Stroke

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Brain arteries: lateral and medial aspects
Brain arteries - anterior and posterior circulation
Ant. Communicating A.
Recurrence branch (Heubner) (Ant. Cerebral)
Internal Carotid A.
Ant. Cerebral A.
Middle Cerebral A.
Medial and lateral striate (lenticulostriate) arteries
Ant. Choroidal A.
Post. Communicating A.
Post. Cerebral A.
Sup. Cerebellar A.
Basilar A.
Int. Auditory A.
Ant. Sup. Cerebellar A.
Vertebral A.
Ant. Spinal A.
Post. Inf. Cerebellar A.
Post. Spinal A.
Carotic and vertebral arteries

Extracranial Circulation (Left Carotid and Left Vertebral Arteries)

- Posterior communicating artery (circle of Willis)
- Basilar artery
- Vertebral artery
- Left internal carotid artery
- Left external carotid artery
- Left common carotid artery
- Left subclavian artery
- Aortic arch
Cerebral blood supply

- a. cerebri anterior
- a. communicans anterior
- a. cerebri media
- a. carotis interna
- a. communicans posterior
- a. cerebri posterior
- a. cerebelli superior
- a. basilaris
- aa. pontinae
- a. cerebelli anterior inferior
- a. vertebralis
Anatomy – Stroke.

- **Motor Cortex** (Movement)
- **Sensory Cortex** (Pain, heat, and other sensations)
- **Central Sulcus**
- **Frontal Lobe** (Judgement, foresight, and voluntary movement)
- **Parietal Lobe** (Comprehension of language)
- **Temporal Lobe** (Hearing)
- **Occipital Lobe** (Primary visual area)
- **Wernicke's area** (Speech comprehension)
- **Broca's Area** (Speech)
- **Frontal Lobe** (Smell)
- **Temporal Lobe** (Intellectual and emotional functions)
- **Brainstem** (Swallowing, breathing, heartbeat, wakefulness center and other involuntary functions)
- **Cerebellum** (Coordination)
Definition Of Stroke

• “Rapidly developed clinical sign of focal disturbance of cerebral function of presumed vascular origin and of more than 24 hours”

  WHO

• TIA (Transient Ischaemic Attack) recovery is complete within 24 hours. 10% of patients will go on to have a stroke.
TIA

• mini-stroke
• short-lived episode (less than 24 hours) of neurological dysfunction caused by a loss of blood supply
• some develop slowly, others rapidly
TIA vs stroke

- all TIAs resolve within 24 hours
- strokes take longer to resolve
- with strokes, complete function may never return → more permanent and serious problem
- TIAs can occur once, multiple times, or precede a permanent stroke
Stroke and mini-stroke

Transient ischemic attacks — TIA, or mini-strokes — result when a cerebral artery is temporarily blocked, decreasing blood flow to the brain. Many strokes result from a complete blockage of a cerebral artery, leading to death of brain cells and permanent loss of certain functions.

**TIA**
Artery temporarily blocked

**Stroke**
Artery completely blocked

[Diagrams showing TIA and Stroke with labeled parts: Site of blockage, Temporarily reduced function, Area of brain cell death, Carotid artery.]
• although most TIAs last only a few minutes, all TIAs should be evaluated with the same urgency as a stroke in an effort to prevent recurrences and/or strokes

• a TIA should be considered an emergency because there is no guarantee that the situation will resolve and function will return
TIA can cause:

- temporary visual loss
- problems with movement or sensation on one side of the body
- paralysis of the arm, leg, and face, all on one side
- double vision, dizziness
- loss of speech, understanding and balance
Symptoms

- location and extension
- hemiplegia or muscle weakness
- numbness
- reduction of sensation

Prognosis: disability
emotional problems

Prevention better than cure!
Symptoms

- Sudden numbness or weakness of face, arm or leg, especially on one side of the body
- Sudden severe headache with no known cause
- Sudden trouble seeing in one or both eyes
- Sudden confusion, trouble speaking or understanding
- Sudden trouble walking, dizziness, loss of balance or coordination
Stroke Assessment

• Motor function
• Muscle tone (high/low)
• Sensation/Proprioception/Co-ordination
• Alignment/Stability in various positions
• Neuromuscular anatomy
• Compensation Strategies
• Balance
• Mobility
Adverse Prognostic Indicators

- Prior Stroke
- Older Age
- Persistent urinal and faecal incontinence
- Visuo-spatial deficits
- Additional Influences
  - Consciousness at onset, severity of paralysis, sitting balance, admission ADL score, level of social support, metabolic rate of glucose outside the infarct area in hypertensive patient.
Types of Stroke

- **Thrombosis**: Clot in intracranial carotid artery extends directly into middle cerebral artery.
- **Embolism**: Clot fragment carried from heart or more proximal artery.
- **Hypoxia**: Hypotension and poor perfusion cause border zone infarcts, no vascular occlusion.

**Subarachnoid hemorrhage** (ruptured aneurysm)

**Intracerebral hemorrhage** (hypertension)
ISCHEMIC STROKE
Definitions of cerebral ischemia

It is the potentially reversible altered state of brain physiology and biochemistry that occurs when substrate delivery is cut off or substantially reduced by vascular stenosis or occlusion.

Stroke is defined as an „acute neurologic dysfunction of vascular origin with sudden (within seconds) or at least rapid (within hours) occurrence of symptoms and signs corresponding to the involvement of focal areas in the brain“ (Goldstein, Barnet et al, 1989)
Pathophysiology

- depletion of oxygen and glucose
- failure of energy-dependent processes
- major cause of neuronal injury: GLUTAMATE
- influx of calcium
- failure of mitochondria
A. Etiopathogenesis of cerebral ischemia

Main pathogenetic mechanisms:

1. microembolisation to brain vessels (due to myocardial infarction, mitral valve damage, others)

2. stenosis of cerebral artery + decreasing of systemic blood pressure

3. tromboembolism of large brain vessels

4. decreased cardiac output (due to decreased myocardial contractility, massive hemorrhage, others)
B. Pathogenetic mechanisms involved in development of cerebral ischemia (CI)

1. The brain is protected against focal interruption of blood supply by a number of extra- and intracranial collateral vessels.

Actual size of the cerebral ischemia depends on:

a) number and vascular tone of the leptomeningeal collateral channels

b) blood viscosity

c) blood perfusion pressure
• The rich anastomotic connections between the carotid and vertebral arteries provide a powerful collateral system which is able to compensate for the occlusion of up to three of these arteries (known from animal experiment)

• A good collateral system results in lesser ischemic area than is a territory supplied by occluded artery

• A bad collateral system results in ischemic area equal to a territory supplied by occluded artery
Mechanisms involved in failure of collateral system

- ↓ systemic BP → ↓ blood flow through collateral circulation → base for hemodynamic theory of stroke development

- ↓ systemic BP + multifocal narrowing of extracerebral arteries → ↓ blood flow initially in the periphery of arterial territories

- since these regions represent the border lines between the supplying territories of the main cerebral arteries, the resulting lesion have been termed "border zone" or watershed infarcts
Ischemic cascade

Lack of oxygen supply to ischemic neurones

\[ \downarrow \]

ATP depletion

\[ \downarrow \]

Membrane ions system stops functioning

\[ \downarrow \]

Depolarisation of neurone

\[ \downarrow \]

Influx of calcium

\[ \downarrow \]

Release of neurotransmitters, including glutamate, activation of N-methyl-D-aspartate and other excitatory receptors at the membrane of neurones

\[ \downarrow \]

Further depolarisation of cells

\[ \downarrow \]

Further calcium influx

Carrol and Chataway, 2006
Cosequences of brain ischemia

Energy failure / depolarisation

Transmitter release and receptor activation

Lipolysis (DAG \(\uparrow\rightarrow\) PKC \(\uparrow\)) →

Protein phosphorylation

Proteolysis

Disaggregation of microtubuli

Enzyme conversion

Breakdown of cytoskeleton

Inhibition of axonal transport, blebbing

Damage to membrane structure and function

Dysfunction of receptors and ion channels

Free radical formation

FFAs \(\uparrow\), LPLs \(\uparrow\)

Ca\(^{2+}\)
2. Hemorheology and microcirculation - their importance in development CI

Relationship between blood viscosity and microcirculation:

\[ \dot{Q} = \frac{\Delta P \cdot r^4}{\eta \cdot 8 \cdot l} \]

- \( \dot{Q} \) = flow rate
- \( \Delta P \) = pressure gradient
- \( r \) = radius of tube
- \( l \) = length of the tube
- \( \eta \) = viscosity of the fluid
• It is clear that flow rate (Q) indirectly depends on blood viscosity – Q will decrease with increase blood viscosity.

Blood viscosity depends on:
- hematocrit,
- erythrocyte deformibility,
- flow velocity,
- diameter of the blood vessels

In the brain macrocirculation (in vessels larger than 100 μ): Blood viscosity depends mainly on:
- hematocrit,
- flow velocity

blood viscosity ↑: by decreasing flow velocity by increasing hematocrit
This is important at low flow velocity, mainly

Why?

- Er aggregation (reversible)
- platelet aggregation (irreversible)

In the brain **microcirculation** (vascular bed distal to the of 30 - 70μm diameters, arterioles into the brain parenchyma)

**blood viscosity** changes with **changes of vessels diameter**, mainly
• Initially, as diameter of vessels falls, the blood viscosity falls, too. When vessels diameter is reduced to less than 5-7 μm, viscosity again increases (inversion phenomenon)

Summary:
Disturbancies of brain microcirculation accompanied by hemorheologic changes at low blood flow velocity are considered as important pathogenic factor promoting development of cerebral ischemia and cerebral infarction
3. No - reflow phenomenon

**Definition:** Impaired microcirculatory filling after temporary occlusion of cerebral artery

**Result:** This mechanism can contribute to development of irreversibility of cell damage in ischemic region

**Summary:** It can be disputed if no-reflow after transient focal ischemia at normal blood pressure is of pathogenic significance for infarct development or merely accompaniesment of irreversible tissue injury
4. Changes in cerebral blood flow regulation

• cerebral ischemia $\rightarrow$ both CO$_2$ reactivity and autoregulation of cerebral vessels are disturbed

In the center of ischemic territory:

a) CO$_2$ reactivity $-$ abolished or even reversed (i.e. blood flow may decrease with increasing PaCO$_2$)

b) disturbance of autoregulation
   $-$ mainly when BP is decreased local blood perfusion pressure is below the lower limit of the autoregulatory capacity of the cerebrovascular bed $\rightarrow$ vessels are maximally dilated
• Disturbances of flow regulation after stroke are longlasting:
  
  - for autoregulation up to 30 days,
  - for CO₂ reactivity up to 12 days.

• These disturbances contribute to the phenomenon of post – ischemic hypoperfusion which is important pathophysiological mechanism for the development of secondary neuronal injury after global cerebral ischemia.

• Disturbances of flow regulation → luxury perfusion

  luxury perfusion = oxygen supply to tissue exceeds the oxygen requirements of the tissue
Possible mechanism involved:
- vasoparalysis brought about by the release of acidic metabolites from the ischemic tissue

Forms of luxury perfusion:
  a) absolute (true hyperemia)

  b) relative (depending on the level of $O_2$ consumption)
5. Segmental vascular resistance - its importance for development CI

Two different types of brain vessels have to be distinguished:

a) extracerebral (conducting and superficial) vessels
   - extracerebral segment of the vascular bad (a.carotis, a.basilaris,... and leptomeningeal anastomoses)

b) nutrient (penetrating) vessels
   - intracerebral segment of brain circulation (vessels penetrating to brain tissue and capillary network supplied by them)
Both of segments are involved in autoregulation of blood flow through brain, but intracerebral segment react to CO$_2$, only

Middle cerebral artery constriction $\rightarrow$↑ resistance of extracerebral conducting vessels $\rightarrow$↓pial arterial BP$\rightarrow$
$\rightarrow$ autoregulatory dilation of intracerebral vascular segment
6. Intracerebral steal phenomena (syndrome)

- The interconnection of ischemic and non-ischemic vascular territories by anastomotic channels may divert blood from one region to the other, depending on the magnitude and the direction of BP gradient across the anastomotic connections.

- The associated change of regional blood flow is called "steal," if it results in a decrease of flow, or "inverse steal" if it results in an increase of flow (Robin Hood syndrome) in ischemic territories.

Mechanism in steal phenomena occurrence:

- Vasodilation in non-ischemic brain regions (\( \uparrow \text{pCO}_2 \), anesthesia) \( \rightarrow \downarrow \) BP in pial arterial network \( \rightarrow \downarrow \) of the collateral blood supply to the ischemic territory.
Mechanism of inverse steal phenomena:

- vasoconstriction (↓ pCO2) in the intact brain regions (or indirectly - to a decrease of intracranial pressure causing an improvement of blood perfusion) → ↑ of blood flow in ischemic brain region

Summary:

Despite of existing knowledge about steal and inverse steal phenomena, it is not possible to predict alterations of degree and extent of ischemia when blood flow in the non-ischemic territories is manipulated. Such manipulations are not recommended up to now for the treatment of stroke.
7. Thresholds of ischemic injury

In the intact brain metabolic rate can be considered as the sum of:

a) activation metabolism - supports the spontaneous electrical activity
   (synaptic transmission, generation of action potentials)

b) basal (residual) metabolism - supports the vital functions of the cell (ion homeostasis, osmoregulation, transport mechanisms, production of structural molecules)
The working brain consumes about:

1/3 of its energy for maintenance of synaptic transmission
1/3 for transport of Na\(^+\) and K\(^+\)
1/3 for preserving of structural integrity

Gradual ↓ of oxygen delivery →

→ a) reversible disturbances of coordinating and electrophysiological functions
   
b) irreversible structural damage occurs

Ischemic thresholds for functional and structural damage of brain due to ischemia are showed in scheme (Fig. 1)
FIG. 2. Ischaemic thresholds for electrical failure and release of cellular K⁺. In this figure, the term “ischaemia” is restricted to a reduction of local cerebral blood flow (CBF) of a degree leading to impaired tissue function, while “oligaemia” is taken to mean a reduction in blood flow that leaves functions unaltered. This diagram was taken from Astrup and co-workers (7), with permission of the authors and the American Heart Association.
Thresholds for functional disturbances:

a) the appearance of functional changes (clinical symptoms and signs) when focal blood flow rate was below 0.23 ml/g/min

b) complete hemiplegia was present when blood flow rate decline to 0.08 - 0.09 ml/g/min

c) threshold of the suppression of EEG activity begins at the flow rate 0.20ml/g/min and EEG became isoelectric when blood flow rate is between 0.15-0.16 ml/g/min

d) depolarization of cell membranes occurs at flow levels below 0.08 - 0.10 ml/g/min (sudden increase extracellular K⁺ and associated fall of extracellular Ca++ (threshold for ion pump failure - it is the lower level of the penumbra range)
Threshold for morphological injury

Development of morphological lesions requires:

a) minimal time (manifestation or maturation time)

b) certain density of ischemia

- permanent ischemia $0.17 - 0.18 \text{ ml/g/min}$ → histological changes

- 2 hours ischemia $0.12 \text{ ml/g/min}$ → histological changes

- 1 hour ischemia $0.05 - 0.06 \text{ ml/g/min}$ → histological changes
8. The concept of diascchisis

**Diaschisis** = the term for remote disturbances of brain cells due to the suppression of neurons connected to the injured (ischemic) region.

**Possible mechanism** involved in diascchisis occurrence:

- the neurons in remote focus of brain from ischemic injury suffer a kind of shock when they are deprived from some of their afferent input coming from ischemic focus.
• it is reasonable to assume that deactivation of nerve fiber system connecting the areas involved causes a depression of functional activity because decrease of blood flow and metabolic rate are coupled.

• a possible molecular mediator of diaschisis is a disturbed neurotransmitter metabolism.

Time characteristic of diaschisis development

• diaschisis appears within 30 min after the onset of ischemia.

• reversal of the phenomena has been observed after a few months.
C. Consequences of cerebral ischemia

Neurophysiological disturbances

a) **neurological deficit** (forced ambulation with circling, tonic deviation of the head and neck toward the side of the occluded artery... active movements cease → opposite limbs become weak, development of apathetic or akinetic state

b) **suppression of electrocortical activity**

c) **suppression of cortical evoked potentials**
Clinical Signs of Carotid Artery Ischemia

Ocular

Transient blindness in one eye from temporary occlusion by platelet-fibrin or cholesterol emboli (on side of involved artery)

Partial blindness may be detected by covering one eye at a time to determine if defect is monocular or binocular.
Cerebral hemisphere

Occasional headache (usually supraorbital or temporal)

Homonymous visual defects in field opposite involved artery

Language defect only when dominant hemisphere involved

Hemiparesis or hemiplegia on side opposite involved artery (only arm or leg may be affected); may be transient or permanent and may appear with or without sensory deficits

Patient may awaken from sleep unable to move affected side
Clinical Signs of Posterior Circulation Ischemia

- Abnormal eye movements, double vision, and nystagmus. Horner syndrome may be present.
- Hemianopsia (frequently bilateral).
- Motor and sensory deficits in face may be unilateral, bilateral, or alternating.
- Vertigo and ataxia; motor and sensory deficits may be unilateral, bilateral, or alternating.
- Headache and vomiting.
- Dysphagia.
- Dysphonia.
- Altered consciousness (drowsiness or stupor) may be transient or prolonged.
THROMBOTIC STROKE

• part of the brain supplied by the clotted blood vessel is deprived of blood and oxygen
• blockage of an artery in the brain by a clot (thrombosis) - most common cause of stroke
• cells of that part of the brain die
Coronal section of the brain showing middle cerebral artery

Atherosclerotic clot

Blood clot

Ischemic Stroke

Blood clot stops the flow of blood to an area of the brain

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Stroke Classification

- TACI (Total Anterior Circulation Infarct)
- PACI (Partial Anterior Circulation Infarct)
- LACI (Lacunar Infarct)
- POCI (Posterior Circulation Infarct)
RISK FACTORS:

• high blood pressure (hypertension)
• high cholesterol
• diabetes
• smoking
Embolic stroke

- blockage of an artery by an embolus

An embolus is...
- thrombus
- fat
- air
- cancer cells
- clumps of bacteria
- amniotic fluid
• embolus - arises from elsewhere, most commonly from the heart
• source must be identified
• symptoms - maximal at start
• symptoms may be transient
AF and stroke

- Stroke is the most serious ongoing risk associated with AF\(^1\)
- In patients with AF, blood clots tend to form in the atria, particularly within the left atrial appendage, as a result of abnormal blood flow and pooling\(^2,3\)
- These clots may travel to the brain, causing an ischaemic stroke\(^2\)
- Approximately 20% of ischaemic strokes are caused by blood clots that originate in the heart (cardioembolic); of these, AF is the most common cause\(^4\)

Patients with AF have an ~fivefold increased risk of ischaemic stroke.

Framingham Heart Study (N=5,070)

Risk ratio = 4.8

\( p < 0.001 \)

Among stroke survivors, AF increases the likelihood of a recurrent stroke.

**Italian population-based study**¹
Estimates of recurrent stroke in patients with and without AF ($p=0.0398$)

**Spanish retrospective cohort study**²
Estimates of recurrent stroke in non-anticoagulated patients with and without AF ($p<0.0001$)

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### CHADS\textsubscript{2} score and stroke risk in patients with AF

<table>
<thead>
<tr>
<th>Item</th>
<th>Points</th>
<th>CHADS\textsubscript{2}</th>
<th>Stroke rate (95 %CI)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestive heart failure</td>
<td>1</td>
<td>6</td>
<td>18.2 (10.5–27.4)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1</td>
<td>5</td>
<td>12.5 (8.2–17.5)</td>
</tr>
<tr>
<td>Age ≥75 years</td>
<td>1</td>
<td>4</td>
<td>8.5 (6.3–11.1)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1</td>
<td>3</td>
<td>5.9 (4.6–7.3)</td>
</tr>
<tr>
<td>Stroke/TIA</td>
<td>2</td>
<td>2</td>
<td>4.0 (3.1–5.1)</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>1</td>
<td>2.8 (2.0–3.8)</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>0</td>
<td>1.9 (1.2–3.0)</td>
</tr>
</tbody>
</table>

*Add points together

*Per 100 patient-years without antithrombotic therapy

## CHADS\textsubscript{2} and CHA\textsubscript{2}DS\textsubscript{2}-VASc

<table>
<thead>
<tr>
<th>Risk factors</th>
<th>CHADS\textsubscript{2}</th>
<th>CHA\textsubscript{2}DS\textsubscript{2}-VASc</th>
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<tbody>
<tr>
<td>Age (years)</td>
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</tr>
<tr>
<td>65–74</td>
<td>+1</td>
<td></td>
</tr>
<tr>
<td>≥75</td>
<td></td>
<td>+2</td>
</tr>
<tr>
<td>&gt;75</td>
<td></td>
<td>+1</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>+1</td>
<td>+1</td>
</tr>
<tr>
<td>Hypertension</td>
<td>+1</td>
<td>+1</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>+1</td>
<td>+1</td>
</tr>
<tr>
<td>Stroke/TIA</td>
<td>+2</td>
<td>+2</td>
</tr>
<tr>
<td>Vascular disease*</td>
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<td>+1</td>
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<tr>
<td>Female gender</td>
<td></td>
<td>+1</td>
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<tr>
<td><strong>Cumulative score</strong></td>
<td><strong>0–6</strong></td>
<td><strong>0–9</strong></td>
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</table>

*M1, peripheral artery disease or aortic plaque

Stroke prevention in AF: VKAs vs placebo

Reduction of risk of thromboembolism in AF

Study, year
AFASAK I, 1989; 1990
SPAF I, 1991
BAATAF, 1991
CAFA, 1991
SPINAF, 1992
EAFT, 1993

All trials (n=6)

Relative risk reduction (95% CI)

Favours VKA

Favours placebo

ACTIVE W: VKA is more effective than dual antiplatelet therapy

Cumulative risk of stroke

<table>
<thead>
<tr>
<th></th>
<th>Clopidogrel + ASA</th>
<th>Warfarin</th>
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<tbody>
<tr>
<td>Number at risk</td>
<td>3,335</td>
<td>3,371</td>
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<tr>
<td></td>
<td>3,168</td>
<td>3,232</td>
</tr>
<tr>
<td></td>
<td>2,419</td>
<td>2,466</td>
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<tr>
<td></td>
<td>941</td>
<td>930</td>
</tr>
</tbody>
</table>

RR=1.72 (1.24–2.37), p=0.001
VKAs have a narrow therapeutic window

Adjusted odds ratios for ischaemic stroke and intracranial bleeding in relation to intensity of anticoagulation

Data on bleeding and stroke risk support recommendation for narrow INR target range of 2.0–3.0

Risk of major bleeding increases with the HAS-BLED score ($p=0.007$)

**Clinical characteristic**

<table>
<thead>
<tr>
<th>Points</th>
<th>Hypertension (SBP &gt;160 mmHg)</th>
<th>Abnormal renal or liver function</th>
<th>Stroke</th>
<th>Bleeding</th>
<th>Labile INRs</th>
<th>Elderly (age &gt;65 years)</th>
<th>Drugs or alcohol</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td>1 + 1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1 + 1</td>
</tr>
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</table>

**Cumulative score**

Range 0–9

HAS-BLED score

<table>
<thead>
<tr>
<th>Number of patients</th>
<th>798</th>
<th>1,286</th>
<th>744</th>
<th>187</th>
<th>46</th>
<th>8</th>
<th>2</th>
<th>0</th>
<th>0</th>
<th>0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of bleeding events</td>
<td>9</td>
<td>13</td>
<td>14</td>
<td>7</td>
<td>4</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Paradoxical embolism

- atrial septal defect
Cardiac causes

- atrial fibrillation
- rheumatic disease
- artificial heart valves
- dilated cardiomyopathy
- Libman-Sacks endocarditis
- infective endocarditis
- marantic endocarditis
- left atrial myxoma
Ischemic stroke therapy

- **Possibilities:**
  - **Antithrombotic**
    - Aspirin has only small effect on acute strike, but reduces the risk of future CV death
  - **Anticoagulant**
    - Anticoagulant therapy shows no effect on stroke, however patients with AF have reduced risk of stroke after AC therapy
  - **Fibrinolytic**
    - Improves the outcome, but must be initiated not later than 3 hours after the stroke
INTRACRANIAL BLEEDING
Hemorrhagic stroke

SUBARACHNOIDAL AND INTRACEREBRAL BLEEDING
Alternative names

• brain bleeding
• brain hemorrhage
• stroke – hemorrhagic
• hemorrhagic cerebrovascular disease
Pathophysiology

- A small blood vessel inside the brain becomes weak and bursts
- Blood seeps into the brain tissue
- The flow of blood after the blood vessel ruptures directly damages brain cells
- An expanding hematoma causes compression of tissue which results in tissue injury
The three major causes of hemorrhagic stroke:
- High blood pressure (hypertension) → HYPERTENSIVE INTRACEREBRAL HEMORRHAGE
- Ruptured arterial aneurysms
- ARTERIOVENOUS MALFORMATIONS (AVMs)

Also very common in head trauma
Risk factors

- Advanced age
- Cigarette smoking (active and passive)
- Heavy alcohol consumption and drug use
- Diabetes mellitus
- Lack of physical activity, obesity and unhealthy diet
- Some apply only to women (pregnancy, childbirth, menopause)

Arteriovenous malformation
Symptoms

- Sudden start (seconds or minutes)
- Depend on the area of the brain affected
- The most common signs of a stroke are:
  - weakness down one side of the body, ranging from numbness to paralysis that can affect the arm and leg
  - weakness down one side of the face, causing the mouth to droop
  - speech may be difficult or become difficult to understand
  - swallowing may be affected
  - loss of muscle coordination or balance
  - brief loss of vision
  - severe headache
  - confusion
Diagnosis

• Medical history
• Physical examination
• Face-arm-speech test (FAST) for early recognition:
  - Facial weakness: can the person smile? Has the mouth or eye drooped?
  - Arm weakness: can the person raise both arms?
  - Speech problems: can the person speak clearly and understand you?
  - Test these symptoms

CT scan of a patient who has had a left middle cerebral artery stroke. The arrow indicates the location of the stroke.
Types of hemorrhagic stroke

• *intracerebral hemorrhage*
• *subarachnoid hemorrhage*
Arteria cerebri media and penetrating arteries

- Corpus striatum (caudate and lenticular nuclei)
- Callosomarginal arteries
- Pericallosal arteries (branches of anterior cerebral arteries)
- Trunk of corpus callosum
- Internal capsule
- Septum pellucidum
- Rostrum of corpus callosum
- Anterior cerebral arteries
- Recurrent artery (of Heubner)
- Anterior communicating artery
- Optic chiasm
- Middle cerebral artery
- Internal carotid artery
- Temporal lobe
- Lateral cerebral sulcus (Sylvian fissure)
- Insula
- Medial and lateral lenticulostriate arteries
- Falx cerebri
Microaneurysms in penetrating arteries

Microaneurysm formed in penetrating artery of brain as result of hypertension. Lenticulostriate vessels (shown) most commonly involved, but similar process may occur in other parts of brain, especially lobar white matter, thalamus, pons, or cerebellum.
Moderate-sized intracerebral hemorrhage, involving left putamen, with rupture into lateral ventricle. Brain distorted to opposite side. Scar of healed hemorrhage on right side.

CT scan shows large putaminal hemorrhage, with blood in ventricles.
Distribution of congenital cerebral aneurysms

Anterior cerebral 30%
  Distal anterior cerebral 5%
  Anterior communicating 25%

Internal carotid 30%
  Ophthalmic 4%
  Posterior communicating 18%
  Bifurcation 4%
  Anterior choroidal 4%

Middle cerebral 25%

Posterior cerebral 2%
  (Posterior communicating and distal posterior cerebral)

Basilar 10%
  Bifurcation 7%
  Basilar trunk 3%

Vertebral–posterior inferior cerebellar 3%

Anterior circulation 85%

Posterior circulation 15%
Causes

- high blood pressure - hypertension (ICH)
- trauma
- infections
- tumors
- blood-clotting deficiencies
- abnormalities in blood vessels (arteriovenous malformations)
- Aneurysm (SAH)
- accumulation of amyloid
- drugs - cocaine
Warning signs and symptoms

• sudden trouble seeing in one or both eyes
• sudden confusion, trouble speaking or understanding
• sudden trouble walking, dizziness, loss of balance or coordination
• sudden severe headache with no known cause
• sudden numbness or weakness of face, arm or leg, especially on one side of the body
Possible complications

• permanent loss of movement or sensation of a part of the body
• joint contractures
• muscle spasticity
• permanent loss of cognitive or other brain functions
• disruption of communication, decreased social interaction
• decreased ability to function or care for self
• decreased life span
• urinary and respiratory tract infections
EPIDURAL AND SUBDURAL HEMATOMA
Layers of the Meninges

- Skin of scalp
- Periosteum
- Bone of skull
- Periosteal Meningeal
- Dura mater
- Arachnoid mater
- Pia mater
- Arachnoid villus
- Blood vessel
- Falx cerebri (in longitudinal fissure only)
Epidural Hematoma

- Accumulation of blood in the potential space between dura mater and bone
- Mortality rate estimated to be 5-50%
- Skull fractures occur in 85-95%
- Extension of the hematoma limited by suture lines
- The temporoparietal region and the middle meningeal artery (66%)
Subfrontal and occipital hematoma

nerve leading to ipsilateral pupil dilatation and 3rd cranial nerve muscle palsy

Subfrontal hematoma

Frontal trauma: headache, poor cerebration, intermittent disorientation, anisocoria

Posterior fossa hematoma

Occipital trauma and/or fracture: headache, meningismus, cerebellar and cranial nerve signs, Cushing’s triad
History

• Head trauma
• *Lucid interval* between the initial loss of consciousness at the time of impact and a delayed decline in mental status (10-33% of cases)
• Headache
• Nausea/vomiting
• Seizures
• Focal neurological deficits (eg, visual field cuts, aphasia, weakness, numbness)
Diagnostic Imaging

- **Noncontrast CT scanning** of the head
- *Hyperdense biconvex or lenticular-shaped* mass situated between the brain and the skull (regions of hypodensity may be seen with serum or fresh blood)
- *MRI* also demonstrates the evolution of an epidural hematoma (not appropriate for patients in unstable condition)
Subdural Hematoma

• Rapidly clotting blood collection below the inner layer of the dura but external to the brain and arachnoid membrane

• Low-pressure venous bleeding of bridging veins (between the cortex and venous sinuses) dissects the arachnoid away from the dura and layers out along the cerebral convexity

• It conforms to the shape of the brain and the cranial vault, exhibiting concave inner margins and convex outer margins (crescent shape)

• Mortality
  – Simple SDH (no parenchymal injury) is associated with a mortality rate of about 20%
  – Complicated SDH (parenchymal injury) is associated with a mortality rate of about 50%
Anatomy of brain vessels
History

- Moderately severe to severe blunt *head trauma*
- Acute deceleration injury from a fall or motor vehicle accident, but rarely associated with skull fracture
- Generally *loss of consciousness*
- Any degree or type of coagulopathy should heighten suspicion of SDH
- Commonly seen in alcoholics because they’re prone to thrombocytopenia, prolonged bleeding times, and blunt head trauma
- Patients on anticoagulants can develop SDH with minimal trauma and warrant a lowered threshold for obtaining a head CT scan
Diagnostic Imaging

- MRI is superior for demonstrating the size of an acute SDH and its effect on the brain,
- Noncontrast head CT: the primary means of making a diagnosis and suffice for immediate management purposes
  - *Hyperdense (white) crescentic mass* along the inner table of the skull, most commonly over the cerebral convexity in the parietal region. The second most common area is above the tentorium cerebelli
- In the chronic phase, the lesion becomes hypodense and is easy to appreciate on a noncontrast head CT scan
Summary

• **Epidural Hematoma**
  – Potential space between the dura in the inner table of the skull
  – Can’t cross sutures
  – Skull fractures in temporoparietal region
  – Middle meningeal artery
  – Lenticular or biconvex shape
  – Lucid interval
  – Common in alcoholics
  – Medical emergency
  – CT without contrast
  – Evacuate via burr holes

• **Subdural Hematoma**
  – Between the dura mater and the arachnoid mater
  – Can cross sutures
  – Cortical bridging veins
  – Crescent shape
  – Loss of consciousness
  – Common in elderly
  – Common in alcoholics
  – Medical emergency
  – CT without contrast
  – Evacuate via burr holes
Intracranial bleeding

- Epidural bleeding
- Subdural bleeding
- Subarachnoidal bleeding
- Intracerebral bleeding

Arterial

Venous

Traumatic

Stroke