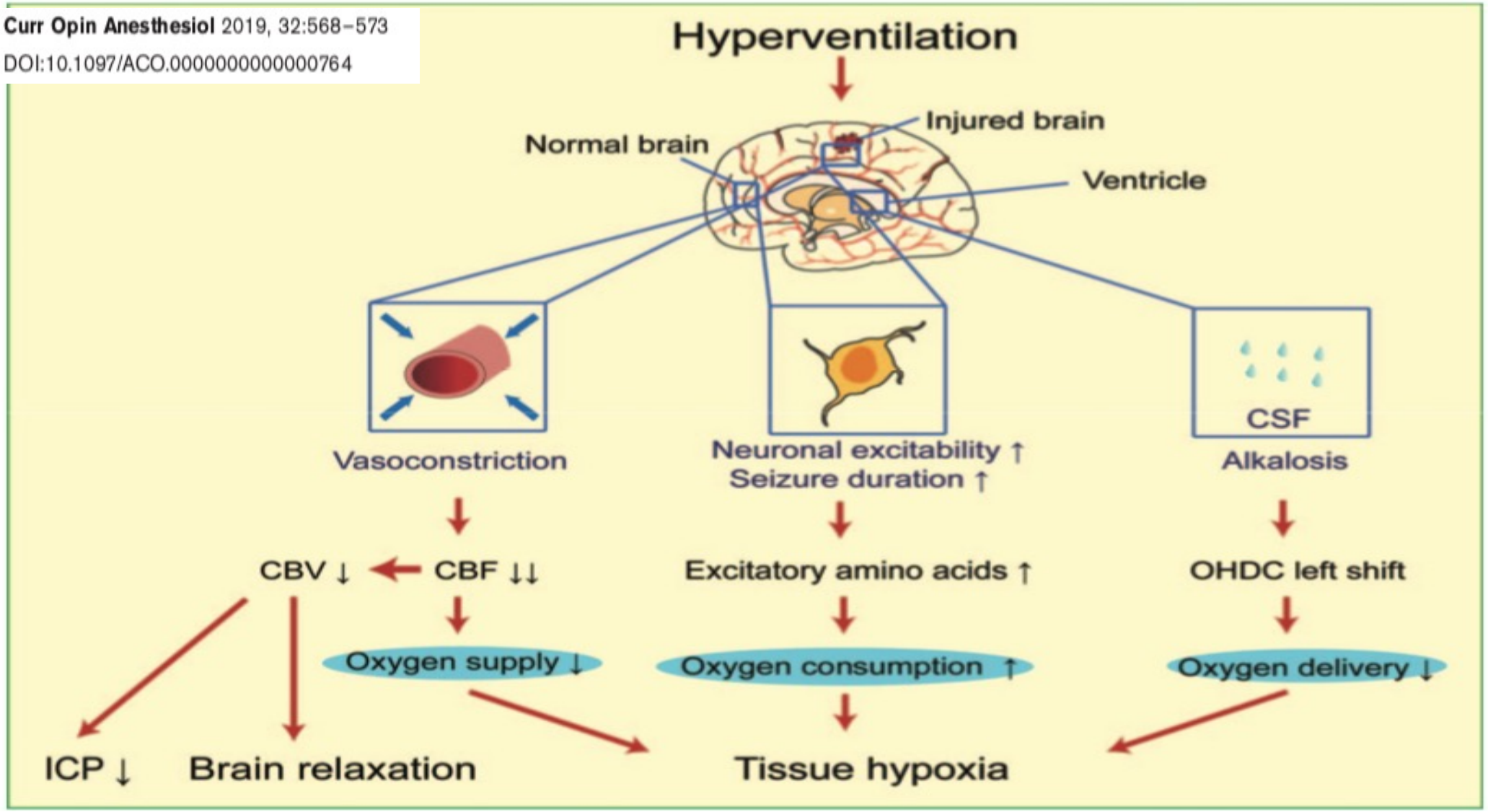
	CSW	SIADH	Diabetes insipidus
Volume Status	Hypovolemia	Normovolemic or hypervolemia	Hypovolemia
Serum Sodium Concentration	Decreased	Decreased	Increased
Urine Sodium Concentration	Increased	Increased	Decreased
Urine Output	Increased	Normal	Increased
Mechanism	Excess secretion of sodium and water	Water retention due to elevated ADH (vasopresin)	Free water loss due to decreased ADH (vasopresin)

Vätske och elektrolytrubbningar är vanliga

- Optimalt Hb?
- Vetenskap saknas.
- Pga hypoxirisk vid vasospasm - Hb>100 g/L

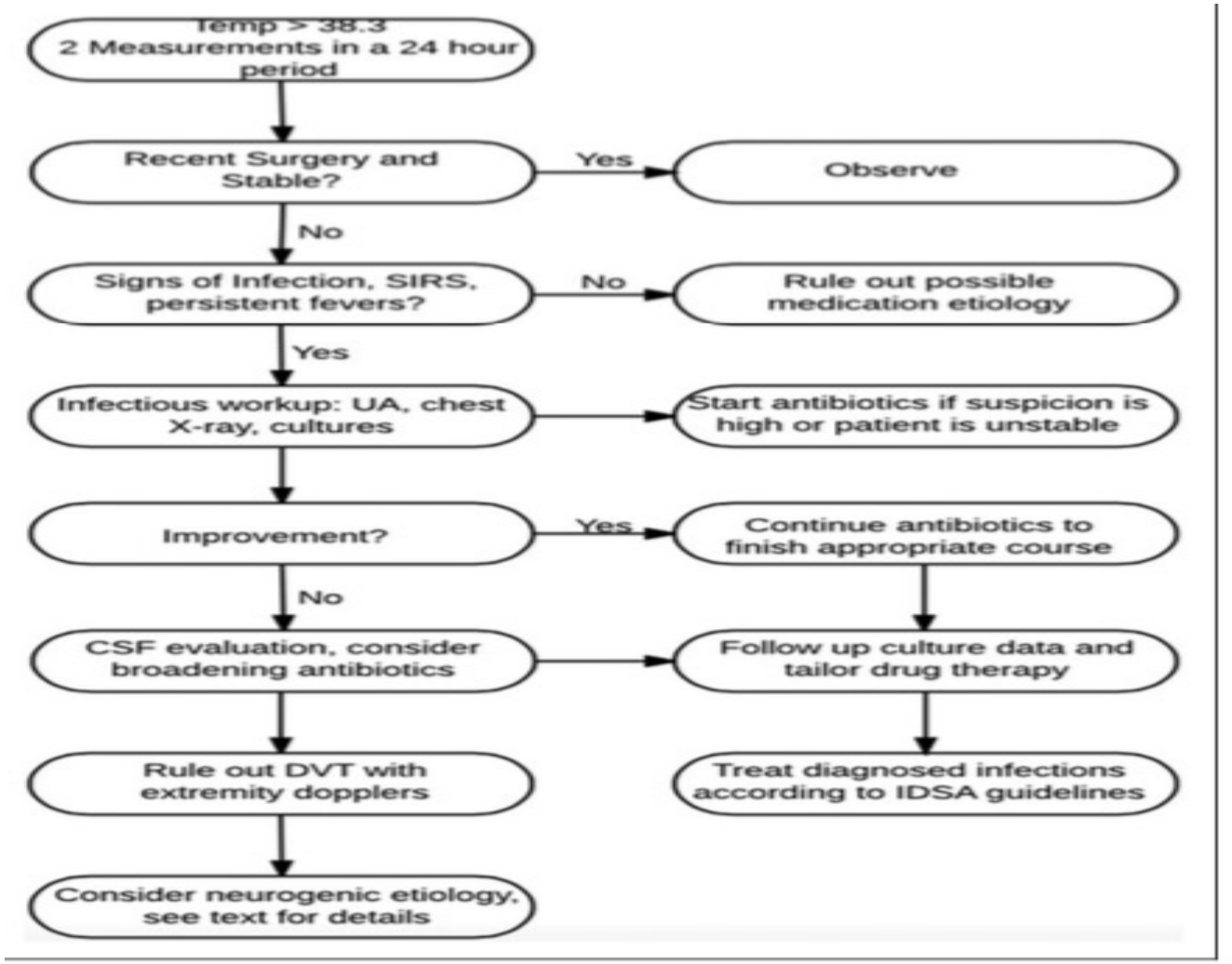
5. **The use of packed red blood cell transfusion to treat anemia might be reasonable in patients with aSAH who are at risk of cerebral ischemia. The optimal hemoglobin goal is still to be determined *(Class IIb; Level of Evidence B).*** (New recommendation)

Curr Opin Anesthesiol 2019, 32:568–573
DOI:10.1097/ACO.0000000000000764



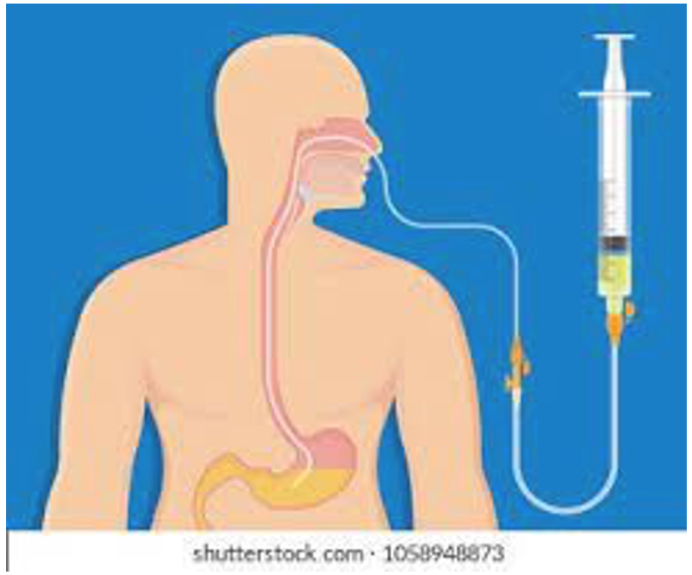
FEBER

3. **Aggressive control of fever to a target of normothermia by use of standard or advanced temperature modulating systems is reasonable in the acute phase of aSAH *(Class IIa; Level of Evidence B).*** (New recommendation)


Temp > 38.3
2 Measurements in a 24 hour period
Recent Surgery and Stable?
Yes
Observe
No
Signs of Infection, SIRS, persistent fevers?
No
Rule out possible medication etiology
Yes
Infectious workup: UA, chest X-ray, cultures
Start antibiotics if suspicion is high or patient is unstable
Improvement?
Yes
Continue antibiotics to finish appropriate course
No
CSF evaluation, consider broadening antibiotics
Follow up culture data and tailor drug therapy
Rule out DVT with extremity dopplers
Treat diagnosed infections according to IDSA guidelines
Consider neurogenic etiology, see text for details

Trombosprofylax

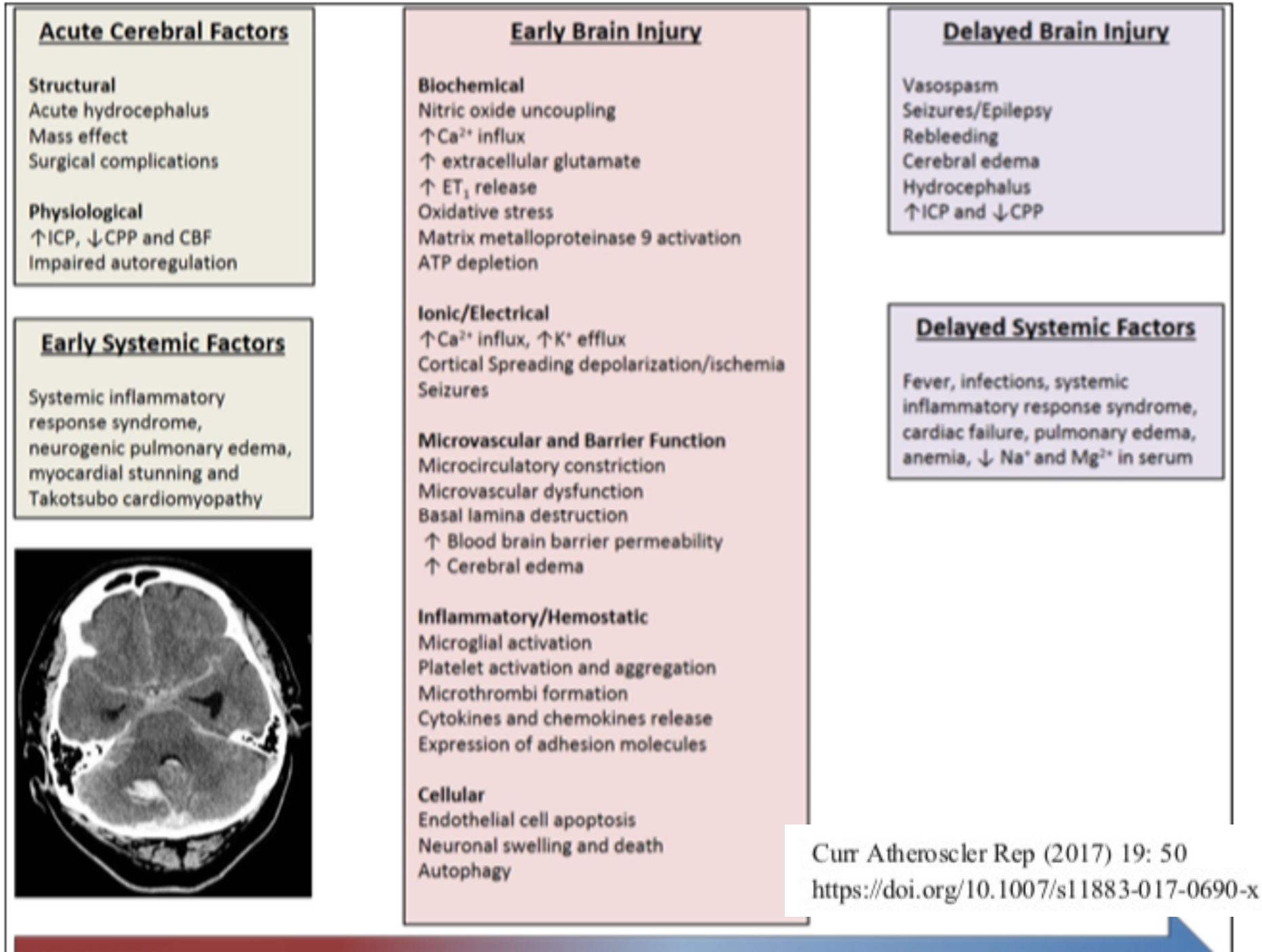
… men inte första dygnet efter åtgärd – OM INTE NEURORADIOLOGEN ORDINERAT ANNAT!



4. **Careful glucose management with strict avoidance of hypoglycemia may be considered as part of the general critical care management of patients with aSAH *(Class IIb; Level of Evidence B)*.**

- Tidig start EN
- 20 kcal/kg/dygn
- B-glucos 5-10 mmol/L

Fig. 1 Factors contributing to brain injury in SAH. Following aneurysm rupture, injury can be divided into early and delayed stages, which include both systemic and cerebral factors. Head CT scan of a 40-year-old woman with subarachnoid hemorrhage showing right cerebellar hemorrhage with local mass effect, intraventricular extension, and early hydrocephalus represented by the distention noted at the temporal horns of the lateral ventricles



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outcome

Modified Ranking Scale

Modified Rankin Scale (MRS)	
0	No symptoms
1	No significant disability, despite symptoms; able to perform all usual duties and activities
2	Slight disability; unable to perform all previous activities but able to look after own affairs without assistance
3	Moderate disability; requires some help, but able to walk without assistance
4	Moderately severe disability; unable to walk without assistance and unable to attend to own bodily needs without assistance
5	Severe disability; bedridden, incontinent, and requires constant nursing care and attention
6	Death

förväxla inte TBI och SAH
Två olika fysiologiska resonemang


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