SAFETY

simulation for medical practice

SIMULATION APPROACH FOR EDUCATION AND TRAINING IN EMERGENCY

Neurological & Psychiatric Emergencies

Katharina Grujic, LMU

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AUTHORS

Katharina Grujic

Summary

STROKE

1. Abstract

The majority of strokes are ischemic strokes (cerebral infarctions). These lead to an acute cerebral circulatory disorder (e.g. due to stenosis) in the arterial flow area. The main symptoms are a newly occurring hemiparesis, speech and vision disorders, with a wide variety of impairments being possible depending on the location of the lesion. The sudden onset of symptoms is also characteristic. The most important diagnostic procedure is the cranial CT to rule out a causal bleeding. In the case of an ischemic stroke, the aim should then be to recanalize the area as quickly as possible in order to save the area of relative ischemia - **"time is brain!"**

2. Symptoms

A stroke is defined as a sudden onset and persistent or spontaneously remitting neurological deficit due to focal brain dysfunction.

ISCHEMIC AND HEMORRHAGIC STROKE

There are two main types of strokes, the ischemic stroke and the hemorrhagic stroke.

The most common type is the ischemic stroke (about 80-85% of all strokes), while hemorrhagic stroke is less often (about 10-15% of all strokes). In the ischemic stroke, plaque or blood clot interrupts the blood flow in a brain artery. The hemorrhagic stroke is causally related with blood vessel rupture or an abnormal vascular structure, like aneurysm, with bleeding in the brain.

THROMBOTIC STROKES

Intracranial atherosclerosis may be the cause of thrombotic stroke in patients with widespread atherosclerosis.

Thrombotic strokes are generally thought to originate on ruptured atherosclerotic plaques. Arterial stenosis can cause turbulent blood flow, which can promote thrombus formation and platelet adherence. All cause the formation of blood clots that either embolize or occlude the artery.

In especially younger patients, other causes should be considered, including the following:

- Hypercoagulable states (e.g., antiphospholipid antibodies, protein C deficiency, protein S deficiency, pregnancy)
- Sickle cell disease
- Fibromuscular dysplasia
- Arterial dissections
- Vasoconstriction associated with substance abuse (e.g., cocaine, amphetamines)

Signs and symptoms of a stroke can present in different ways and often develop quickly. The type of symptoms depends on the brain area that is affected. Cardinal symptoms of the stroke can be an acute focal neurological deficit. Most of the neurological deficits can be assigned to a specific arterial supply area:

Anterior Cerebral Artery	Cortical branches: medial frontal and parietal lobe Medial lenticulostriate branches: caudate head, globus pallidus, anterior limb of internal capsule

Middle Cerebral Artery	Cortical branches: lateral frontal and parietal lobes, lateral and anterior temporal lobe Lateral lenticulostriate branches: globus pallidus and putamen, internal capsule
Anterior Choroidal Artery	Optic tracts, medial temporal lobe, ventrolateral thalamus, corona radiata, posterior limb of the internal capsule
Posterior Cerebral Artery	Cortical branches: occipital lobes, medial and posterior temporal and parietal lobes Perforating branches: brainstem, posterior thalamus and midbrain
Posterior Inferior Cerebellar Artery	Inferior vermis; posterior and inferior cerebellar hemispheres
Anterior Inferior Cerebellar Artery	Anterolateral cerebellum
Superior Cerebellar Artery	Superior vermis; superior cerebellum

Stroke should be considered in any patient presenting with acute neurologic deficit or any alteration in level of consciousness. Common stroke signs and symptoms may include the following:

- Abrupt onset of hemiparesis, monoparesis, or (rarely) quadriparesis
- Hemisensory deficits
- Monocular or binocular visual loss
- Visual field deficits
- Diplopia
- Dysarthria
- Facial droop
- Ataxia
- Vertigo (rarely in isolation)
- Nystagmus
- Aphasia
- Sudden decrease in level of consciousness

ANY NEUROLOGICAL DEFICIT THAT OCCURS SUDDENLY INDICATES A STROKE AND MUST BE TREATED AS AN EMERGENCY!

SYMPTOMS ACCORDING TO AFFECTED BRAIN AREA

- **Infarcts in cortical areas**
  - Frontal: drive depleted, olfactory disorder, Broca's aphasia
  - Temporally: Anxious to irritable mood, prone to epilepsy, Wernicke's aphasia
  - Parietal: Constructive apraxia, aphasia, neglect
  - Coat edge syndrome: sensorimotor paresis of the legs, possibly with bladder disorders

- **Infarcts in subcortical areas**
  - Capsula interna: Contralateral hemiparesis and contralateral cranial nerve deficits
  - Thalamus, basal ganglia, frontal medulla (so-called "strategic centers"): disturbance of cognition, memory, orientation and practical skills
  - Thalamus isolated: Contralateral hemidysesthesia, finger hyperkinesia,
  - Cerebellar infarctions:
    - Cerebellar ataxia
    - gait ataxia
    - trunk ataxia
    - dysmetria
    - dysdiadochokinesis
    - dysarthria
    - muscle hypotonia

ANAMNESIS

- Identification of the symptoms:
  What symptoms or deficits have occurred?
  Is there or was there a relevant disorder of language, speech, vision, sensorimotor function or coordination?
- Temporal characteristics of the symptoms:
  Onset (sudden)? Dynamic (ongoing or already remitted)? Exact time window known?
  - Start: Are the symptoms apoplectiform, i.e. did they appear suddenly in their full manifestation?
  - Dynamics: Are there persistent deficits or are the symptoms already declining or completely remitted?
  - Exact time window: What time did the symptoms appear or when was the last time when you were definitely symptom-free?

COMPLETION OF THE ANAMNESIS BEYOND THE CLARIFICATION OF THE ABOVE QUESTIONS TAKES PLACE PARALLEL TO THE MEASURES THAT NOW FOLLOW, WITHOUT DELAYING THEM!

- Previous vascular diseases?
- Known cardiovascular risk factors?
- Known disease with increased risk of bleeding?
- Are there other pre-existing conditions?
- Premedication?

COMPLICATIONS

- Intracranial pressure
  Intracranial pressure elevations may occur following ischemic infarction on the basis of secondary hemorrhage and/or the development of cerebral edema.

- Secondary bleeding

- Iatrogenic
- Postoperative cerebral hyperperfusion syndrome
  - Definition: Sudden hyperperfusion of a cerebral flow area hours to days after an ischemic event with subsequent interventional revascularization
  - Clinic: One-sided headache, focal neurological deficits, epileptic seizures with focal onset, signs of intracranial pressure
  - Therapy: Medicinal blood pressure reduction, e.g. by administering urapidil or (in the case of existing tachycardia) short-acting beta blockers (e.g. esmolol)
  - Clinically relevant cerebral edema: especially in malignant media infarction and space-occupying cerebellar infarction

- Malignant media infarction
  - Definition:
    - Extensive ischemia in the middle cerebral artery (usually >⅔ of the middle cerebral artery) after occlusion of the M1 section of the middle cerebral artery or the distal internal carotid artery ("carotid T") with the development of cerebral edema and resulting cerebral edema Compression of the lateral ventricles and midline shift. The frequency is up to 10% of media infarcts. The development of cerebral edema usually occurs within the first 2-5 days after the infarction
  - Clinic:
    - Signs of intracranial pressure in addition to symptoms of the medial infarctio

- Therapy
  - General: Early intubation, ventilation and positioning appropriate to the situation
  - Operative intracranial pressure reduction: decompressive hemicraniectomy, possibly with subsequent hypothermia
  - As early as possible (within 48 hours) after the event
  - In the case of sequelae that have already occurred (e.g. signs of entrapment), the benefit of a relief trepanation is unclear
  - Subsequent possible therapeutic hypothermia (32–34 °C for 24–72 h)
  - Conservative lowering of intracranial pressure: only if hemicraniectomy is contraindicated/rejected
  - Possibly osmotherapy (e.g. mannitol)
  - Possibly hypothermia (32–34 °C for 24–72 h)
- Epileptic seizures
- Dysphagia
- Cardiac complications
- Post-stroke-depression

3. Differential Diagnose

- Hypoglycaemia
- Migraine with aura
- Epileptic attack
- Severe infection or sepsis
- Peripheral nerve damage
- Alcohol intoxication

4. Diagnostic and skill needed

Initial assessment and basic measures

- Checking and securing of the vital functions according to the ABCDE scheme
- Monitoring of vital parameters: 12-lead ECG (with diagnosis!), heart rate, blood pressure, respiration, SpO2, body temperature
- Intravenous access: secure a stable IV Access
- Blood gas analysis with glucose measurement
- Basic laboratory diagnostics for stroke: complete blood count, CRP, glucose, electrolytes (Na+, K+), lactate, creatinine, urea, GFR, bilirubin, ALT, AST, γ-GT, troponin, CK, TSH, INR, pTT, ethanol

Signs of a stroke can be different. The F-A-S-T test is an easy way to remember them:

- **F = Face Drooping** – Does one side of the face droop or is it numb? Ask the person to smile. Is the person's smile uneven?
- **A = Arm Weakness** – Is one arm weak or numb? Ask the person to raise both arms. Does one arm drift downward?
- **S = Speech Difficulty** – Is speech slurred?
- **T = Time to call**

Other Stroke Symptoms

Watch for Sudden:

- **NUMBNESS** or weakness of face, arm, or leg, especially on one side of the body
- **CONFUSION**, trouble speaking or understanding speech
- **TROUBLE SEEING** in one or both eyes
- **TROUBLE WALKING**, dizziness, loss of balance or coordination
- **SEVERE HEADACHE** with no known cause

Essential components of the neurologic examination include the following evaluations:

- Cranial nerves
- Motor function
- Sensory function
- Cerebellar function
- Gait
- Deep tendon reflexes
- Language (expressive and receptive capabilities)
- Mental status and level of consciousness

	Neurological examination	Pathological findings
Speech	• Assessment of speech comprehension, speech production and articulation in patient conversations • Bedside testing using image/object description, repeating	• Impaired language comprehension and/or language production: aphasia • Impaired articulation: dysarthria
Visual field	Perimetry in lateral comparison of the eyes	• Monocular visual loss • Homonymous hemianopsia or quadrant anopia

Oculomotor skills	• Inspection: Pupil size, • Spontaneous eyeball position, gaze position • Random gaze movements in all directions	• Bilateral miosis or mydriasis, anisocoria • Gaze deviation • Paralysis • Saccaded gaze sequence
Facial motor skills	• Inspection: position of the corners of the mouth, salivation from a corner of the mouth, nasolabial folds • Checking of frown, eyelid closure, lip motor function, also against resistance	Central facial paresis
Facial sensitivity	Checking the sensitivity in the different facial regions	Lateralized sensory loss
Brainstem reflexes	• Pupillary reflex (direct and indirect)· • Corneal reflex • Vestibulo-ocular reflex • Gag reflex	Reflex failure or weakening
Force test	Raising arm/leg	Unilateral weakness/paresis
Reflexes	• Triceps reflex • Abdominal skin reflexes (especially in suspected paraplegia symptoms) • Patellar tendon reflex • Achilles's tendon reflex • Babinski reflex	• Reflexes weakened/increased • Babinski reflex positive

Tested item	Title	Score	Test results
1A	**Level of consciousness**		
		0	Alert; Responsive
		1	Drowsy
		2	Obtunded
		3	Coma/unresponsive
1B	**Orientation questions**		
		0	Correctly answers both questions
		1	Correctly answers one question
		2	Does not correctly answer either question
1C	**Response to commands**		
		0	Correctly performs both tasks
		1	Correctly performs 1 task
		2	Does not correctly perform either task
2	**Gaze**		
		0	Normal; horizontal movements
		1	Partial gaze palsy
		2	Total gaze paresis
3	**Visual field**		
		0	No visual field defect
		1	Partial hemianopia
		2	Complete hemianopia
		3	Bilateral Blindness

4	**Facial movement**		
		0	Normal
		1	Minor facial weakness
		2	Partial facial weakness
		3	Complete unilateral palsy
5	Motor function (arm) a. left b. right		
		0	No arm drift
		1	Drift before 10s
		2	Falls before 10s
		3	No effort against gravity
		4	No movement
6	Motor function (leg) a. left b. right		
		0	No leg drift
		1	Drift before 5s
		2	Falls before 5s
		3	No effort against gravity
		4	No movement
7	Limb Ataxia		
		0	no ataxi
		1	Ataxia present in 1 limb
		2	Ataxia present in 2 or more limbs
8	Sensory		
		0	No evidence of sensory loss
		1	Mild sensory loss
		2	Severe sensory loss
9	Language		
		0	Normal
		1	Mild aphasia
		2	Severe aphasia
		3	mute or global aphasia
10	Articulation		
		0	Normal
		1	Mild dysarthria
		2	Severe dysarthria
11	Extinction and inattention		
		0	Absent
		1	Mild loss (1 sensory modality lost)
		2	Severe loss (2 modalities lost)

5. Therapy and skills needed

The goal for the emergent management of stroke is to complete the following within **60 minutes or less** of patient arrival:

- Assess airway, breathing, and circulation (ABCs) and stabilize the patient as necessary
- Complete the initial evaluation and assessment, including imaging and laboratory studies
- Initiate reperfusion therapy, if appropriate

Prehospital management in ischemic stroke

- Pre-registration and transport to a hospital with a stroke unit
- Positioning: preferably 30° upper body elevation
- Monitoring of vital parameters (ECG, heart rate, blood pressure, respiratory rate, oxygen saturation, temperature)
- Needs-based safeguarding/stabilization of vital functions
    - Administration of 2–4 L of oxygen via a nasal tube (goal: ≥95% SaO2)
    - Generally, tolerate arterial hypertension since perfusion of the penumbra is directly dependent on the mean arterial pressure (MAP).
    - Arterial hypotension: research into causes (cardiac arrhythmias, reduced ejection capacity or hypovolemia?)
        - If necessary, add volume (crystalloid solutions)
        - If necessary, norepinephrine

NO ADMINISTRATION OF COAGULATION-ACTIVE SUBSTANCES!

Critical treatment decisions focus on the following:

- The need for airway management
- Optimal blood pressure control
- Identifying potential reperfusion therapies (e.g., intravenous fibrinolysis with rt-PA (alteplase) or intra-arterial approaches
- CT imaging

Basic neuroprotective therapy in ischemic stroke

- Blood pressure management
- Antihypertensive therapy only if critical blood pressure limits are exceeded
- Aim for mild hypertension in the first few days after stroke
- Avoid rapid and drastic lowering of blood pressure
- Consistent treatment of hypotension

- Target values:

1. In patients with acute ischaemic stroke not treated with intravenous thrombolysis or mechanical thrombectomy and blood pressure >220/120mmHg, careful blood pressure reduction (<15% systolic blood pressure reduction in 24hours) is reasonable and likely to be safe. No specific blood pressure lowering agent can be recommended.
2. In patients with acute ischaemic stroke undergoing treatment with intravenous thrombolysis (with or without mechanical thrombectomy) we suggest maintaining blood pressure below 185/110mmHg before bolus and below 180/105mmHg after bolus, and for 24 hours after alteplase infusion. No specific blood pressure-lowering agent can be recommended.
3. In patients with acute ischaemic stroke undergoing treatment with intravenous thrombolysis (with or without mechanical thrombectomy) we suggest against lowering systolic blood pressure to a target of 130–140mmHg compared to <180mmHg during the first 72 hours following of symptom onset.
4. In patients with hyperacute (<6hours) intracerebral haemorrhage, we suggest lowering blood pressure to below 140mmHg (and to keep it above 110mmHg) to reduce haematoma expansion.

IN THE CASE OF AN ACUTE ISCHEMIC APOPLEXY, BLOOD PRESSURE SHOULD GENERALLY NOT BE LOWERED WITH MEDICATION!

- Glycemic control, avoidsevere hyperglycaemia
  - Correction: from ≥200 mg/dL, usually with insulin
  - Prevention of hyperthermia, goal of normothermia <37.5 °C
  - Prevent electrolyte disequilibrium
  - Consider a nasogastric tube for feeding
- Initially hold oral anticoagulants
- Start thromboprophylaxis and antiplatelet drugs only after exclusion of intracranial bleeding with CT

Recanalization strategies, including intravenous recombinant tissue-type plasminogen activator (alteplase or rt-PA) and intra-arterial approaches, attempt to establish revascularization so that cells within the ischemic penumbra can be rescued before irreversible injury occurs. Restoring blood flow can mitigate the effects of ischemia only if performed quickly.
In particular, rt-PA must be given within 3 hours of stroke onset and only after CT scanning has ruled out hemorrhagic stroke.

Based on recent European data, the American Heart Association and American Stroke Association recommended expanding the treatment window from 3 hours to 4.5 hours, with more stringent exclusion criteria for the later period.
The FDA has not approved rt-PA for this expanded indication, but this has become the community standard in many institutions.

Updated guidelines from the American Heart Association (AHA) and the American Stroke Association (ASA) extend the time limit on mechanical clot removal from 6 to 24 hours in select patients.
The new guidelines recommend thrombectomy in eligible patients 6 to 16 hours after a stroke. They also broaden the eligibility criteria by allowing patients ineligible for IV tPA to undergo mechanical thrombectomy within 6 hours.

HEMOSTASIS AND COAGULOPATHY RECOMMENDATIONS
Door to Treatment 90 minutes. Do not delay transfer to administer these treatments
1. Patients with severe coagulation factor deficiency or severe thrombocytopenia should receive appropriate factor replacement therapy

2. Confer with receiving hospital provider about treatments prior to transfer and possible delays in transfer

3. Consider reversal options if available:
- If elevated INR due to vitamin K antagonist (Warfarin), consider:
  - 4PCC (KCentra) - recommended: INR 1.8-3.9: 25 units/Kg (max. 2500 units), INR 4-6: 35 units/kg (max 3500 units), INR >6: 50 units/Kg (max. 5000 units).
  - IV vitamin K: recommended dose is 5 to 10 mg. The effect takes 12 – 24 hours to set in.
  - Fresh Frozen Plasma (FFP): dose will depend on INR. Several units might be needed. A practical formula is 1-2 units up to 20 ml/Kg. May repeat every 6-12 hours
- For patients with ICH and history of using dabigatran, rivaroxaban, or apixaban treatment:
  - Pradaxa:
    - Idarucizumab (Praxbind): recommended dose is 5g IV x 1 either bolus or infusion.
  - Apixaban/Rivaroxaban:
    - Andexanet Alfa: dosing will depend on patient's current apixaban or rivaroxaban dose
    - Low dose: 400 mg IV bolus ~ 30 mg/min followed by an IV infusion of 4 mg/min up to 120 minutes (low dose is Apixaban ≤ 5mg / Rivaroxaban ≤ 10 mg)
    - High dose: 800 mg IV bolus followed by an IV infusion of 8 mg/min up to 120 minutes (high dose: Apixaban > 5mg / Rivaroxaban > 10 mg or unknown dose) iii.
    - Consider 4PCC (Kcentra) 50 units/Kg (max. 5000 units)

Administration of thrombolysis with rt-PA
- Requirement: systolic blood pressure <185 mmHg and diastolic blood pressure <110mmHg
- Start as soon as possible within 4.5 hours after the onset of symptoms with continuous monitoring of vital parameters including close blood pressure monitoring
- If there are no indications of a coagulation disorder and no oral anticoagulation in the previous medication, it is also possible to start before the coagulation diagnostics arrive
- Start of lysis therapy immediately after imaging (0.9 mg/kg body weight IV tPA, of which 10% as a bolus in the 1st minute and 90% over 60 minutes)

Updated guidelines from the American Heart Association (AHA) and the American Stroke Association (ASA) extend the time limit on mechanical clot removal from 6 hours to up to 24 hours in select patients.
The new guidelines recommend thrombectomy in eligible patients 6 to 16 hours after a stroke. They also broaden the eligibility criteria by allowing patients who are ineligible for IV tPA to undergo mechanical thrombectomy within 6 hours.

SYSTEMIC LYSIS THERAPY IS THE ONLY VIABLE RECANALIZING THERAPY FOR MOST PATIENTS WITH CEREBRAL INFARCTION THAT CAN PREVENT PERSISTENT DISABILITY. THE DECISION AGAINST LYSIS THERAPY SHOULD THEREFORE ONLY BE MADE IF THERE ARE ABSOLUTE CONTRAINDICATIONS AND AFTER CAREFUL BENEFIT/RISK ASSESSMENT!

Possible complications during/after lysis therapy

- Intracranial hemorrhage: worsening of the neurological deficit, impaired vigilance, rapid increase in blood pressure, headache, vomiting
  Proceed:
  - Discontinue lysis therapy
  - Emergency CT control
- Extracranial hemorrhage: Symptoms vary depending on localization
- Angioneurotic edema: 30 to 120 min after the start of the infusion, mostly mild swelling of the lips and tongue, then extension to the oropharynx
  Proceed:
  - Discontinue lysis therapy
  - Emergency therapy with i.v. Glucocorticoid , H2 blocker and H1 blocker

Seizures and Anti-convulsant treatment

- Prophylactic anti-convulsant medication is not recommended
- Clinical seizures should be treated with anti-convulsant drugs.
- Options for anti-convulsant medications
  a. Levetiracetam (Keppra) 40 – 60 mg/Kg IV x 1
  b. Fosphenytoin 20 mg

INTRACRANIAL HEMORRHAGE

1. Abstract

Intracerebral hemorrhage is bleeding into the brain parenchyma and is the second most common type of stroke. The most common causes include high blood pressure, cerebral amyloid angiopathy, vascular malformations and therapy with anticoagulants. Depending on the location and extent of the bleeding, there are different deficits. Symptoms often include reduced vigilance, paresis and headaches.
Clinically, intracerebral hemorrhage cannot be reliably distinguished from an ischemic stroke, which is why CT imaging must be performed as soon as possible. In this, the acute bleeding presents itself as a hyperdense mass. As part of the acute therapy, the frequently increased blood pressure and any hemorrhagic diathesis must be managed. If the intracranial pressure is elevated, measures to lower the intracranial pressure may be indicated. Neurosurgical evacuation of the hematoma represents a therapeutic decision on a case-by-case basis and is used in particular in the case of lobar and cerebellar hemorrhage. Complications such as bleeding entering the ventricular system or the development of hydrocephalus contribute to the high lethality of intracerebral hemorrhage.

2. Symptoms

There are four types of ICH

- epidural hematoma
- subdural hematoma
- subarachnoid hemorrhage
- intracerebral hemorrhage

Symptoms usually appear suddenly. The signs and symptoms of ICH vary depending on the type, but they usually include:

- a sudden and severe headache
- a headache associated with a recent blow into the head
- a mild and long-lasting headache
- a headache accompanied by neck stiffness
- confusion
- drowsiness
- vomiting more than twice in 24 hours
- seizure
- coma

Clinical manifestations of intracerebral hemorrhage are determined by the size and location of the hemorrhage but may include the following:

- Hypertension, fever, or cardiac arrhythmias
- Nuchal rigidity
- Subhyaloid retinal hemorrhages
- Altered level of consciousness
- Anisocoria
- Focal neurological deficits

- Basal ganglia
  - Contralateral hemiparesis
  - Conjugated gaze deviation to the lesion side (so-called deviation conjuguée)
  - Aphasia (if the dominant hemisphere is affected)
  - Homonymous hemianopia
- Thalamus
  - Reduced vigilance
  - Contralateral sensorimotor hemisymptoms
  - Vertical gaze palsy
- Cerebellum: vomiting, ataxia, dizziness, spontaneous nystagmus, dysarthria
- Pons
  - Coma and tetraparesis
  - Contralateral hemisymptoms
  - Isolated cranial nerve deficits and/or crossed brainstem syndromes
- Lobar hemorrhage: Symptoms depend on the extent and the affected lobe
  - Focal sensory or motor deficits
  - More frequent epileptic seizures
  - Occipital: Contralateral homonymous hemianopia
- Headache
- Epileptic seizures
- Symptoms may progress as a result of hematoma expansion

3. Differential Dioagnose

- Ischemic stroke
- Subarachnoid hemorrhage
- Cerebral Aneurysms
- Cerebral Venous Thrombosis
- Head Injury
- Herpes Simplex Encephalitis
- Hydrocephalus
- Arteriovenous Malformations

4. Diagnostic and skills needed

The main goals of the diagnostic are to identify hemorrhage or ischemia as quickly as possible and to clarify the etiology of the bleeding. A head CT is the gold standard for the detection of intracranial hemorrhage.

Intracerebral Hemorrhage (ICH) Score
The ICH score is a prognostic model for predicting mortality among patients with spontaneous ICH. The points are allocated for Glasgow coma scale (GCS) score, ICH volume, the presence of intraventricular hemorrhage (IVH), age, and infratentorial origin to predict 30-day mortality, which steadily increases with increasing scores.

Components for ICH score
GCS score

- 3-4: 2 points
- 5-12: 1 point
- 13-15: 0 points

ICH volume
- ≥30 cm3: 1 point
- < 30 cm3: 0 points

IVH
- Yes: 1 point
- No: 0 points

Infratentorial origin of ICH
- Yes: 1 point
- No: 0 points

Age
- Age 80 years or older: 1 point
- Younger than 80 years: 0 points

Mortality rate based on ICH Score

ICH scores with corresponding mortality risk are as follows:
- 0 points: 0%
- 1 point: 13%
- 2 points: 26%
- 3 points: 72%
- 4 points: 97%
- 5 points: 100%
- 6 points: 100% (estimated)

5. Therapy

Intracerebral hemorrhage - monitoring and stabilization
- **Admission to the stroke or intensive care unit**
- **Continuous (intensive care) monitoring and needs-based care**
  - Airway management: At the latest, when vigilance is reduced, the airway should be secured by intubation, and mechanical ventilation must be initiated
  - Venous access: At least 2 peripheral venous catheters, if necessary, CVC system
  - Continuous monitoring: (arterial) blood pressure measurement, pulse oximetry, ECG, if necessary EEG, if necessary ICP via parenchymal probe
  - Hourly monitoring: pupillary motor function, focal deficits, vigilance, body temperature, urine output
- **Analgesia, depending on the intensity of the pain, NSARs or opioids**
- **Basic neuroprotective measures**
  - Aim for normoglycemia
  - Fever reduction with the goal of normothermia (<37.5°C)
  - If necessary, stress ulcer prophylaxis (e.g. ventilation >48 hours, sepsis, kidney failure)
  - Antihypertensive therapy
  - Early lowering of blood pressure: Target systolic pressure <140 mmHg
  - Short-acting IV Medication (good controllability)
  - Urapidil: Initially titrated administration, after reduction of blood pressure continuous maintenance dose via continuous infusion.continuation via Perfusor
  - Clonidine
  - Esmolol: Loading dose and then a maintenance dose

  - No administration of nitrates (Nitrates can contribute to an increase in intracranial pressure by increasing cerebral blood volume)
- **Normalization of coagulation**
  - Stop anticoagulation or platelet aggregation inhibitors
  - Coagulation normalization in the case of an existing coagulation disorder or previous therapy with anticoagulants

  antagonism
    - Heparin: protamine
    - Vitamin K antagonists and increased INR: Vitamin K in combination with coagulation factors to achieve short-term coagulation normalization
    - Dabigatran: Idarucizumab
    - Apixaban and Rivaroxaban: andexanet alfa
    - Non-specific: coagulation factor concentrate PPSB or FFP
    - Special feature: No procoagulant substances (e.g. rFVIIa) in spontaneous ICH

Intracranial pressure therapy

- Factors for increased intracranial pressure
  - Hematoma
  - Perifocal edema
  - Possibly hydrocephalus
  - Possibly secondary ischaemia
- Indication: Intracranial pressure (ICP) >20 mmHg
- Measures
  - Elevated upper body by 30°
  - Sufficient analgesic sedation
  - Osmotic intracranial pressure therapy
    - Glycerin
    - Mannitol
    - Control: serum osmolarity 2×/day (target value: 300–320 mOsm)
  - External ventricular drainage (EVD) for hydrocephalus
    - Target pressure 15-20mmHg
    - Drainage of liquor
    - Measurement of intracranial pressure via an integrated pressure transducer
- Hyperventilation → pCO2↓ → Cerebral vessels contract → Cerebral Blood Pressure (CBP)↓ and Cerebral Blood Volume (CBV)↓ → ICP↓
- Inadequately studied in intracerebral hemorrhage, only temporarily possible with target pCO2 30–35 mmHg
- Hypothermia
- Hematoma evacuation
- Not recommended: Dexamethasone

Surgical therapy (hematoma evacuation)

- Always a case-by-case decision
- Factors in the decision-making process: location and extent of bleeding, ventricular intrusion, vigilance, age, comorbidities
- Cerebellar hemorrhages with the risk of space-occupying effects (compression/displacement of IV ventricle, deterioration in vigilance) need immediate surgery
- Surgical therapy is not indicated in:
  Thalamic and brainstem hemorrhages
  Extensive bleeding with poor prognosis

EPILEPSY

1. Abstract

A seizure is an episode of neurologic dysfunction resulting in change in motor activity or sensory perception caused by abnormal firing of neurons. Seizures can be generalized, with abnormal activity in both hemispheres of the brain and a change in mental status, or partial, involving only certain parts of the brain.
Simple partial seizures cause no change in mental status, while complex partial seizures do result in some degree of altered consciousness. Status epilepticus is defined as 5 minutes of continuous seizing or two or more episodes of seizures without a return to baseline between episodes.

2. Symptoms

Type of epilepsy
Epilepsy can manifest itself in several different types of seizures.
(Use of the following categories assumes that epilepsy has been diagnosed according to the 2014 ILAE Definition)

- Focal epilepsy
- Generalized Epilepsy
- Combined generalized and focal epilepsy
- Unclassified/ Unknown

Generally, a seizure should be considered an emergency in the following situations:

- Seizures that do not stop within a few minutes
- Prolonged confusion remains after the seizure (more than 10-15 minutes)
- The person is not responsive after a seizure
- The person has trouble breathing
- The person is injured during the seizure
- The seizure is a first-time seizure
- There is a significant change in the type or character of the seizure from that person's usual seizure pattern

The Sheldon questionnaire.

The patient has seizures if point score ≥1, and syncope if score is <1.

Questions shown to distinguish seizures from syncope	**Points (if yes)**
At times do you wake with a cut tongue after your spells?	2
At times do you have a sense of “déjà vu” or “jamais vu” before your spells?	1
At times is emotional stress associated with losing consciousness?	1
Has anyone noted your head turning during a spell?	1

Has anyone ever noted that you are unresponsive, have unusual posturing or have jerking limbs during your spells or have no memory of your spells afterwards? (Score as yes for any positive response)	1
Has anyone ever noted that you are confused after a spell?	1
Have you ever had lightheaded spells?	-2
At times do you sweat before your spells?	-2
Is prolonged sitting or standing associated with your spells?	-2

Parameters for acute symptomatic seizures proposed by the International League against Epilepsy

Provoking insult	**Time-frame**
Stroke/hypoxia	<1 week
Traumatic brain injury without subdural haematoma	<1 week
Traumatic brain injury with subdural haematoma	Up to 1 month
Intracranial surgery	<1 week
Arteriovenous malformation at time of haemorrhage	<1 week
CNS infection	Until laboratory and clinical signs of infection have resolved
Multiple sclerosis	<1 week (of relapse)
Alcohol withdrawal	7–48 hours from last alcoholic drink
Serum glucose (within 24 hours)	<36 mg/dL (2 mM) or >450 mg/dL (25 mM) and ketoacidosis
Serum sodium (within 24 hours)	<115 mg/dL (<5 mM)
Serum calcium (within 24 hours)	<5 mg/dL (<1.2 mM)
Serum magnesium (within 24 hours)	<0.8 mg/dL (<0.3 mM)
Urea nitrogen (within 24 hours)	>100 mg/dL (>35.7 mM)
Creatinine (within 24 hours)	>10 mg/dL (>884 uM)

3. Differential Diagnose

The most common differential diagnoses of epileptic seizures with loss of consciousness, which must be distinguished in each acute case, are syncope and psychogenic non-epileptic seizures. These three phenomena differ significantly in terms of prognosis and management. Differential diagnoses that are much rarer and can often only be distinguished with great effort are, for example, sleep attacks in the context of narcolepsy, REM sleep behavior disorders or non-REM parasomnias (in the case of sleep-related seizures).

4. Diagnostic and skill needed

- Routine blood tests to exclude infection or metabolic disturbance
- Brain CT/ Brain MRI
- EEG

5. Therapy and skills needed

Emergency treatment for convulsive status epilepticus

Generalized status epilepticus is currently defined as either:

- Ongoing convulsive seizure > 5 minutes
- Recurrent seizures without normalization of consciousness between seizures.

The duration of status epilepticus which may cause permanent brain damage is unknown, with experts currently suggesting thirty minutes. Persistent status epilepticus may cause aspiration, hyperkalemia, rhabdomyolysis, hyperthermia, myocardial infarction, and arrhythmia.

Initial ED management of status epilepticus

- Stabilise patient, call for help
- ABCDE
- Monitor vital signs and institute cardiac monitoring
- Establish IV access and take venous blood samples for glucose, Ca2+; FBC (= full blood count); LFT = (liver function tests); Mg2+ = magnesium; U+E = urea and electrolytes
- toxicology screening and antiepileptic drug levels
- IV Lorazepam 4mg (repeat once in 4 mins prn) or IM Midazolam 10mg
- If no response to first dose of IV benzodiazepine, start phenytoin/fosphenytoin (avoid in tox), valproate or levetiracetam
- Prepare to intubate via RSI with propofol or “ketofol” and rocuronium (if sugammadex is available or seizure >20-25 mins) or succinylcholine
- Consider immediate life-threats that require immediate treatment with specific antidotes:
  - Vital sign extremes: hypoxemia (O2), severe hyperthermia (cooling)
  - Metabolic: hypoglycemia (glucose), hyponatremia (hypertonic saline), hypomagnesemia (Mg2+), hypocalcemia (Ca2+)
  - Toxicologic: anticholinergics (HCO3), isoniazid (pyridoxine), lipophilic drug overdose (lipid emulsion) etc.
  - Eclampsia: typically > 20 weeks of pregnancy and up to 8 weeks postpartum (IV MgSO4 4-6 g over 15-20 min, then infusion 1-2 g/h)
- Brain CT to rule out space occupying lesion/ICH

First line treatment in adult status epilepticus: Benzodiazepines

1. **Lorazepam IV:** 0.1 mg /kg IV, max. 4mg, may repeat once in 4 minutes
2. **Midazolam IV :** 0,2 mg/kg, max 10mg

If neither of these 2 options are available, choose one of the following:

1. **Diazepam IV:** 0.15 mg/kg, max 10mg
2. **Diazepam PR:** 0.2 – 0.5 mg/kg, max 20 mg, single dose
3. **Phenobarbital IV:** 15 mg/kg, single dose
4. **Midazolam IM** (0.2mg/kg, max 10mg) or **buccal** (0.3mg/kg, max 10mg) (Level B evidence)

In patients without established IV access, IM midazolam is preferred. However, the most important determinant of benzodiazepine efficacy in terminating seizures is ***time to administration*** rather than choice of benzodiazepine or the choice of route. The longer a patient seizes, the more refractory to medications they become.

UNDERDOSING OF THE BENZODIAZEPINE USED IS ONE OF THE MOST COMMON REASONS WHY STATUS EPILEPTICUS CANNOT BE INTERRUPTED IN TIME! THEREFORE, CARE SHOULD BE TAKEN TO ENSURE A SUFFICIENTLY HIGH DOSAGE.

Second line treatment for status epilepticus

If benzodiazepines fail and the patient is still seizing, start second line medications. Status epilepticus can progress into non-convulsive status epilepticus and it can be difficult to diagnose without EEG monitoring. In the ED, observe for a progressive return to baseline within 60 minutes. If observed seizing cases but there is no return to near-baseline mental status within 60 minutes, there should be concern for non-convulsive status epilepticus. For patients requiring ongoing infusions of sedating medication or are have received a paralytic, non-convulsive status can only be ruled out by EEG. Bottom line is if there are ongoing subtle motor movements or no progression towards baseline mental status, err on the side of caution and continue to treat for status epilepticus until EEG monitoring is available.

Choose one of the following equivalent second line options as a ***single dose***:

- Levetiracetam 60 mg/kg IV, max 4500mg
- Fosphenytoin or Phenytoin 20 mg/kg IV, max 1500mg
  - avoid in toxicologic causes of seizure
- Valproate 40 mg/kg IV, max 3000mg
  - contraindicated in pregnancy

Refractory status epilepticus

If the patient continues to seize after first and second line treatment, they are in refractory status epilepticus. Therapeutic options include midazolam infusion, ketamine or another second line anti-epileptic medication not already used.

Medication options in refractory status epilepticus:

- **Propofol** 2-5 mg/kg IV, then infusion of 50-80 mcg/kg/min (3-5 mg/kg/hr)
- **Midazolam** 0.2 mg/kg IV, then infusion of 0.05-2mg/kg/hr
- **Ketamine** 0.5-3 mg/kg IV, then infusion of 0.3-4mg/kg/hr
- **Lacosamide** 400 mg IV over 15min, then maintenance of 200mg q12h PO/IV
- **Phenobarbital** 15-20mg/kg IV at 50-75mg/min
- Consider consulting anesthesia for **inhaled anesthetics**

The longer convulsive SE continues, the less convulsive it appears clinically, and continuous EEG monitoring should be instituted as soon as feasible.

PSYCHIATRIC EMERGENCIES

1. Abstract

Diagnosis and therapy of psychiatric emergencies and crises are part of the indispensable basic knowledge because of their frequency and importance. The terms “emergency” and “crisis” cannot be clearly separated. A psychiatric emergency is a medical condition that requires immediate action. It requires immediate, targeted therapy based on the acute symptoms in order to avert danger to the health of the patient and possibly other people. The most important psychiatric emergencies are: acute suicidal tendencies, acute states of restlessness and excitement, anxiety disorders, disturbances of consciousness, delirious syndromes/ confusional states, stupor and catatonia, intoxications and other substance-related disorders. The psychiatric crisis is characterized less by direct vital threats than by the absence or the collapse of individual and/or social coping strategies in the context of stressful illness or environmental conditions. Psychiatric crisis intervention strives to achieve a causal change in the underlying conditions in several steps within days or weeks. This requires non-medical professional help to a greater extent than in a psychiatric emergency and in contrast to a “normal crisis”.

2. Symptoms

- Clouding of consciousness
- Confusional states
- States of arousal
- States of intoxication
- Delirious syndrome
- Suicidality
- Risk of extended suicide
- Threat or acute foreign aggression

Absolute emergency indication	**Relative emergency indication/no urgent emergency medical indication**
• Successful suicide attempt • specific suicide plans or preparations • Highly aroused state • Aggressiveness/violence in the context of mental illness • specific intentions to kill someone else in the context of mental illness • severe intoxication • delirium	• Confusion • Withdrawal syndrome without delirium • Suicidal thoughts without concrete plans • Fear and panic • acute stress reaction

3. Differential Diagnosis

The essential diagnostic measure is to consider a psychiatric emergency in the first place and to recognize it reliably. Psychiatric emergencies are rarely a purely medical problem. As a rule, additional personality factors of

the person concerned, social or environmental factors and legal aspects play an important role. In addition, neurological and internal diseases as well as adverse drug reactions must always be considered in the diagnosis.

4. Diagnostic

In the acute situation, in addition to the collection of a differentiated psychopathological finding, a physical examination and often the use of apparatus-based examination procedures (laboratory examinations, EEG, ECG, imaging procedures such as cranial computed tomography [CCT] or magnetic resonance imaging [MRT]) are required.

Neurological examination
The neurological examination should be carried out as an orienting neurological emergency examination, with focus on:

- Evidence of a new focal neurological deficit?
- Signs of a meningeal syndrome?
- Evidence of a previous epileptic seizure?
- Signs of head trauma
- Assessment of qualitative awareness
- Orientation test: in terms of time, location, one's own person, the situation
- Assesment of language disorder
- Psychomotor disorders
- Cognitive disorders and thought disorders, especially optical, acoustic or tactile hallucinations, e.g.
  - Patient seems to see imaginary things, grabs at them or follows them with their eyes
  - Patient verbalizes the misperception
  - Content thought disorders: patient verbalizes delusions (e.g. paranoia)
  - Formal thinking disorders: distracted thinking, flight of ideas
- Cerebrospinal fluid diagnostics: To rule out an acute inflammatory cause
- cMRI: To rule out a structural cause in the CNS that was not detected on cCT
- EEG: To rule out non-convulsive status epilepticus
- Depending on the working diagnosis
  - Expand laboratory analysis
  - cognitive tests
  - Consult psychiatric expertise

Internal examination
Focus on indications of an acute internal illness that could explain the confusion

- Infections?
- Metabolic derailment?
- Dehydration?
- Acute abdomen?
- Blood diagnostic
  - Venous BGA (including blood glucose measurement and lactate)
  - Venous blood sampling: CBC, CRP, electrolytes (Na+, K+, Ca2+), creatinine, urea, GFR, ALT, AST, γ-GT, CK, TSH, INR, ethanol, troponin
  - Preserve reserve samples for toxicology/drug monitoring
  - In case of a fever blood culture sets from different puncture sites
- Urine diagnostics

    - Urine dipstick test
        - Urine culture
        - Reserve a reserve sample for toxicology/drug monitoring
- 12-lead ECG

5. Therapy

Psychiatric emergency and crisis situations can rarely be solved by emergency measures alone, and as a rule specific follow-up treatment must follow. Further treatment can be carried out by medical or non-medical, outpatient, semi-inpatient or full inpatient facilities.

Basic management:

- Protection of the vital functions according to the cABCDE scheme
- Vital parameters monitoring: heart rate, ECG, respiration, SpO2, blood pressure, body temperature
- Intravenous access: Min. a stable access (2 would be better)

Advanced management

- psychiatric consultation

Pharmacotherapy

The pharmacotherapy of psychiatric emergencies is mainly related to the dominant symptoms. In many cases, the initial focus is on psychopharmacological therapy, the targeted use of which is intended to create the conditions for further clarification of the situation. In emergency psychiatry, preference should be given to drugs that meet the following requirements:

- antipsychotics (high and low potency)
- antidepressants
- benzodiazepine derivative
- anticonvulsant