

Apopleksi



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Klinisk sygeplejespecialist
cand.scient.san, PhD

Case



Mens Karl spiser morgenmad får han det pludseligt mærkeligt.

Det føles som om hans højre kind er bedøvet og at det prikker i højre arm.

Da han vil samle sine briller op fra bordet har han svært ved at ramme rigtigt og få fat omkring dem med fingrene.

Da han vil forklare til sin kone, at han har det underligt, kan han ikke få de rigtige ord ud. Det er som om han godt kan huske ordet, men når han prøver at sige dem højt kan han godt høre at det er noget vrøvl der kommer ud.

Klinisk definition

Pludseligt opstået

Fokale neurologiske udfald

Vaskulær basis

Enten infarkt eller blødning

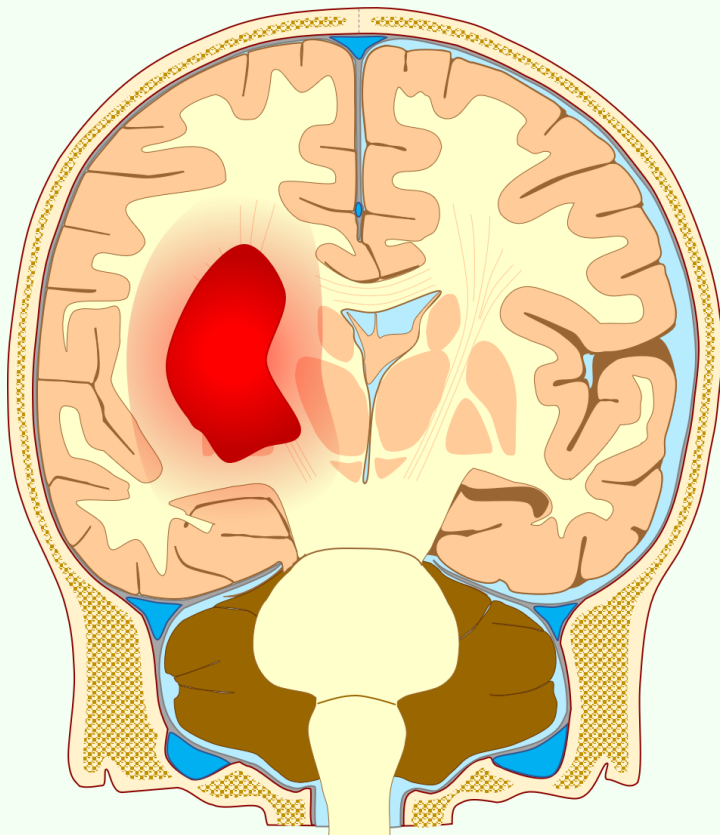
Varige symptomer (> 24 timer)

(ved varighed < 24 timer = TCI) ^{A,B}

^A Mere end halvdelen har problemer med fatigue og op imod halvdelen har problemer med hukommelse, koncentration og multi-taskning efter tre måneder (Fens *et al.* 2013)

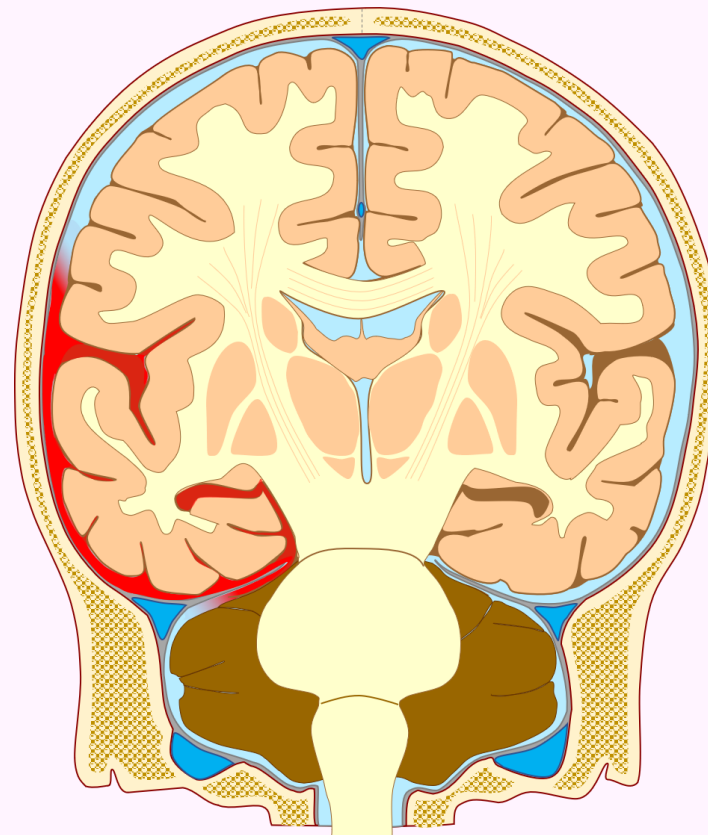
^B 30-50% af patienter med TCI har vævsforandringer på MR DWI (Easton *et al.* 2009)

Blødning inde i hjernen



ICH = Apopleksi

Blødning uden på hjernen



SAH ≠ Apopleksi

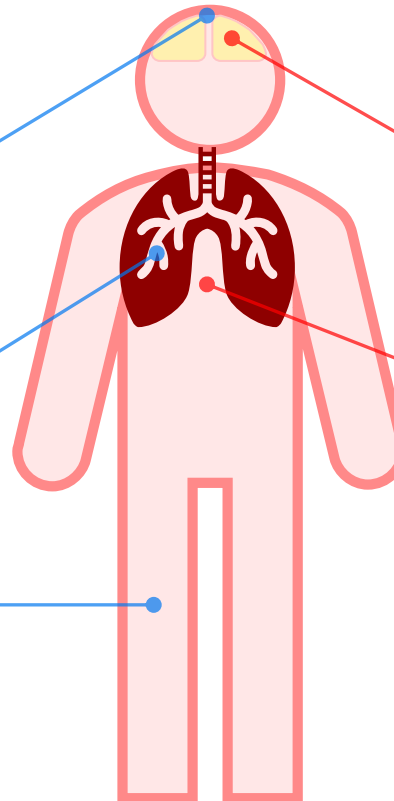
Blodpropper

Venøs

Hjernen
Sinus trombose

Lunge
Lunge emboli

Ekstremiteter
Dyb venetrombose



Arteriel

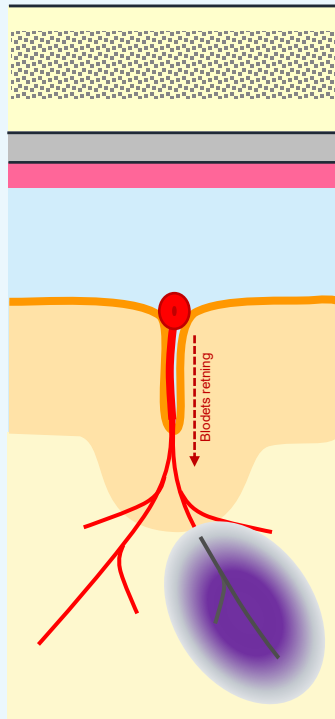
Hjernen
Apopleksi/TCI

Hjertet
Myokardie infarkt

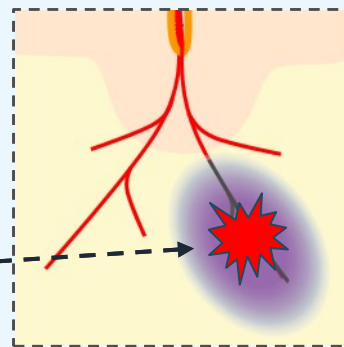
To typer apopleksi

Iskæmisk apopleksi

Skyldes blodprop
Ca. 90%
(9-10.000/år)



Ca. 15% får blødning i infarkt
(hæmorrhagisk transformation)



Intracerebral hæmorrhagi

Skyldes blødning
Ca. 10%
(1000-1500/år)

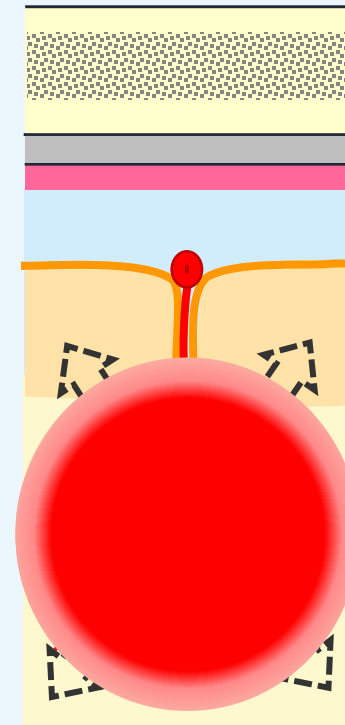


Illustration: Jacob Liljehult 2021

Iskæmisk apopleksi

Iskæmi skyldes en nedsat arteriel blodforsyning til et område af hjernen

Trombe

- Opstår lokalt i et kar i hjernen
- Hyppige årsager: Aterosklerose

Emboli

- Enten fra hjertet eller fra en af de arterier, som forsyner hjernen (aorta, carotis og vertebralis)
- Hyppige årsager: Atrieflimmer, arteriosklerotiske plaques, kardissektion

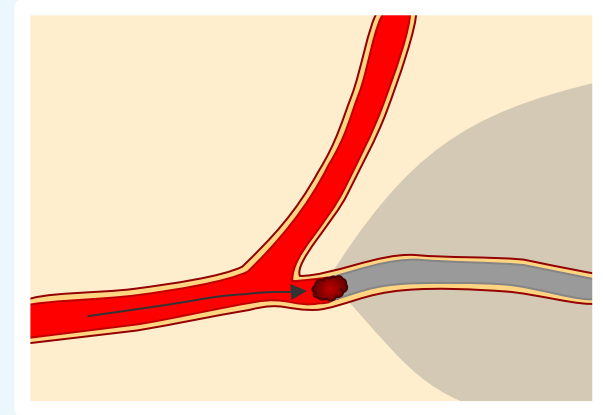
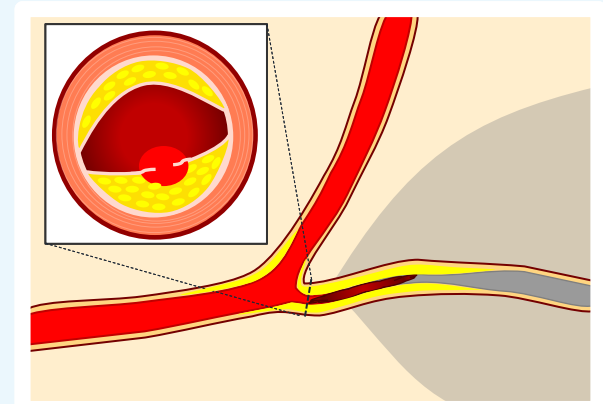
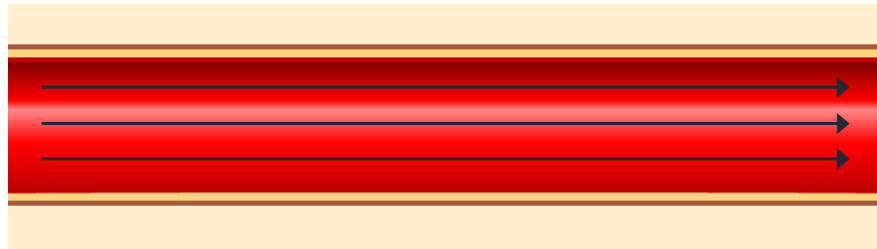
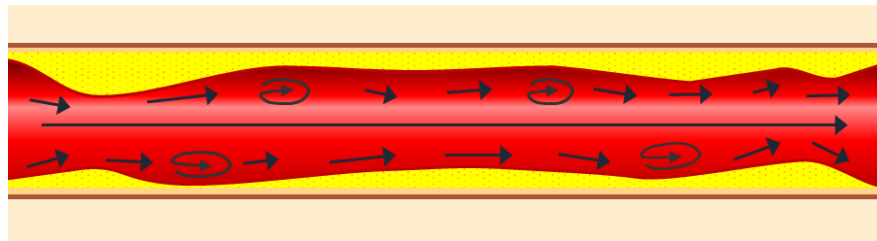


Illustration: Jacob Liljehult 2021

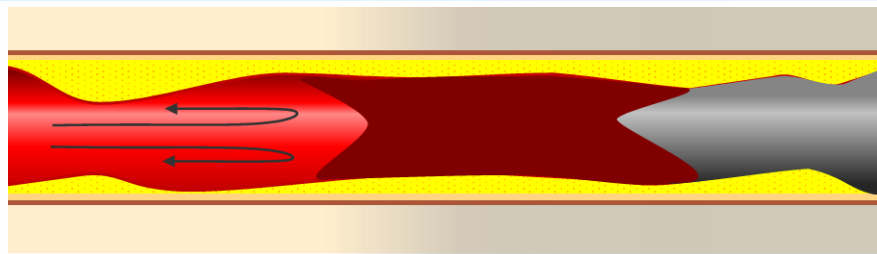
Trombose



Normal arterie
Normalt flow



Arteriosklerotisk arterie
Aflejring af fedt og kalk
Forstyrrelse af blod-flow

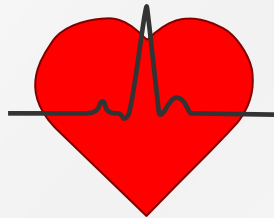


Arteriosklerotisk arterie
Dannelse af trombe

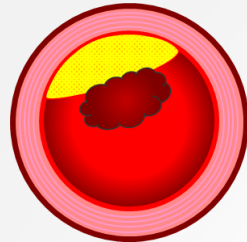
Emboli

Hyppige embolikilder

Hjertet



Arteriosklerose



Dissektion

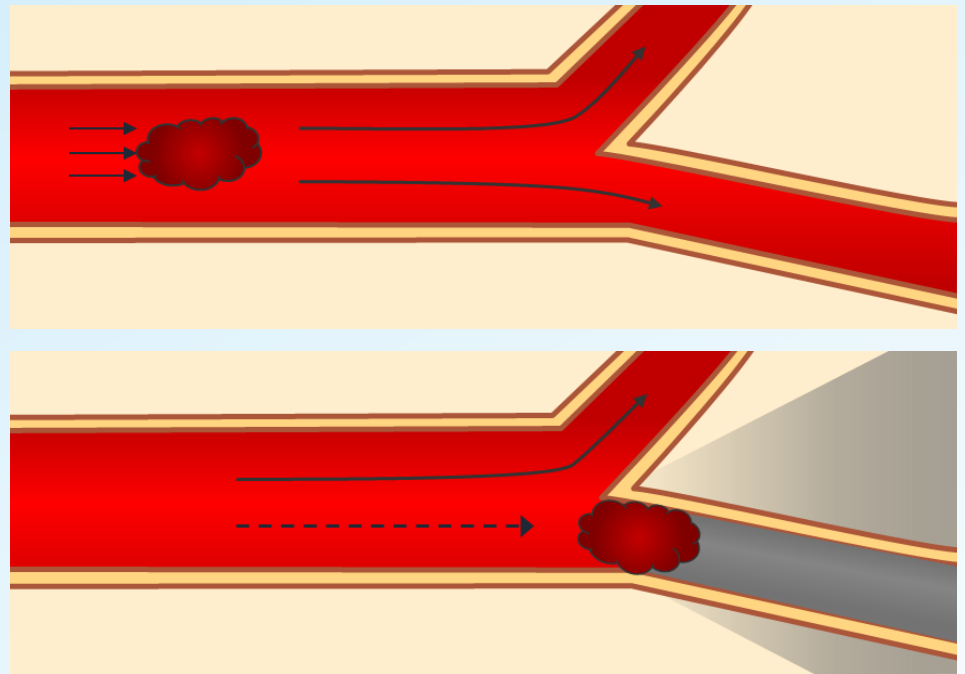
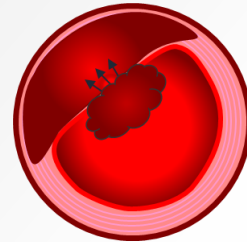


Illustration: Jacob Liljehult 2021

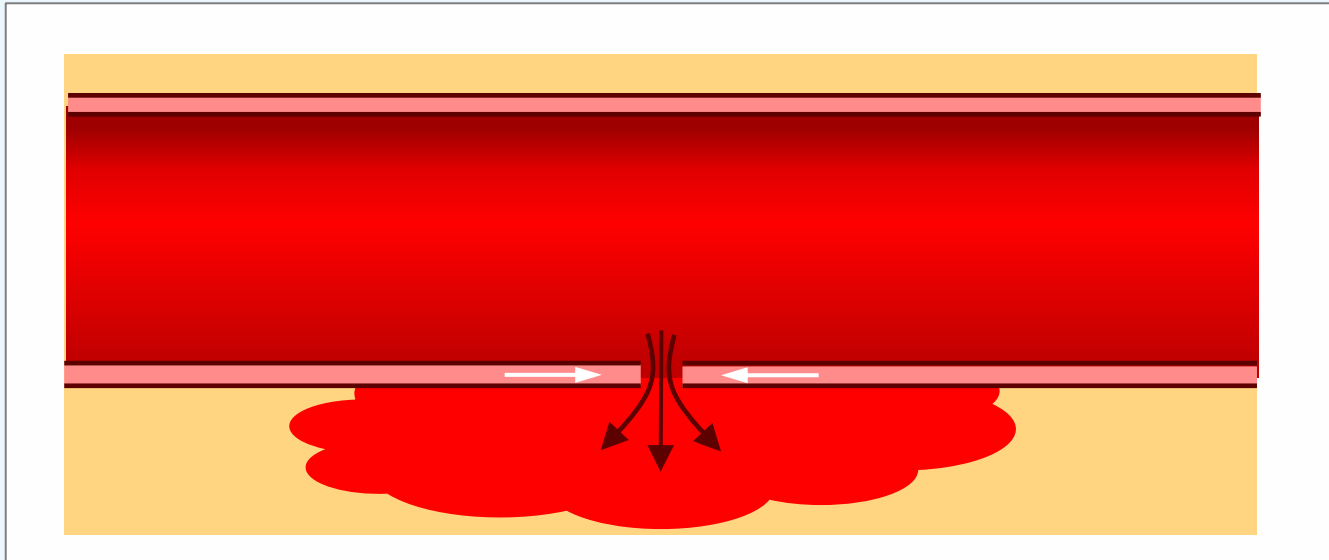
Hæmostase

Kan både aktiveres af faktorer fra *blodet* og faktorer fra *væv*

- 1. Reaktion fra karvæggen** → Karret trækker sig sammen
- 2. Dannelse af en *pladeprop***
 - Trombocytterne aktiveres → de binder sig fast til karvæggen og til hinanden og danner en *pladeprop*
- 3. Dannelse af *fibrin***
 - Koagulation af blodet → dannelse af *fibrin* fibre
- 4. Afgrænsning af blodproppen**
 - Fibrinolyse → nedbrydelse af fibrin vha. *plasmin*
 - Inaktivering af koagulationsfaktorer

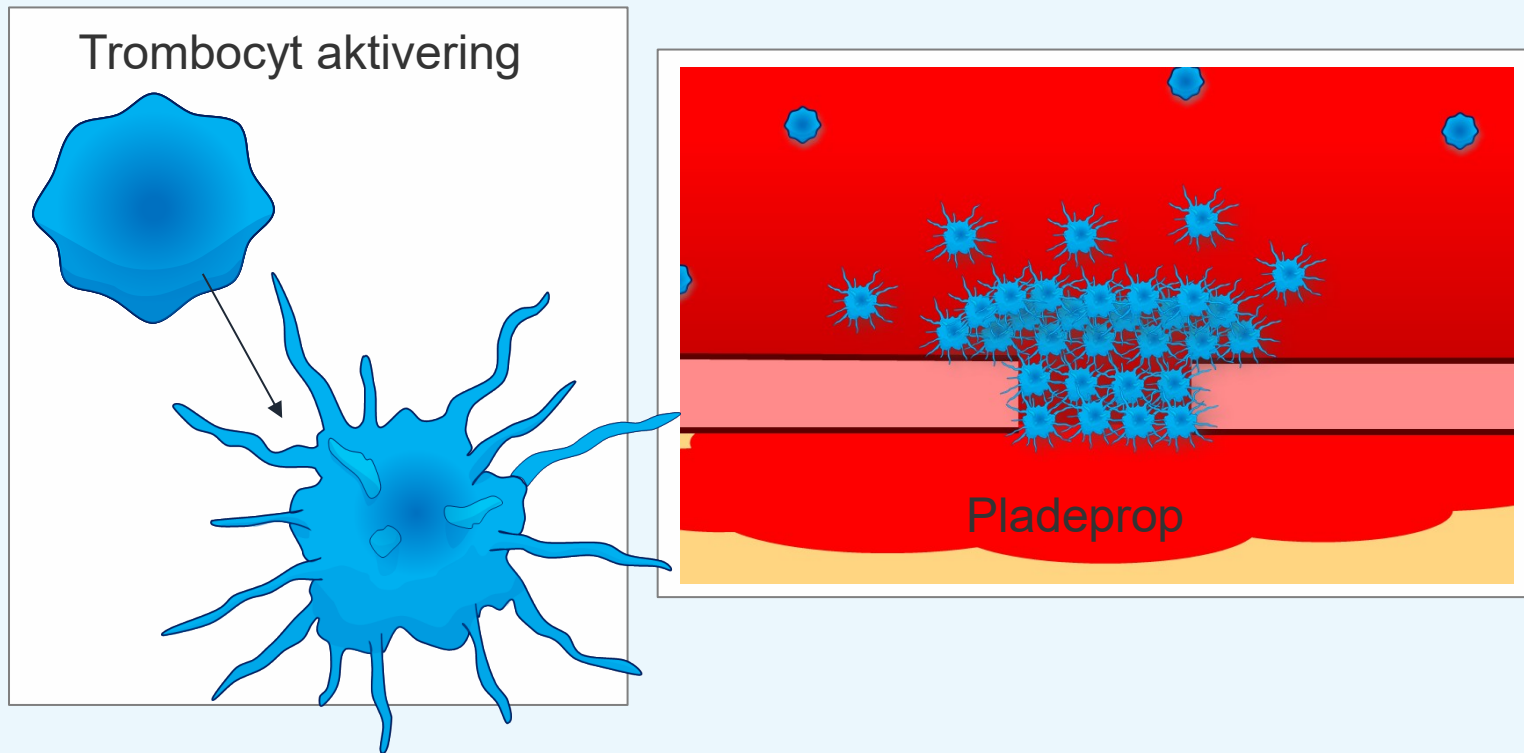
Hæmostase

1. **Reaktion fra karvæggen** → Karret trækker sig sammen



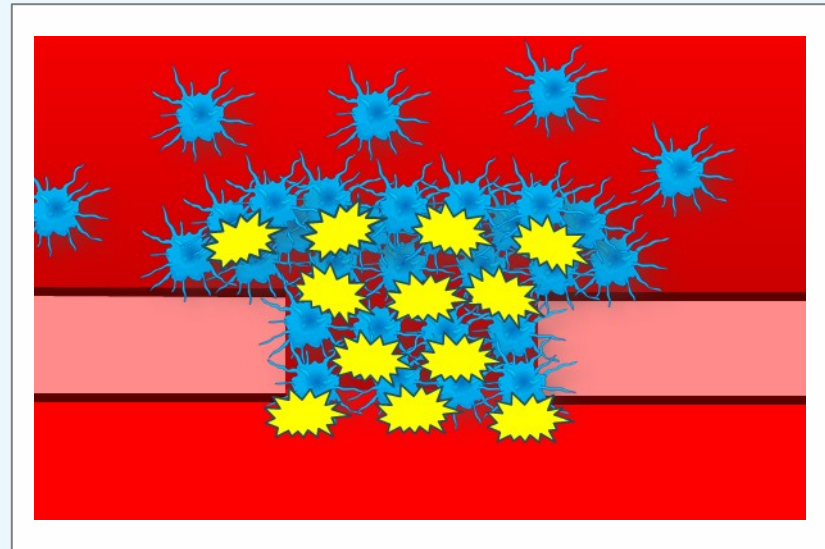
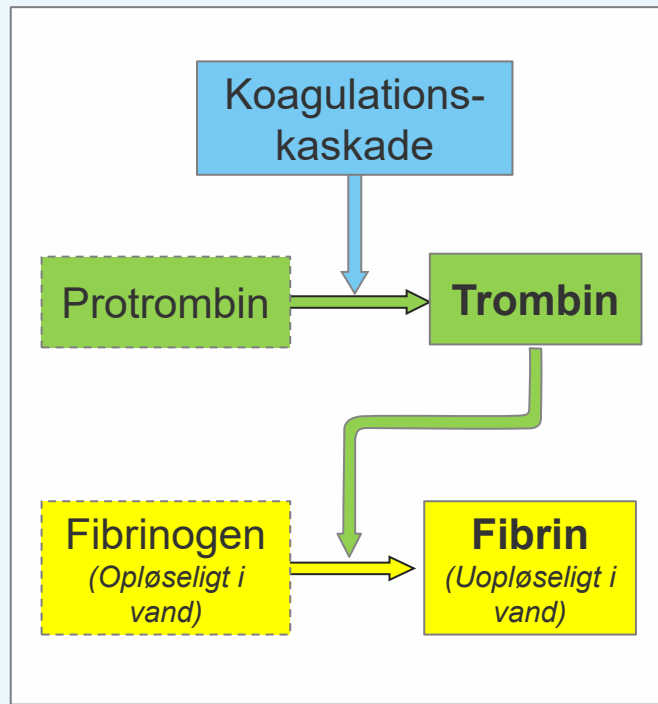
Hæmostase

2. Dannelse af en *pladeprop*



Hæmostase

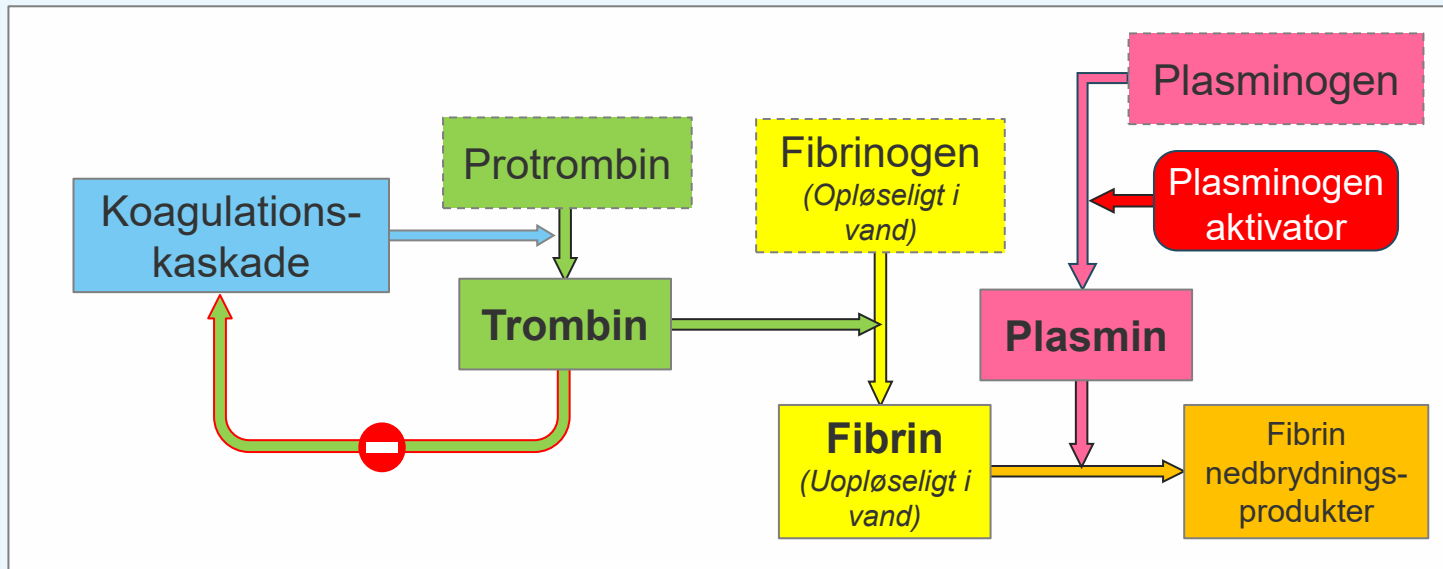
3. Dannelse af *fibrin* (Koagulation)



Hæmostase

4. Afgrænsning af blodproppen

- Fibrinolyse → nedbrydelse af fibrin vha. *plasmin*
- Inaktivering af koagulationsfaktorer



Hæmostase

Kan både aktiveres af faktorer fra *blodet* og faktorer fra *væv*

1. **Reaktion fra karvæggen** → Karret trækker sig sammen

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- Trombocytterne aktiveres → de binder sig fast til karvæggen og til hinanden og danner en

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- Koagulation af blodet → dannelse af *fibrin*

4. **Afgrænsning af blodproppen**

- Fibrinolyse → nedbrydelse af fibrin vha. /
- Inaktivering af koagulationsfaktorer

Pladehæmmere
Magnyl, Clopidogrel,
Brilique

AK: Marevan
DOAK: Eliquis,
Xarelto, Pradaxa
(Hepariner)

Trombolyse
Actilyse (Alteplase)

Tranexamsyre

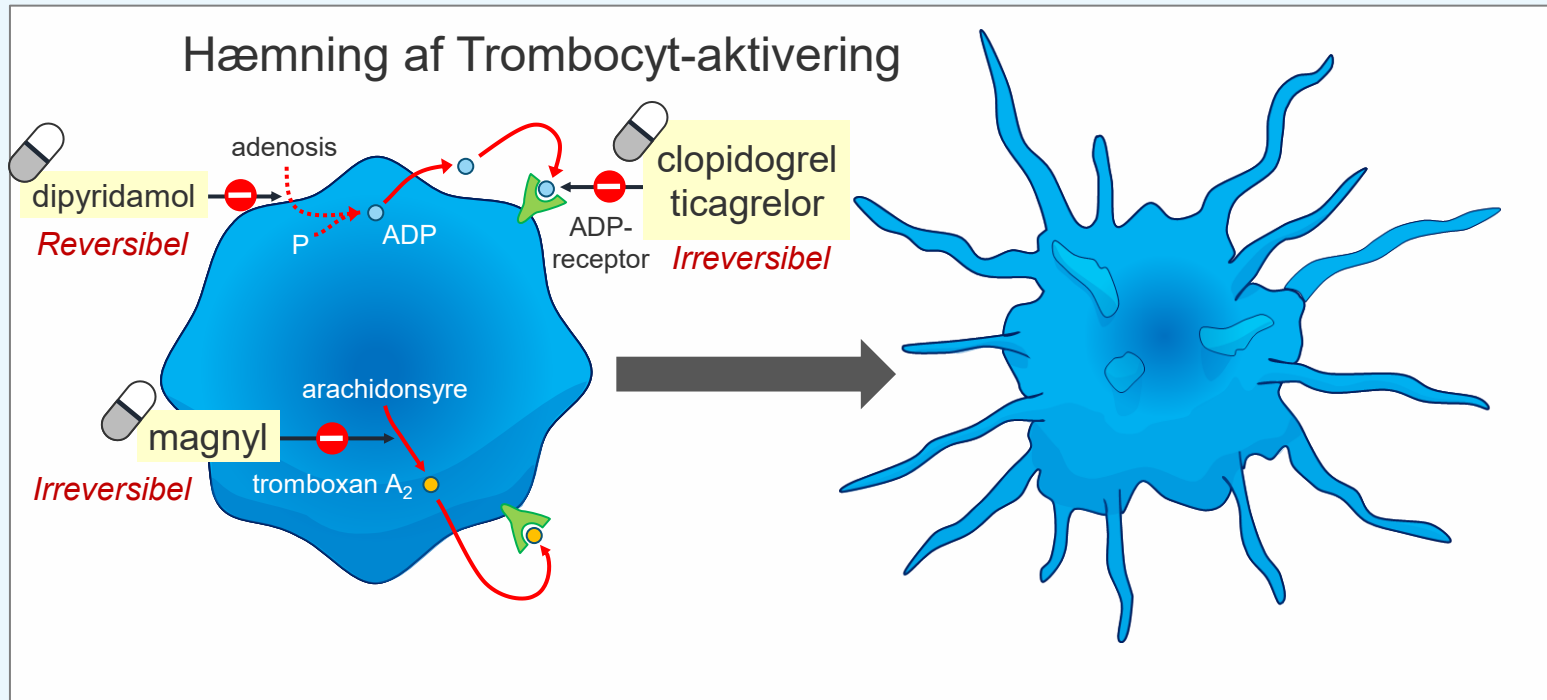
Hæmmer processen

Hæmmer processen

Fremmer processen

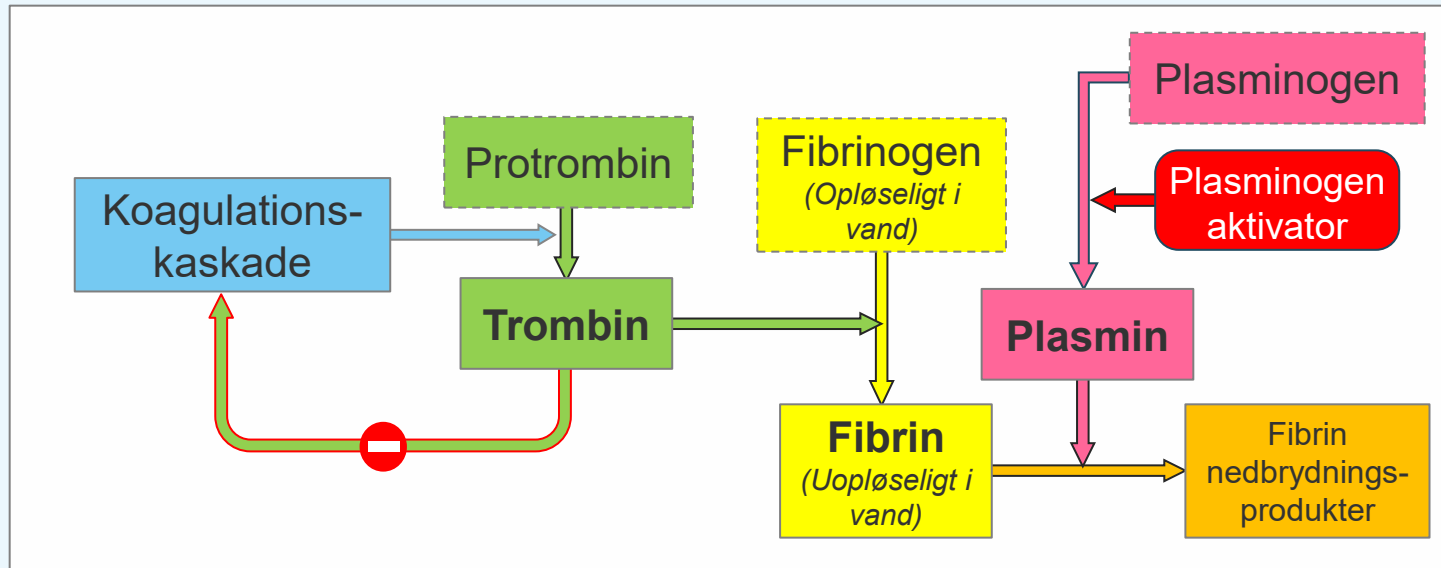
Pladehæmmere

2. Dannelse af en *pladeprop*

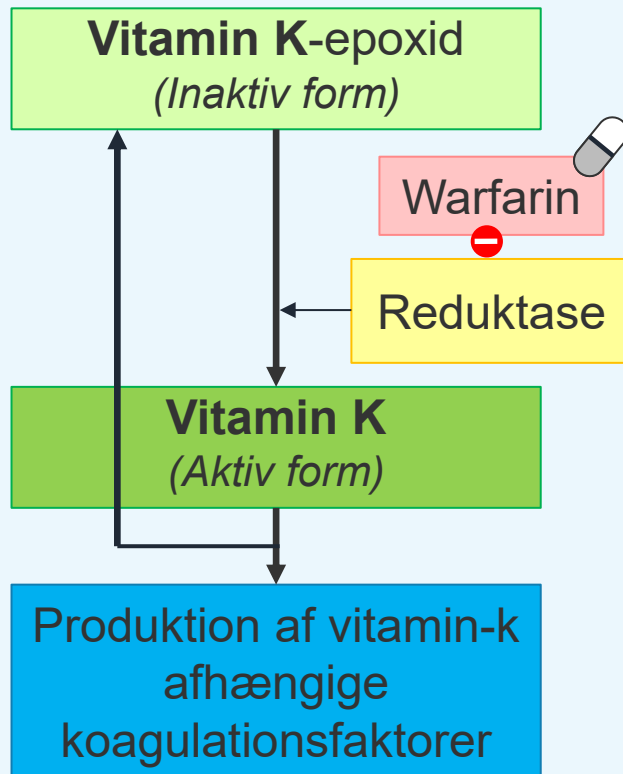


Antikoagulation (AK)- behandling

- Indirekte effekt (Warfarin)
- Direkte effekt (DOAK)
- Antitrombin III (Heparin)

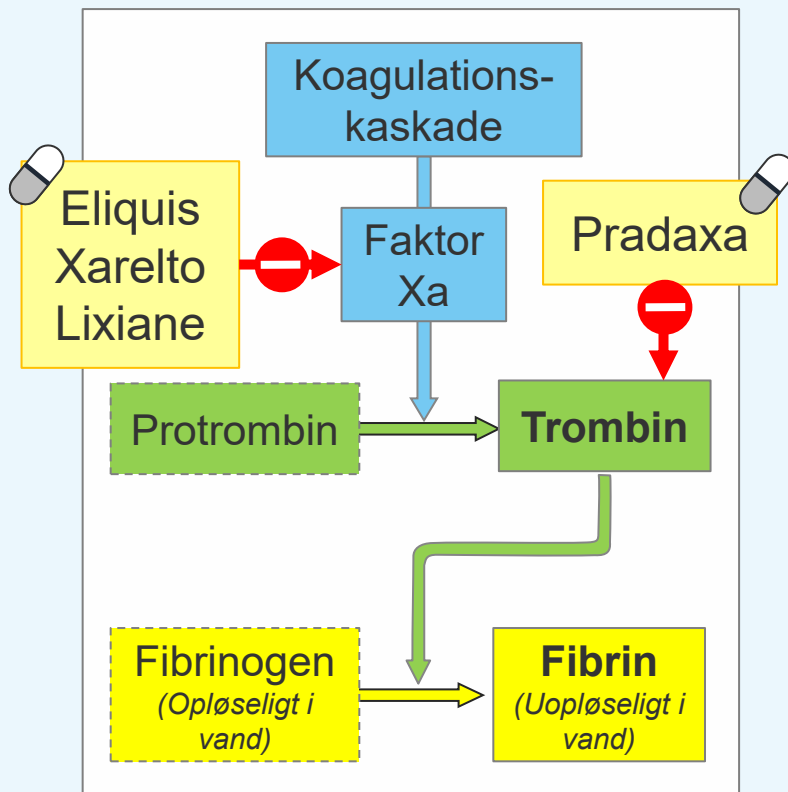


K-vitamin antagonister



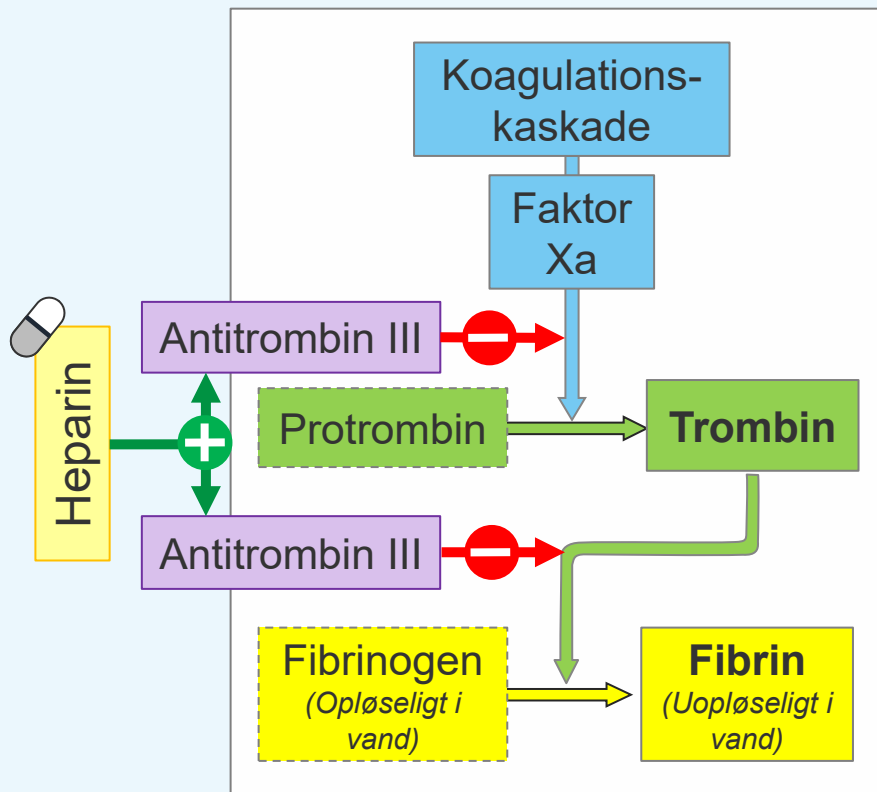
- Koagulationsfaktorerne produceres i leveren
- KF 2, 7, 9 og 10 kan kun produceres ved hjælp af aktivt Vitamin K
- Warfarin hæmmer aktiveringen af Vitamin K epoxid ved at hæmme enzymet *reduktase*
- Derved reduceres mængden af KF

Direkte Oral antikoagulation (DOAK)



- DOAK hæmmer koagulation ved direkte at hæmme enten Trombin eller Xa (Aktiveret Faktor 10)
- Virkningen er reversibel

Hepariner



- Hepariner virker ved at forstærke effekten af Antitrombin III
- Antitrombin III hæmmer både effekten af KF Xa og Trombin (KF IIa)

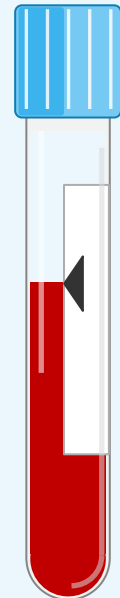
Marevan

DOAK

Effekt	Effekt omtrent ens, dog lidt færre bivirkninger ved DOAK	
Dosis	Variabel	Fast
Kost	Interaktion med vitamin-K- holdige fødevarer	Ingen interaktion Pradaxa kan ikke gives via sonde
Forsigtighed	Obs compliance og ændringer i kostindtag Interaktion med kosttilskud	Nedsat nyrefunktion, leversygdom, koagulationsforstyrrelser
Opfølgning	Behov for blodprøver og dosisjustering	Ingen
Forgiftning	Octaplex (Protrombin) IV vitamin-K og frisk frossen plasma	Pradaxa → PraxBind Aktuelt ingen antidot for Xa-hæmmerne

Biokemi

- **PP** (P-koagulationsfactor 2, 7, 10)
 - Måler indhold/effekt af KF 2, 7 og 10 i blodet
- **INR** | International Normalised Ratio
 - Standardiseret omregning af PP
 - Anvendes til regulering af Marevan/Marcoumar dosis
- **APTT** (Aktiveret partiel tromboplastin tid)
 - Måler effekten af KF 1, 2, 5, 8, 9, 10, 11 og 12



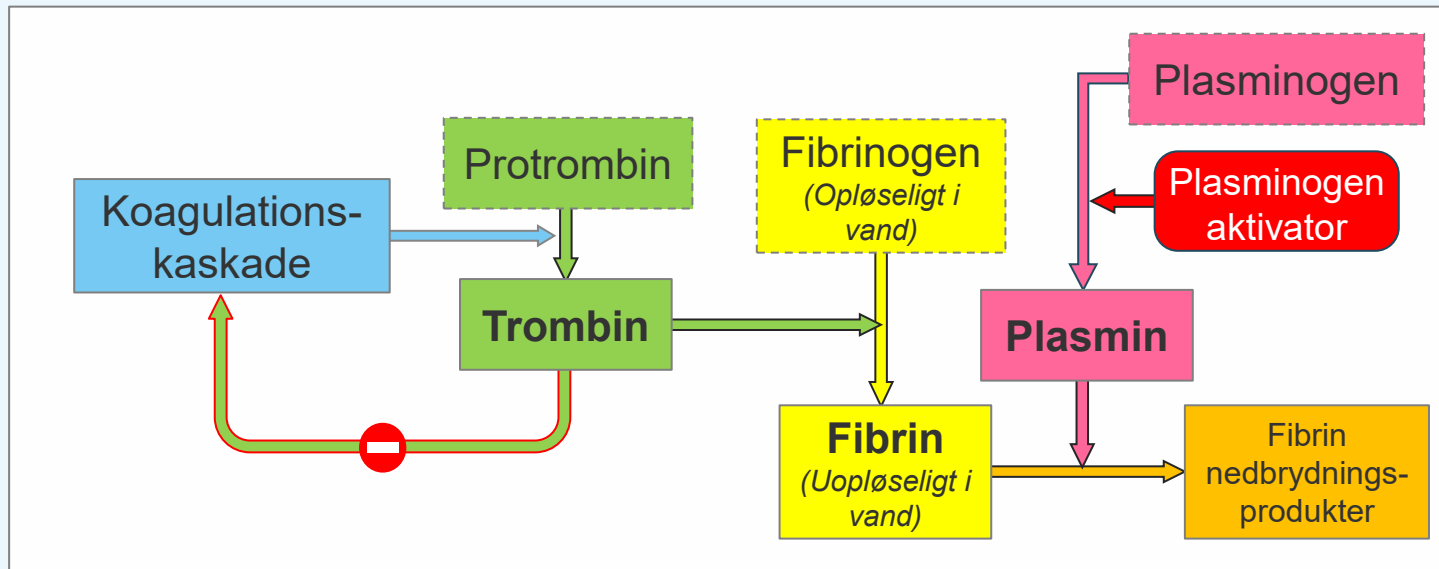
Tromboprophylakse ved iskæmisk apopleksi

Apopleksitype	Loading dosis		Fast dosis
TCl/minor stroke	300 mg Clopidogrel eller 300 mg Magnyl		75 mg Clopidogrel eller 75 mg Magnyl
TCl/minor stroke + øget risiko	300 mg Clopidogrel <u>og</u> 300 mg Magnyl	75 mg Clopidogrel <u>og</u> 75 mg Magnyl i 3 uger	75 mg Clopidogrel eller 75 mg Magnyl
Storkarssygdom	300 mg Clopidogrel <u>og</u> 300 mg Magnyl	75 mg Clopidogrel <u>og</u> 75 mg Magnyl i 3-12 uger	75 mg Clopidogrel eller 75 mg Magnyl
Kardioemboli	300 mg Clopidogrel eller 300 mg Magnyl		DOAK eller Marevan (INR 2-3)
Emboli af anden årsag	300 mg Clopidogrel og/eller 300 mg Magnyl	Eventuelt LMWH	Marevan

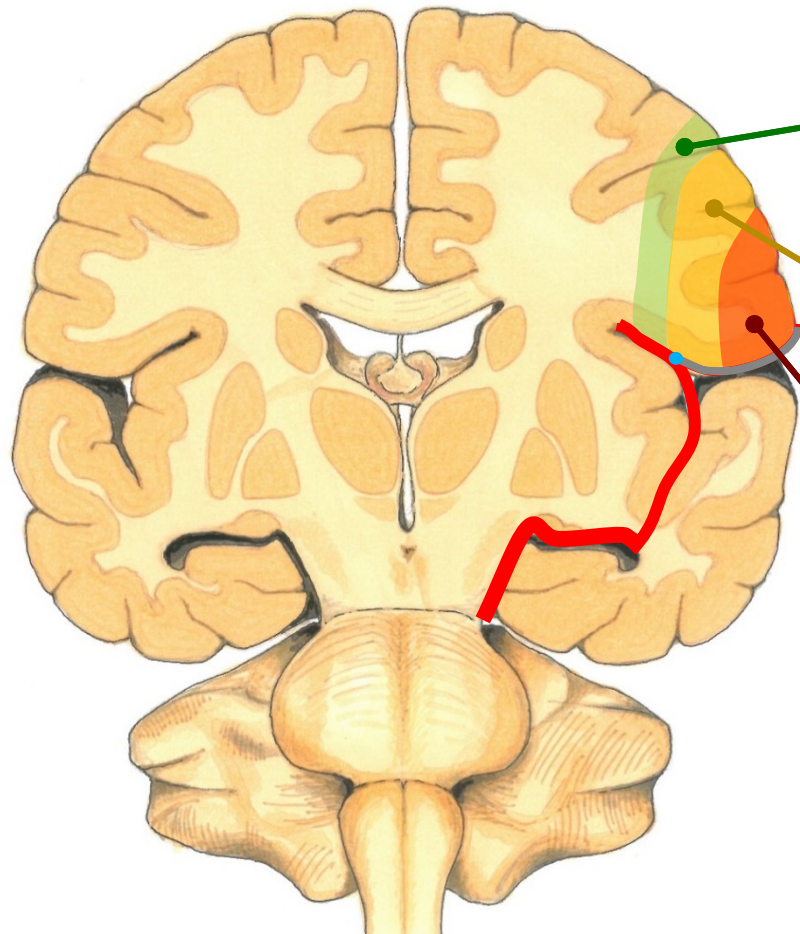
Trombolyse

Alteplase | Human vævsplasminogenaktivator (rt-PA)

Aktivering af plasmin spalter fibrin-netværket i blodproppen, så blodproppen går i stykker



Hvad sker der i vævet?



Perfusion 100-50 %: Normal funktion



Perfusion 50-20 %: **Penumbra**

- Skift fra aerob til anaerob metabolisme
- Nedsat funktion af vævet, men cellerne kan overleve i noget tid



Perfusion <20 %: **Infarkt**

- ATP bliver brugt op i løbet af få minutter
- Alle ATP-afhængige processer ophører
- Cellerne dør



Illustration: Jacob Liljehult 2021

Kapløb med tiden

Articles

Time to treatment with intravenous alteplase and outcome in stroke: an updated pooled analysis of ECASS, ATLANTIS, NINDS, and EPITHET trials

Kamran E. Cox, Jack B. Borstad, Ridger van Eemeren, Thomas G. Brott, Daphne Yon, Jeroen C. Geerds, Gregory W. Albers, Mark L. Frazee, John W. Hart, Scott Alexander, Barbara C. Fitz, Stephen M. Davis, Geoffrey S. Duncan, Wataru Hara, Lorna C. Cole, Elizabeth M. Wiggins, and ERNST n III Study Group Investigators

Summary Early administration of intravenous recombinant tissue plasminogen activator (rt-PA) after ischemic stroke improves outcomes. Previous analysis of combined data from individual patients suggested potential benefit beyond 3 h from stroke onset. We re-examined the effect of time to treatment with intravenous rt-PA (treated) on therapeutic benefit and clinical risk by adding recent trial data to the analysis.

Methods We added data from ECASS III (812 patients) and EPITHET (100 patients) to a pool of common data elements from six other trials of alteplase for acute stroke (OTX patients). We used multivariate logistic regression to assess the relation of stroke onset to time to treatment (OTT) with treatment on favorable 3-month outcome (defined as modified Rankin score 0–1), mortality, and occurrence and measures of clinical risk by any parenchymal hemorrhage. The presence of an arterial occlusion was inferred from the patient's symptoms and absence of hemorrhage or other causes of ischemic stroke. Vascular imaging was not a requirement in the trials. All patients with confirmed OTT within 360 min were included in the analysis.

Findings Treatment was started within 360 min of stroke onset in 3670 patients randomly allocated to alteplase (n=2076) or to placebo (n=1594). Odds of a favorable 3-month outcome increased as OTT decreased (p<0.001) and no benefit of alteplase treatment was seen after around 270 min. Adjusted odds of a favorable 3-month outcome were 2.55 (95% CI 1.44–4.52) for 0–60 min, 1.44 (1.12–2.2) for 61–120 min, 1.14 (0.8–1.48) for 181–270 min, and 1.22 (0.92–1.45) for 271–360 min in favor of the alteplase group. Large parenchymal hemorrhage was seen in 3% (2.5%) of 1856 patients assigned to alteplase and in 11.9% (10%) of 1810 controls, with no clear relation to OTT (p=0.416). Adjusted odds of mortality increased with OTT (p=0.044) and were 0.78 (0.4–1.4) for 0–60 min, 1.13 (0.79–1.62) for 61–120 min, 1.22 (0.87–1.71) for 181–270 min, and 1.1 (0.7–1.62) for 271–360 min.

Interpretation Patients with ischemic stroke selected by clinical symptoms and CT benefits from intravenous alteplase when treated again at 3 h. To increase benefits as a treatment, more often should be taken to shorten delay to initiation of treatment. But not 4–5 h, risk might outweigh benefits.

Funding None.

Introduction The alteplase-controlled trials have shown a substantial benefit of early treatment with intravenous recombinant tissue plasminogen activator (rt-PA) in patients with acute cerebral ischemia: the National Institute of Neurological Disorders and Stroke 2 (NINDS-2) trial in the USA, which started treatment within 3 h of stroke onset, and the European Cooperative Acute Stroke Study (ECASS) III trial in Europe, which started treatment 3–4.5 h from onset.^{1,2} The trials showed, independently or in conclusion, benefits of early rt-PA treatment.^{3,4} Analysis of 14 133 treatment-years from the trials showed that the benefit of treatment decreased as time from stroke onset to start of treatment increased.⁵ Thus, risk-benefit might change as the interval between stroke onset and treatment lengthens. Earlier studies have confirmed that some patients are treated with rt-PA beyond the currently approved time of 3 h from stroke onset.⁶ This approach might become more frequent because evidence from ECASS III suggests that treatment initiated at 3–4.5 h is safe and effective when patients with evidence of cerebral brain injury on CT scan are included.⁷ Since the first trial was designed, investigators have cooperated to ensure that important common data elements were gathered to avoid trials of rt-PA for acute stroke. In this updated pooled analysis, we aimed to assess the effect of time to treatment with intravenous rt-PA alteplase on therapeutic benefit and clinical risk. In particular, we postulated that the benefit of rt-PA would be smaller and the risk greater in patients who were treated at a later time point within the 4.5-hour window after stroke onset, because of reduced recovery of brain function after reperfusion and increased risk of death and brain hemorrhage.

www.thelancet.com Vol 375 May 12, 2010 195

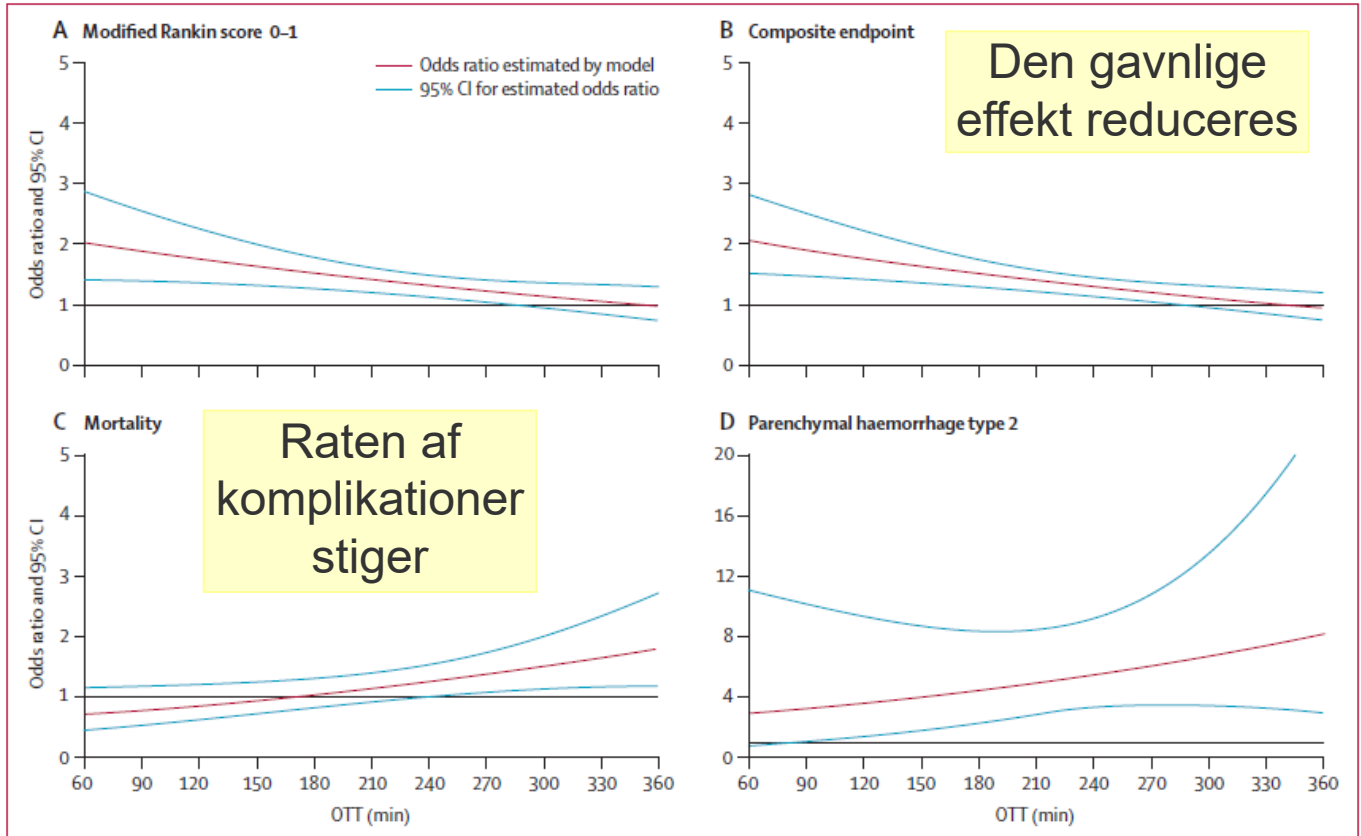
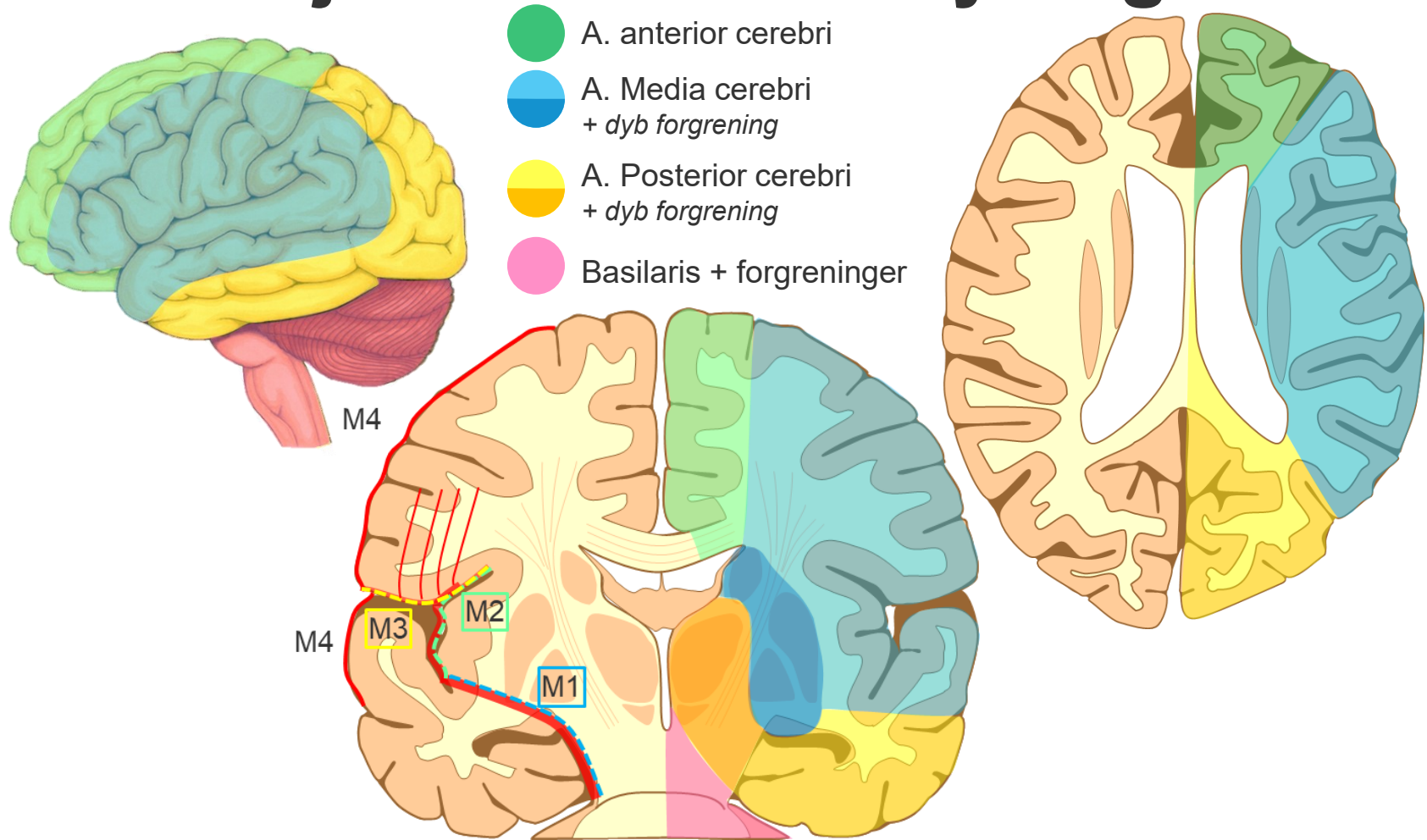


Figure 1: Relation of onset to treatment delay with treatment effect

Hjernens blodforsyning



Kilde: neuroanatomy.ca

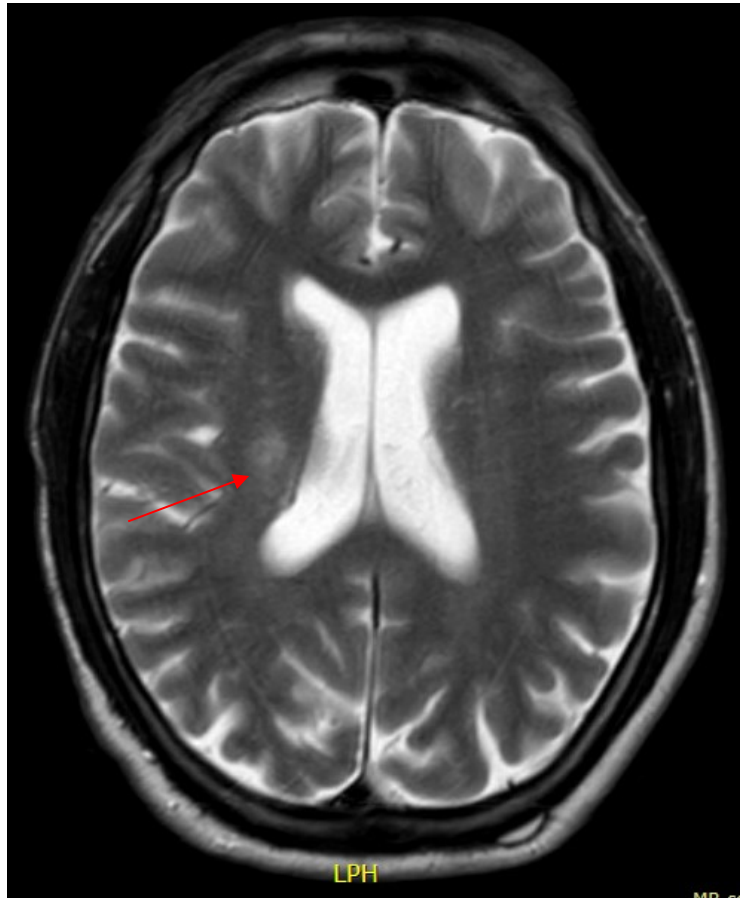


Følger efter
ældre infarkt

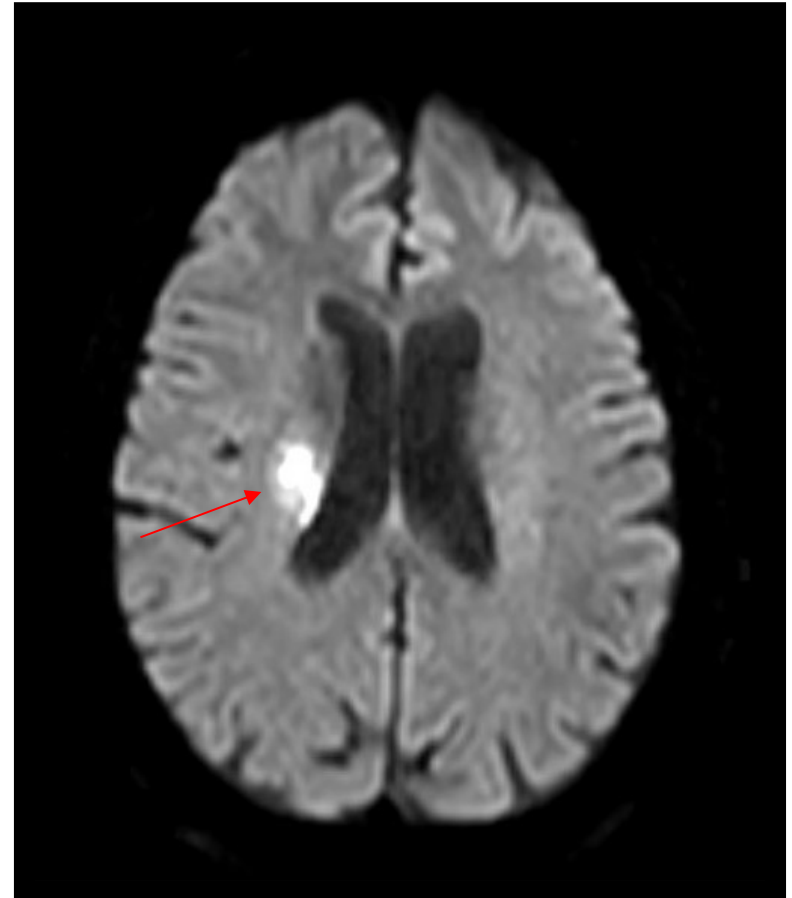
Akut infarkt i
a. media cerebri

- Hypodensitet
(mørkere farve)
- Tryk på lateral-
ventriklen
- Tryk på
overfladesulci

MR



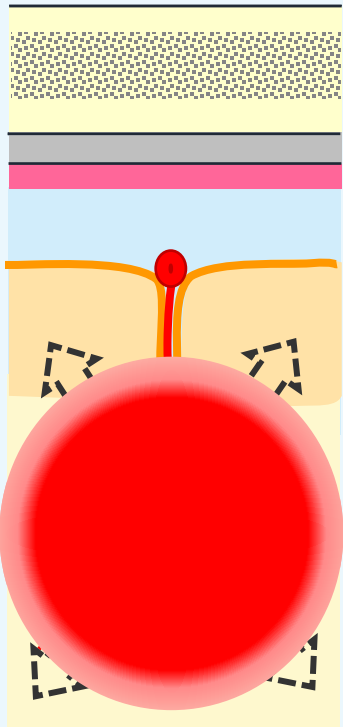
T2-vægtet



Diffusionsvægtet (DWI)

Hæmorrhagisk apopleksi

(Blødning)



Ætiologi

Hæmorrhagi betyder udsivning af blod fra blodbanen til et væv hvor der ikke ellers er blod

- Skyldes en revne på en arterie i hjernen
- Oftest pga. arteriosklerose -> arterierne bliver stive og skrøbelige
- Sjældent pga. aneurisme eller traume.

Hæmorrhagisk apopleksi (Blødning)

Patologi

- 1) **Cellerne i ramte område dør**
 - Direkte skade fra blodkomponenter
 - Blodet koagulerer og hæmmer gasudveksling
- 2) **Blodet fylder og trykker derfor på det omkringliggende hjernevæv**
(Forhøjet intra-kranielt tryk)
- 3) **Ødemdannelse omkring blødningen**
 - Udsivning af plasmaproteiner fra hæmatomet

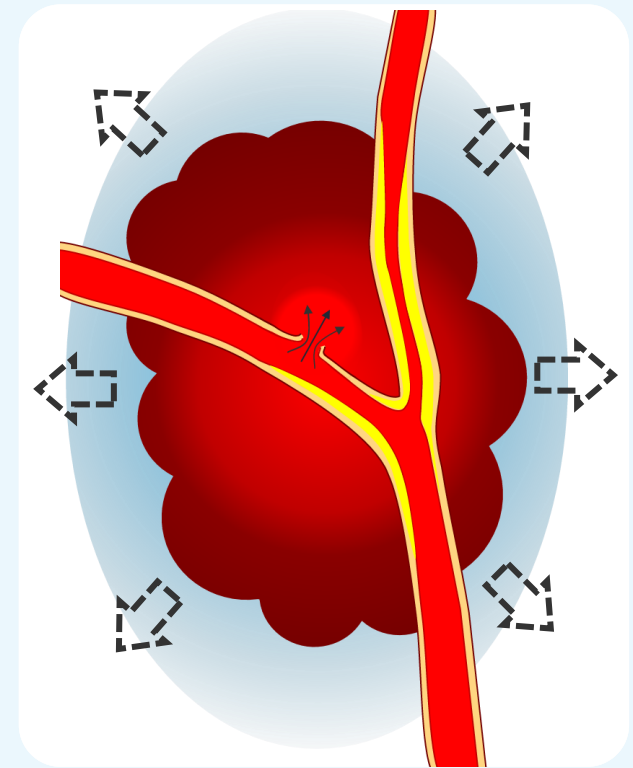
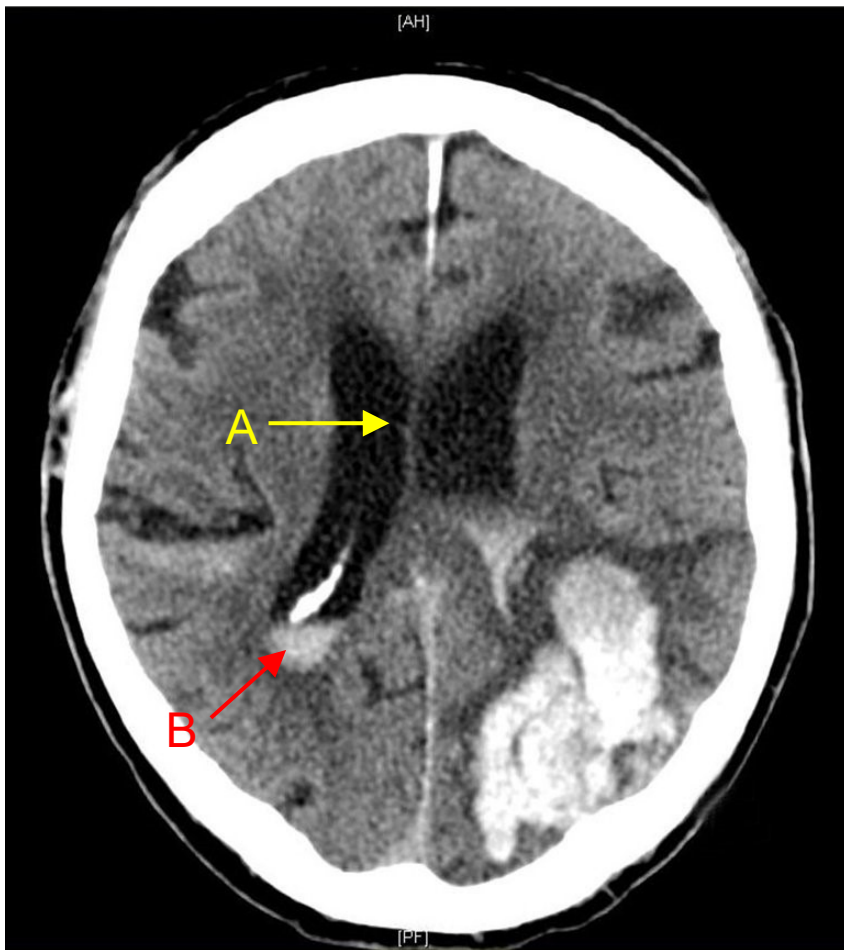
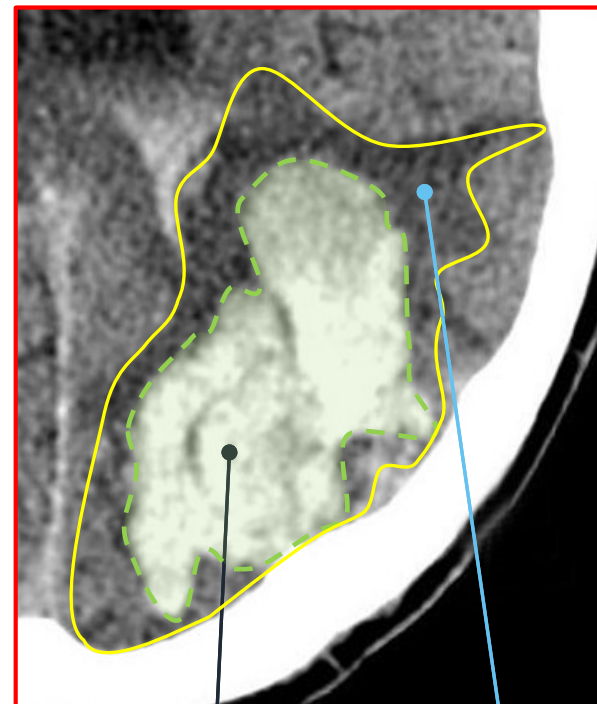


Illustration: Jacob Liljehult 2021



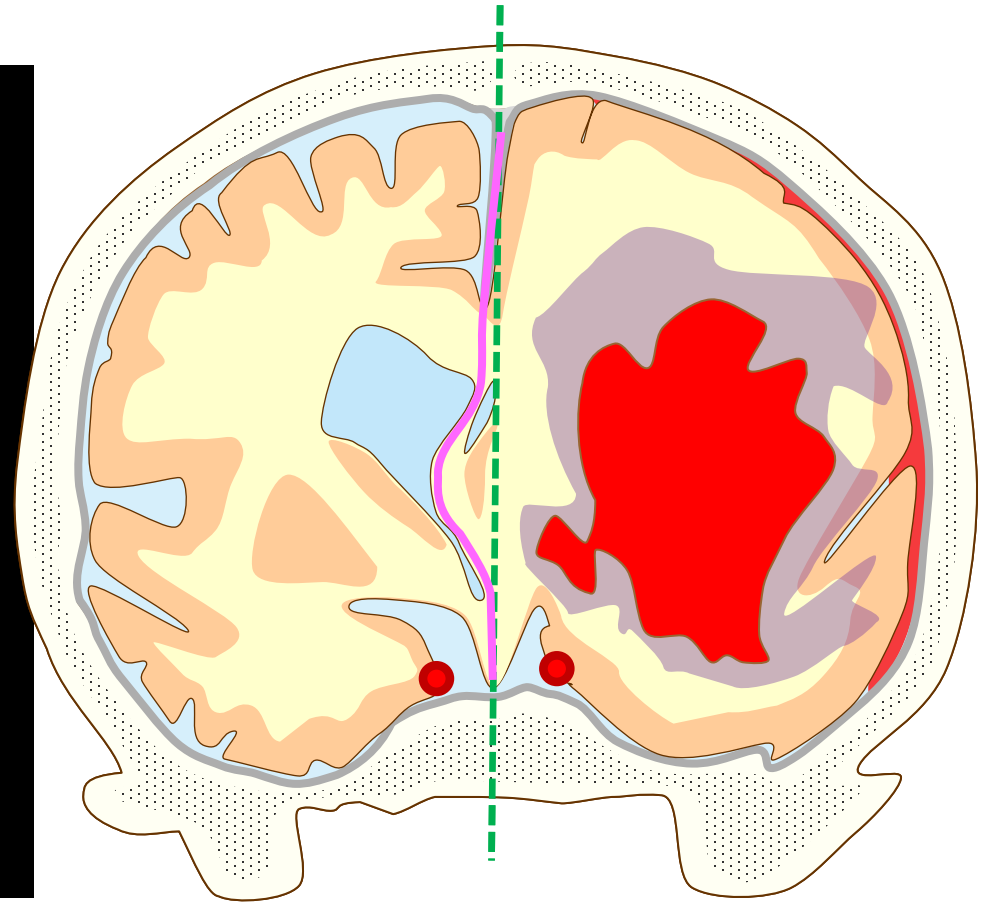
A Forskydning af septum over midtlinjen
B Blodrester i lateralventriklen



Blødning

Ødem

Blødning med gennembrud til begge lateral ventrikler



Blødning med ødem. Bemærk forskydning af midtlinjen og udfladning af overfladesulci

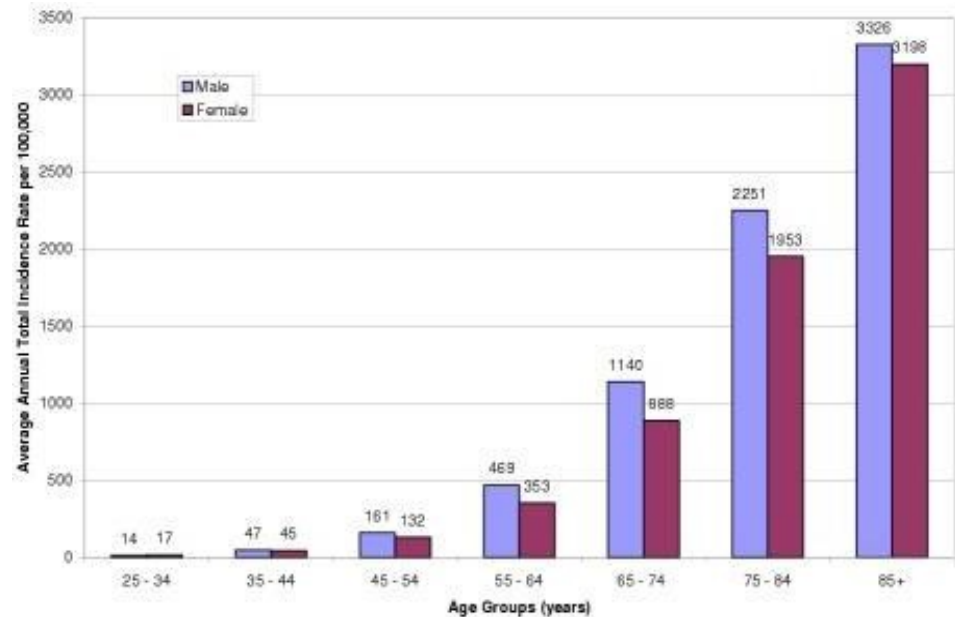
Forekomst

Ca. 12.000 nye tilfælde om året i DK

Incidensrate 3/1000 per år

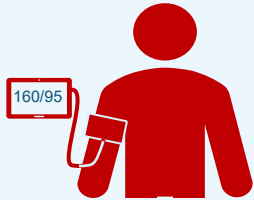
Hyppigheden stiger med alderen

Omkring 40.000 lever med *funktionsnedsættelse* efter apopleksi

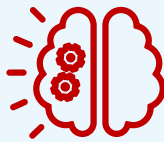


Risikofaktorer

Hypertension



Tidligere apopleksi



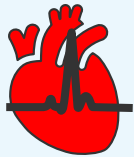
Rygning



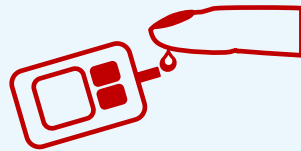
Alkohol



Atrieflimmer



Diabetes



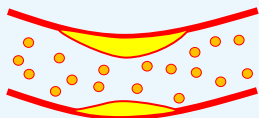
Inaktivitet



Overvægt



Kolesterol



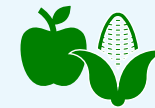
Stress og depression



Kost



Salt



Frugt/
grønt



Kød



Fisk

Risikofaktorer

Articles

Risk factors for ischaemic and intracerebral haemorrhagic stroke in 22 countries (the INTERSTROKE study): a case-control study

Martin J O'Donnell, Denis Xavier, Lisheng Liu, Hongye Zhang, Siu Lim Chin, Purnima Rao-Melacini, Sumathy Rangarajan, Shafiqul Islam, Prem Pais, Matthew J McQueen, Charles Mondo, Albertino Damasceno, Patricia Lopez-Jaramillo, Graeme J Hankey, Antonio L Dans, Khalid Yusoff, Thomas Truelsen, Hans-Christoph Diener, Ralph L Sacco, Danuta Kyjlewicka, Anna Czlonkowska, Christian Weimar, Xingyu Wang, Salm Yusuf, on behalf of the INTERSTROKE investigators*

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DOI:10.1016/S0140-6736(10)60834-3
See Comment page 74
*Members listed at end of paper

Background The contribution of various risk factors to the burden of stroke worldwide is unknown, particularly in countries of low and middle income. We aimed to establish the association of known and emerging risk factors with stroke and its primary subtypes, assess the contribution of these risk factors to the burden of stroke, and explore the differences between risk factors for stroke and myocardial infarction.

Methods We undertook a standardised case-control study in 22 countries worldwide between March 1, 2007, and April 23, 2010. Cases were patients with acute first stroke (within 5 days of symptoms onset and 72 h of hospital admission). Controls had no history of stroke, and were matched with cases for age and sex. All participants completed a structured questionnaire and a physical examination, and most provided blood and urine samples. We calculated odds ratios (ORs) and population-attributable risks (PARs) for the association of all stroke, ischaemic stroke, and intracerebral haemorrhagic stroke with selected risk factors.

Findings In the first 3000 cases (n=2337, 78%, with ischaemic stroke; n=663, 22%, with intracerebral haemorrhagic stroke) and 3000 controls, significant risk factors for all stroke were: history of hypertension (OR 2.64, 99% CI 2.26-3.08; PAR 34.6%; 99% CI 30.4-39.1); current smoking (2.09, 1.75-2.51; 18.9%, 15.3-23.1); waist-to-hip ratio (1.65, 1.36-1.99 for highest vs lowest tertile; 26.5%, 18.8-36.0); diet risk score (1.35, 1.11-1.64 for highest vs lowest tertile; 18.8%, 11.2-29.7); regular physical activity (0.69, 0.53-0.90; 28.5%, 14.5-48.5); diabetes mellitus (1.36, 1.10-1.68; 5.0%, 2.6-9.5); alcohol intake (1.51, 1.18-1.92 for more than 30 drinks per month or binge drinking; 3.8%, 0.9-14.4); psychosocial stress (1.30, 1.06-1.60; 4.6%, 2.1-9.6) and depression (1.35, 1.10-1.66; 5.2%, 2.7-9.8); cardiac causes (2.38, 1.77-3.20; 6.7%, 4.8-9.1); and ratio of apolipoproteins B to A1 (1.89, 1.49-2.40 for highest vs lowest tertile; 24.9%, 15.7-37.1). Collectively, these risk factors accounted for 88.1% (99% CI 82.3-92.2) of the PAR for all stroke. When an alternate definition of hypertension was used (history of hypertension or blood pressure >160/90 mm Hg), the combined PAR was 90.3% (85.3-93.7) for all stroke. These risk factors were all significant for ischaemic stroke, whereas hypertension, smoking, waist-to-hip ratio, diet, and alcohol intake were significant risk factors for intracerebral haemorrhagic stroke.

Interpretation Our findings suggest that ten risk factors are associated with 90% of the risk of stroke. Targeted interventions that reduce blood pressure and smoking, and promote physical activity and a healthy diet, could substantially reduce the burden of stroke.

Hypertension (OR 2.64 [2.26-3.08]*)

Kardielle årsager (OR 2.38 [1.77-3.20]*)

Rygning (OR 2.09 [1.75-2.51]*)

Talje/hofte ratio (OR 1.65 [1.36-1.99]*)

Alkohol (OR 1.51 [1.18-1.92]*)

Fysisk inaktivitet (OR 1.45 [1.11-1.89]*)

Diæt (OR 1.35 [1.11-1.64]*)

Depression (OR 1.35 [1.10-1.66]*)

Stress (OR 1.30 [1.06-1.60]*)

* 99% Konfidensinterval

De mørke områder angiver population attributional risk

Stroke

AHA/ASA GUIDELINE

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

Reviewed for evidence-based integrity and endorsed by the American Association of Neurological Surgeons and Congress of Neurological Surgeons.

Endorsed by the Society of Vascular and Interventional Neurology

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

Endorsed by the Neurocritical Care Society

Steven M. Greenberg, MD, PhD, FAHA, Chair; Wendy C. Zai, MD, MPH, FAHA, Vice Chair; Charlotte Cordonnier, MD, PhD; Dar Dowlatzahi, MD, PhD, FAHA; Brandon Francis, MD, MPH; Joshua N. Goldstein, MD, PhD, FAHA; J. Claude Hemphill III, MD, MAS, FAHA; Ronda Johnson, MBA; Kiffon M. Keigher, MSN, ACNP-BC, RN, SCRNP; William J. Mack, MD, MS, FAHA; J. Mocco, MD, MS, FAHA; Eileena J. Newton, MD; Ilana M. Ruff, MD; Lauren H. Sansing, MD, MS, FAHA; Sam Schulman, MD, PhD; Magdy H. Solim, MD, PhD, FAHA; Kevin N. Sheth, MD, FAHA; Nikola Sprigg, MD; Katharina S. Sunnerhagen, MD, PhD; on behalf of the American Heart Association/American Stroke Association

Key Words: AHA Scientific Statements ■ cerebral amyloid angiopathy ■ cerebral hemorrhage ■ intracranial hemorrhage ■ prevention ■ recovery ■ treatment

TOP 10 TAKE-HOME MESSAGES FOR THE MANAGEMENT OF PATIENTS WITH SPONTANEOUS INTRACEREBRAL HEMORRHAGE GUIDELINE

1. The organization of health care systems is increasingly recognized as a key component of optimal stroke care. This guideline recommends development of regional systems that provide initial intracerebral hemorrhage (ICH) care and the capacity, when appropriate, for rapid transfer to facilities with neurocritical care and neurosurgical capabilities.
2. Hematoma expansion is associated with worse ICH outcome. There is now a range of neuroimaging markers that, along with clinical markers such as time since stroke onset and use of antithrombotic

- agents, help to predict the risk of hematoma expansion. These neuroimaging markers include signs detectable by noncontrast computed tomography, the most widely used neuroimaging modality for ICH.
3. ICHs, like other forms of stroke, occur as the consequence of a defined set of vascular pathologies. This guideline emphasizes the importance of, and approaches to, identifying markers of both microvascular and macrovascular hemorrhage pathogenesis.
 4. When implementing acute blood pressure lowering after mild to moderate ICH, treatment regimens that limit blood pressure variability and achieve smooth, sustained blood pressure control appear to reduce hematoma expansion and yield better functional outcome.

1AHA Stroke Council Scientific Statement Oversight Committee on Clinical Practice Guideline Issues, IAANS/CNS Issues, IAHA Stroke Council Stroke Performance Measures Oversight Committee Issues, SAAN representative.
AHA Stroke Council Scientific Statement Oversight Committee members, see page e337.
Supplemental material is available at <https://www.ahajournals.org/doi/suppl/10.1161/STR.0000000000000407>
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Stroke is available at www.ahajournals.org/journal/str

9.1.5. Lifestyle Modifications/Patient and Caregiver Education

Recommendations for Lifestyle Modifications/Patient and Caregiver Education

Referenced studies that support recommendations are summarized in Data Supplement 82.

COR	LOE	Recommendations
Lifestyle modification		
2a	C-LD	1. In patients with spontaneous ICH, lifestyle modification is reasonable to reduce BF. ⁶³²
2a	C-LD	2. In patients with spontaneous ICH, avoiding heavy alcohol consumption is reasonable to reduce hypertension and risk of ICH recurrence. ^{633–635}
2b	C-LD	3. In patients with spontaneous ICH, lifestyle modification, including supervised training and counseling, may be reasonable to improve functional recovery. ^{636,637}

636. English C, Healy GN, Olds T, Parfitt G, Borkoles E, Coates A, Kramer S, Bernhardt J. Reducing sitting time after stroke: a phase II safety and feasibility randomized controlled trial. *Arch Phys Med Rehabil.* 2016;97:273–280. doi: 10.1016/j.apmr.2015.10.094

637. Liljehult J, Christensen T, Molsted S, Overgaard D, Mesot Liljehult M, Møller T. Effect and efficacy of lifestyle interventions as secondary prevention. *Acta Neurol Scand.* 2020;142:299–313. doi: 10.1111/ane.13308

Prognose

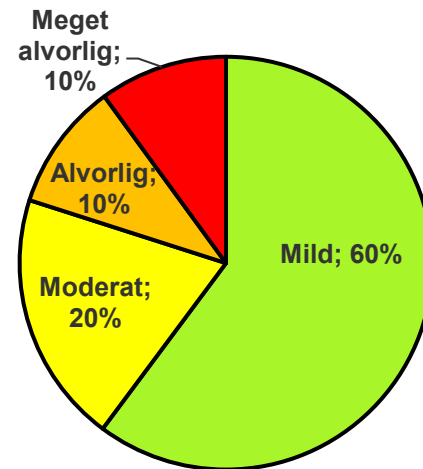
Prognosen afhænger af sværhedsgraden

50 % bliver selvhjulpne
10-20 % bliver svært invaliderede

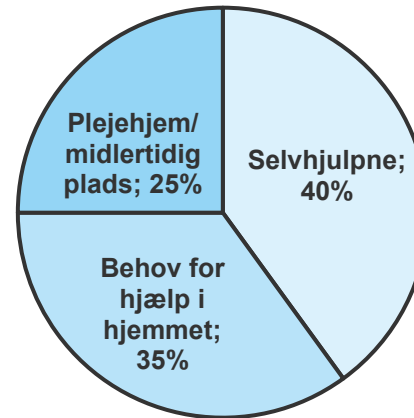
10 % får en ny apopleksi inden for 1 år

9-11 % dør indenfor 30 dage
15-20 % indenfor 1 år

Ca. 10 % udvikler epilepsi



Sværhedsgrad
(Indlæggelsen)



Funktionsniveau
(mRS - udskrivelsen)

Symptomer på apopleksi

Diffuse symptomer

Nedsat
bevidsthed

Utilpashed

Svimmelhed

Hovedpine

Kramper

Fokale udfald

- Motorisk og somato-sensoriske udfald
- Synsforstyrrelser
- Sproglige og kommunikative problemer
- Visuo-spartielle forstyrrelser
 - *Neglekt og inattention, apraksi*
- Adfærds- og opmærksomhedsforstyrrelser
- Anosognosi

Neurologiske udfald

De neurologiske udfald
afhænger af:

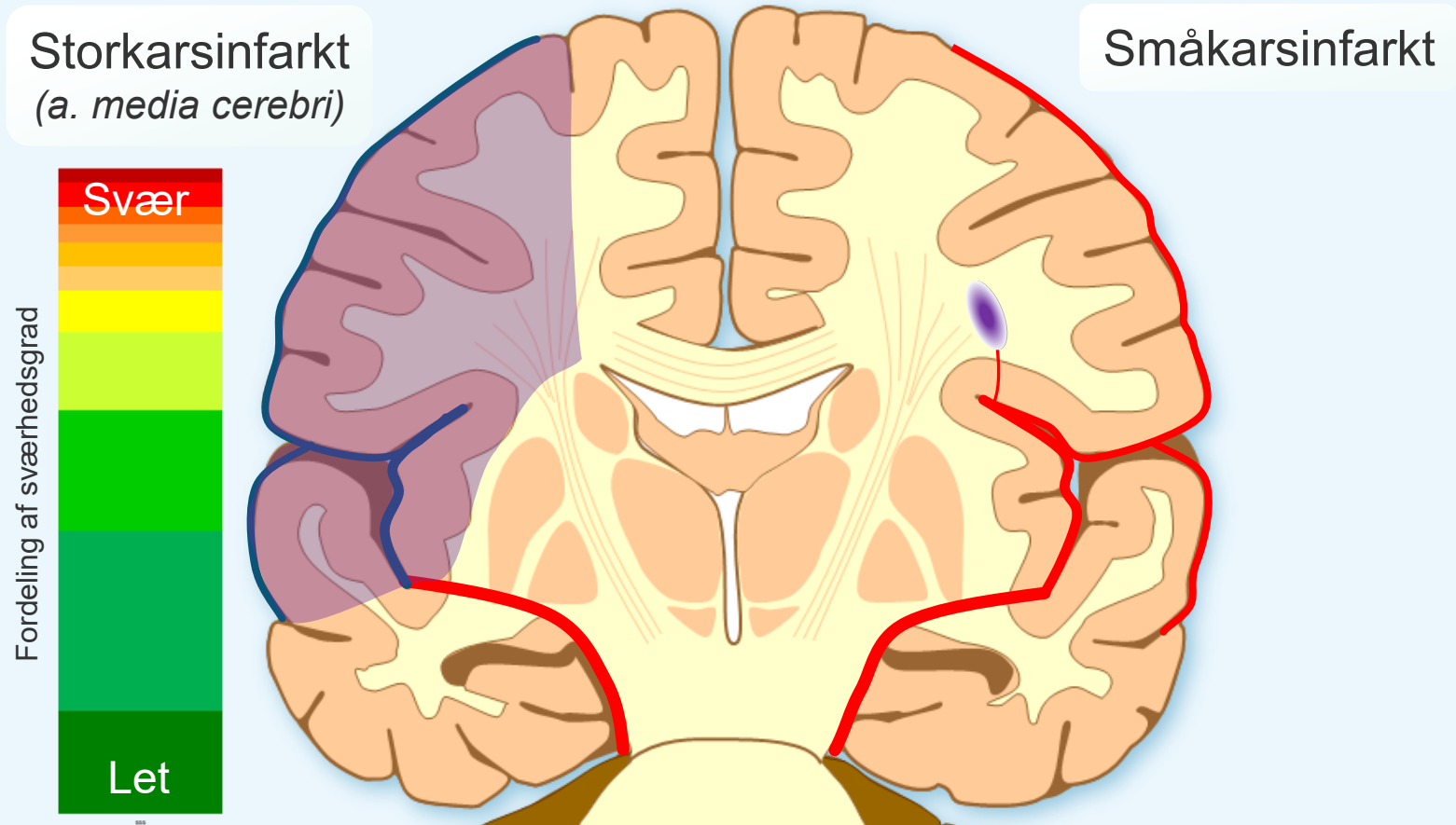
**Omfanget af
læsionen**

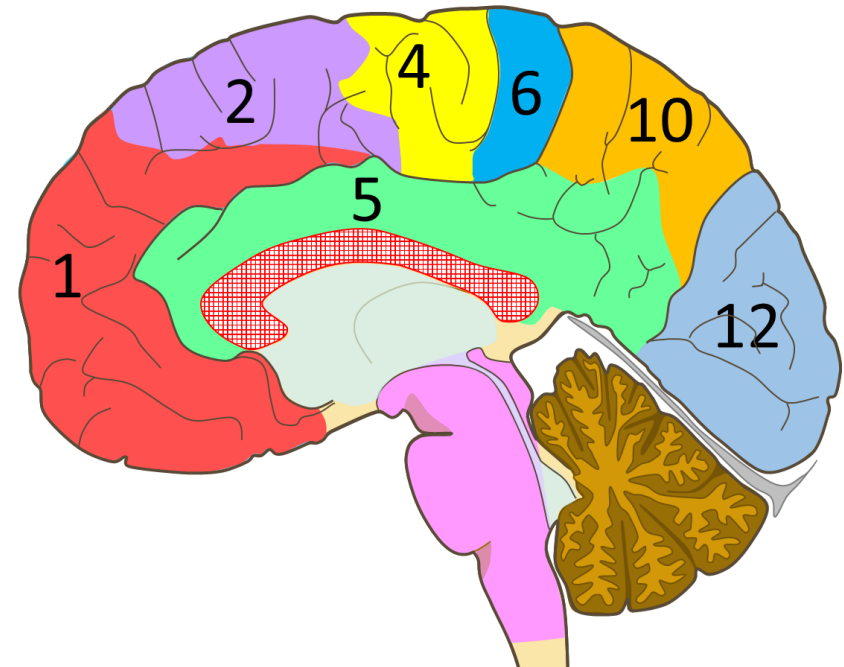
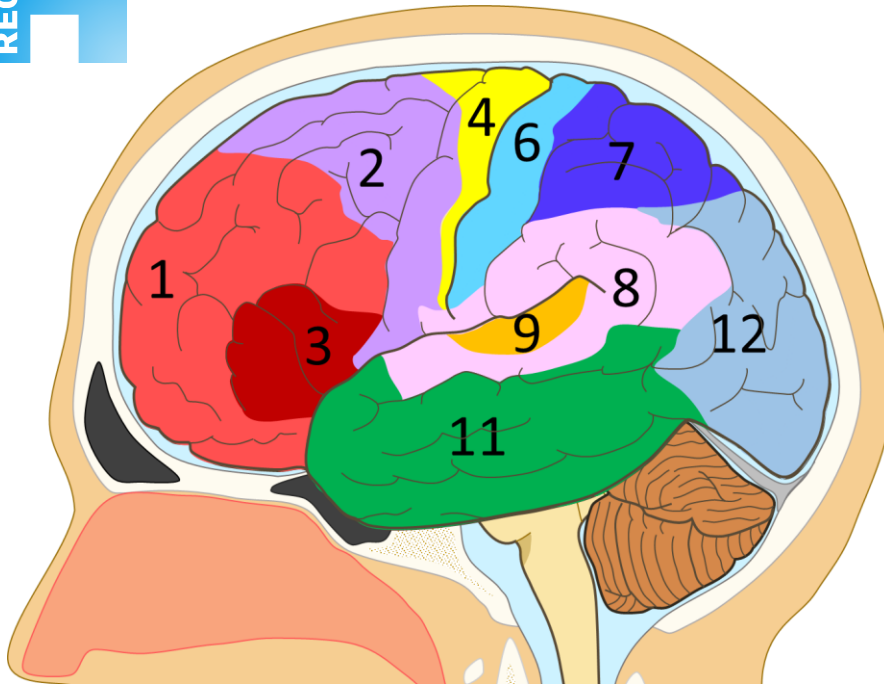
*Hvor stort et
område er ramt?*

**Placeringen af
læsionen**

*Hvilket område
er ramt?*

Omfanget af læsionen





1	Højere mentale funktioner Koncentration, planlægning, dømmekraft, impulshæmning, kreativitet	5	Emotionelt område Smerte, sult, "fight & flight"	10	Associativt sensorisk område
2	Sekundært motorisk område Øjenbevægelser, orientering, skemaer for bevægelser	6	Primært somato-sensorisk område	11	Associativt område Korttidshukommelse, emotioner
3	Broca's område Motorisk del af talen	7	Sekundært somato-sensorisk område Vurdering af tekstur, vægt etc	12	Visuelt område Syn, objektgenkendelse
4	Primært motorisk område Initiering af bevægelser	8	Wernickes område Sprog forståelse	13	Cerebellum Balance, kropsholdning, koordination af bevægelser
		9	Auditorisk område Hørelse		

Illustration: Jacob Liljehult 2020

Hemisfære-lateralisering

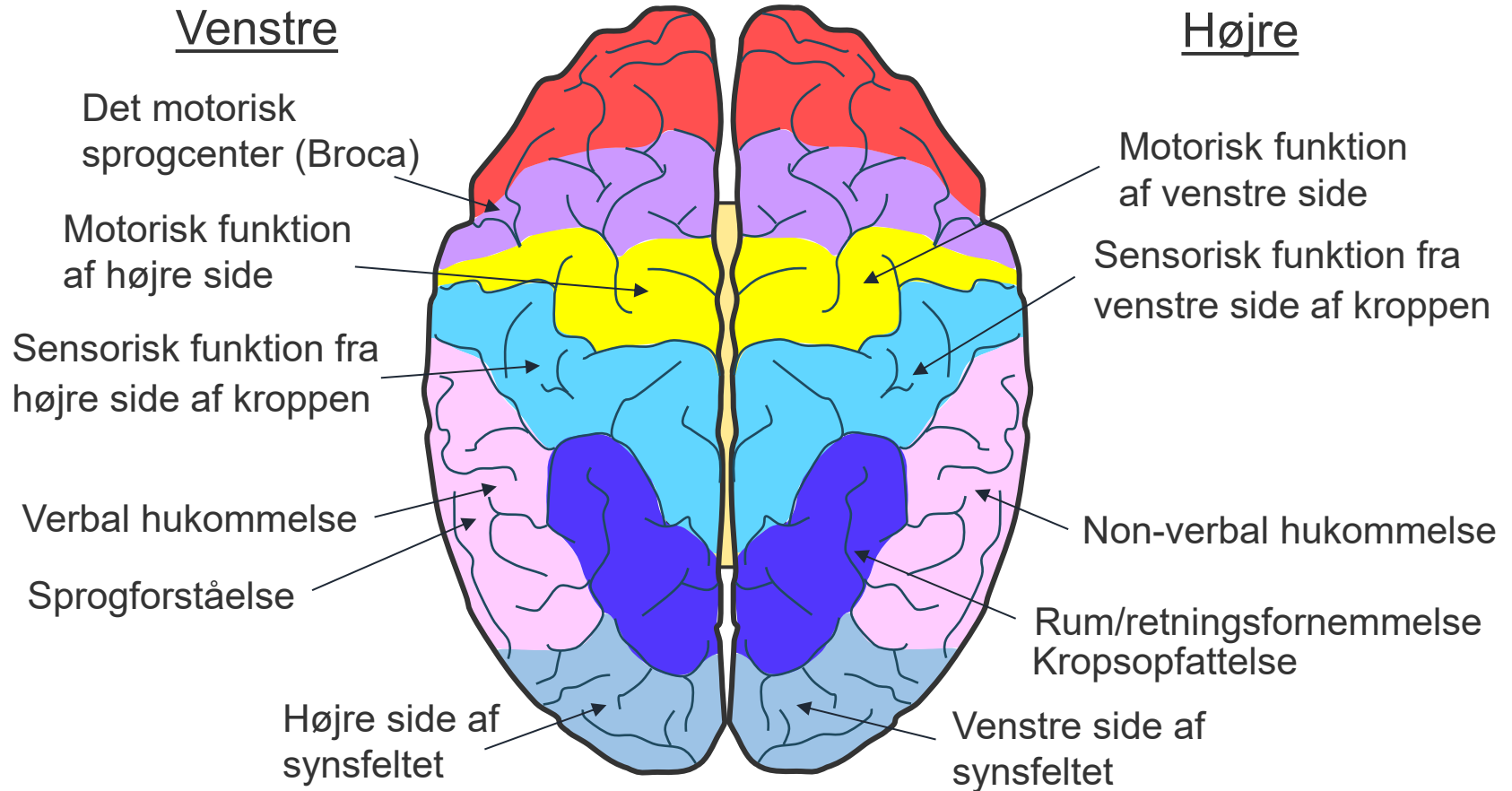
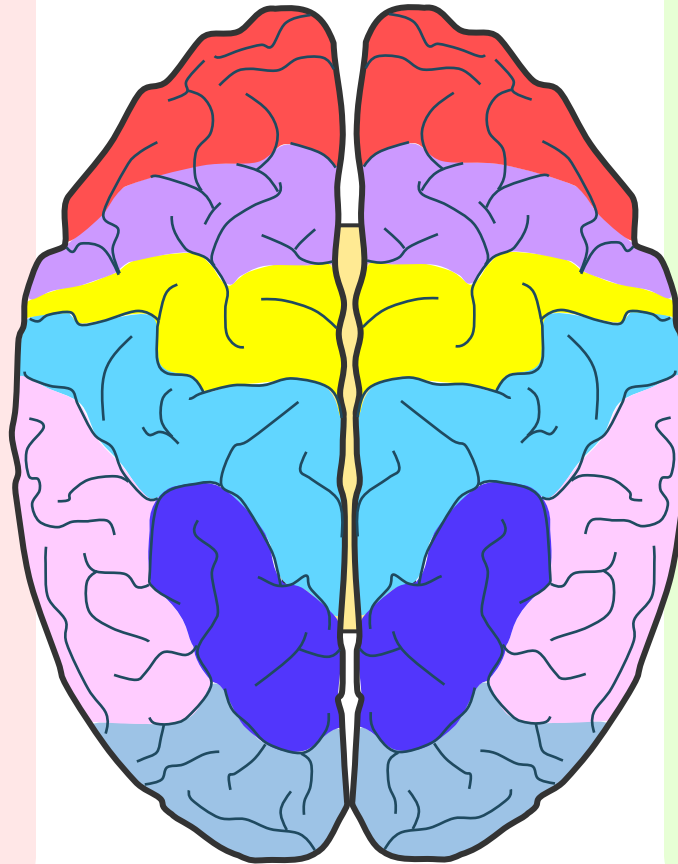


Illustration: Jacob Liljehult 2020

Hemisfære-lateralisering

Venstre

- Højresidige motoriske og sensoriske udfald
- Højresidige synsudfald
- Sproglige udfald (*tale, forståelse, læse, skrive, regne*)
- Apraksi
- Negativ affekt/ nedsat psyko-motorisk tempo



Højre

- Venstresidige motoriske og sensoriske udfald
- Venstresidige synsudfald
- Nedsat sygdoms-erkendelse
- Manglende overblik/ rum-retnings forstyrrelser
- Neglekt
- Positiv affekt/ hurtigt psyko-motorisk tempo

Illustration: Jacob Liljehult 2020

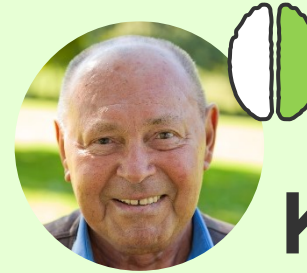
Cases: Apopleksi



Venstre hemisfære

Inge 78 år

Under en frokost bliver hun pludseligt utilpas og får svært ved at løfte sin højre arm ordentligt. Da hun vil fortælle sin veninden, at hun er skidt, kan hun få andet ud end *"Det.. arm.. arm.. nej, nej.. det..."*. Veninden kan hurtigt se at der er noget galt og ringer 1-1-2.

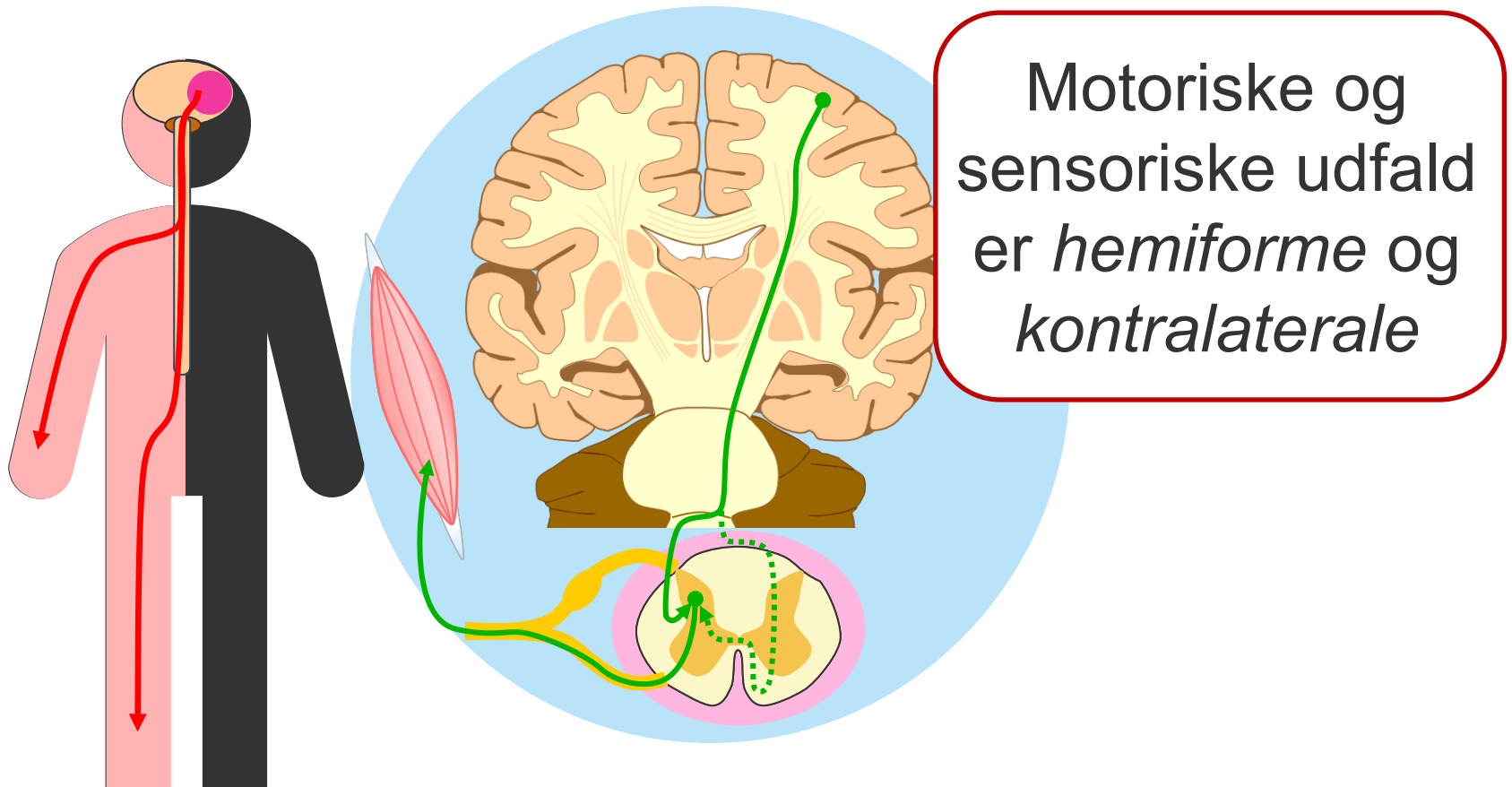


Højre hemisfære

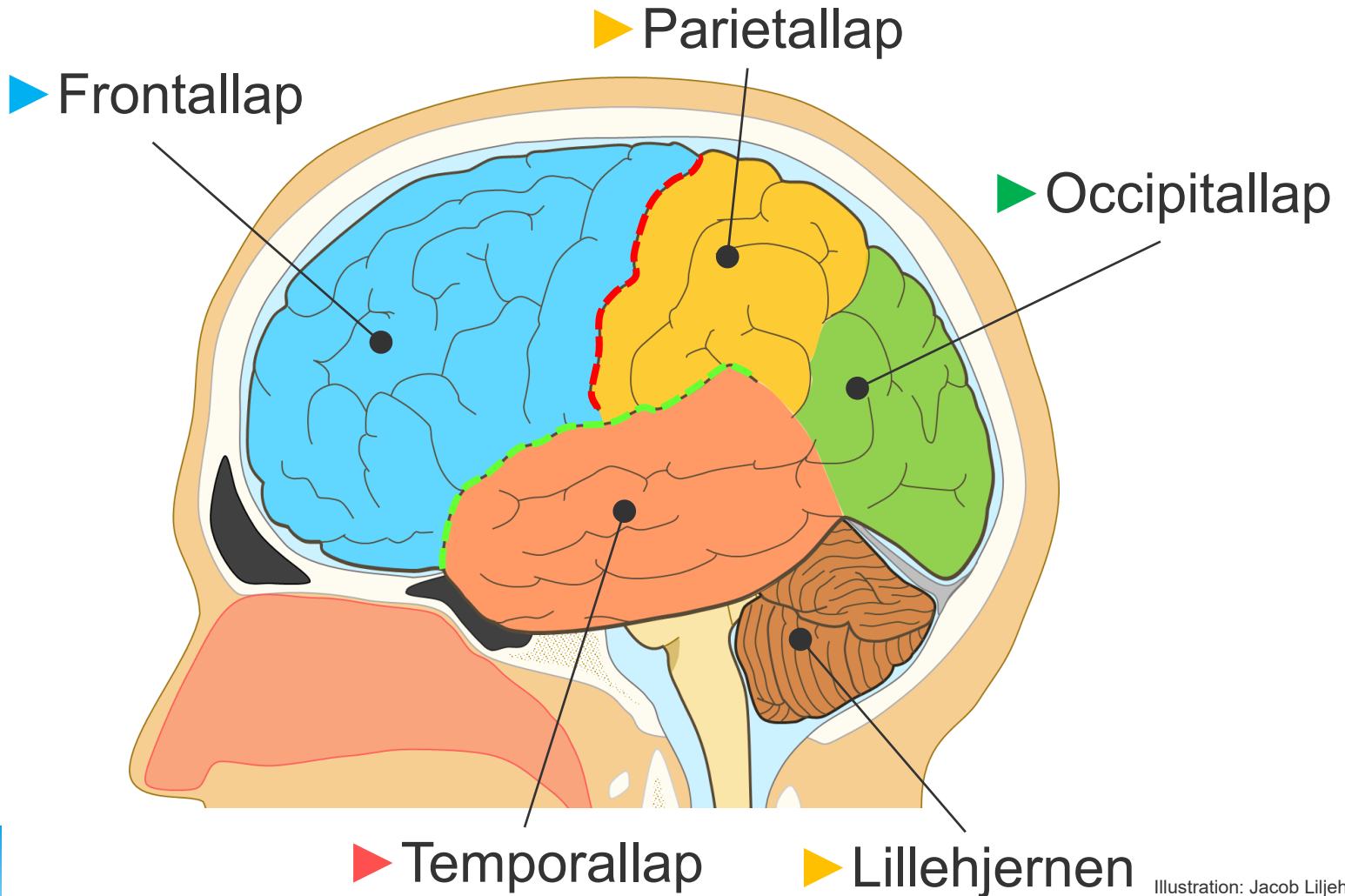
Knud 69 år

Knud indlægges via egen læge mandag formiddag, fordi hans kone syntes han havde været mere klodset hele weekenden. Han har tabt ting og gået ind i møblerne. Lægen finder lammelse af venstre side og neglect mod venstre. Knud synes ikke rigtigt der er noget galt og vil helst bare hjem.

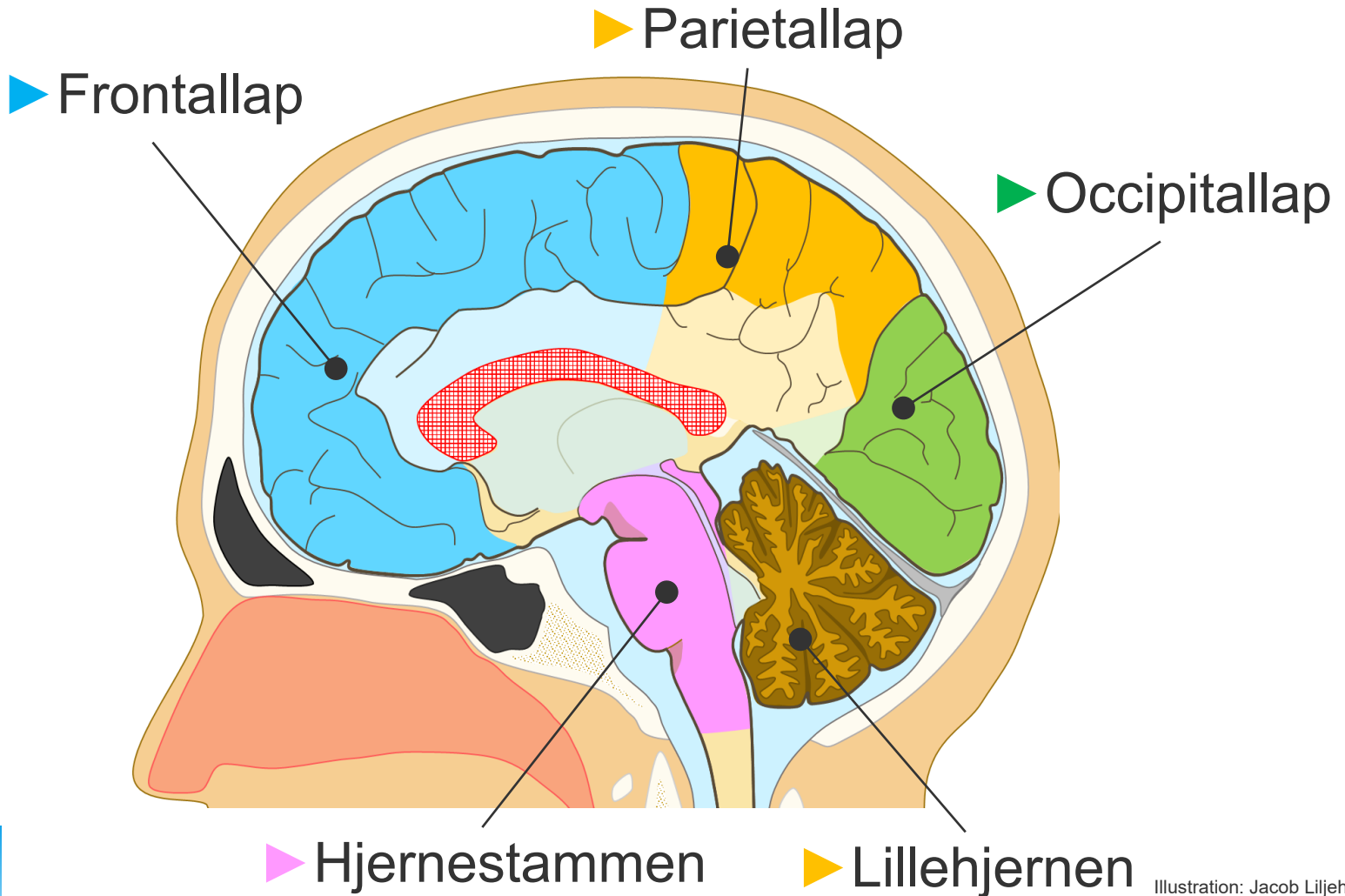
Lateraliserede udfald



Hjernens overflade



Hjernens overflade



Dybe strukturer

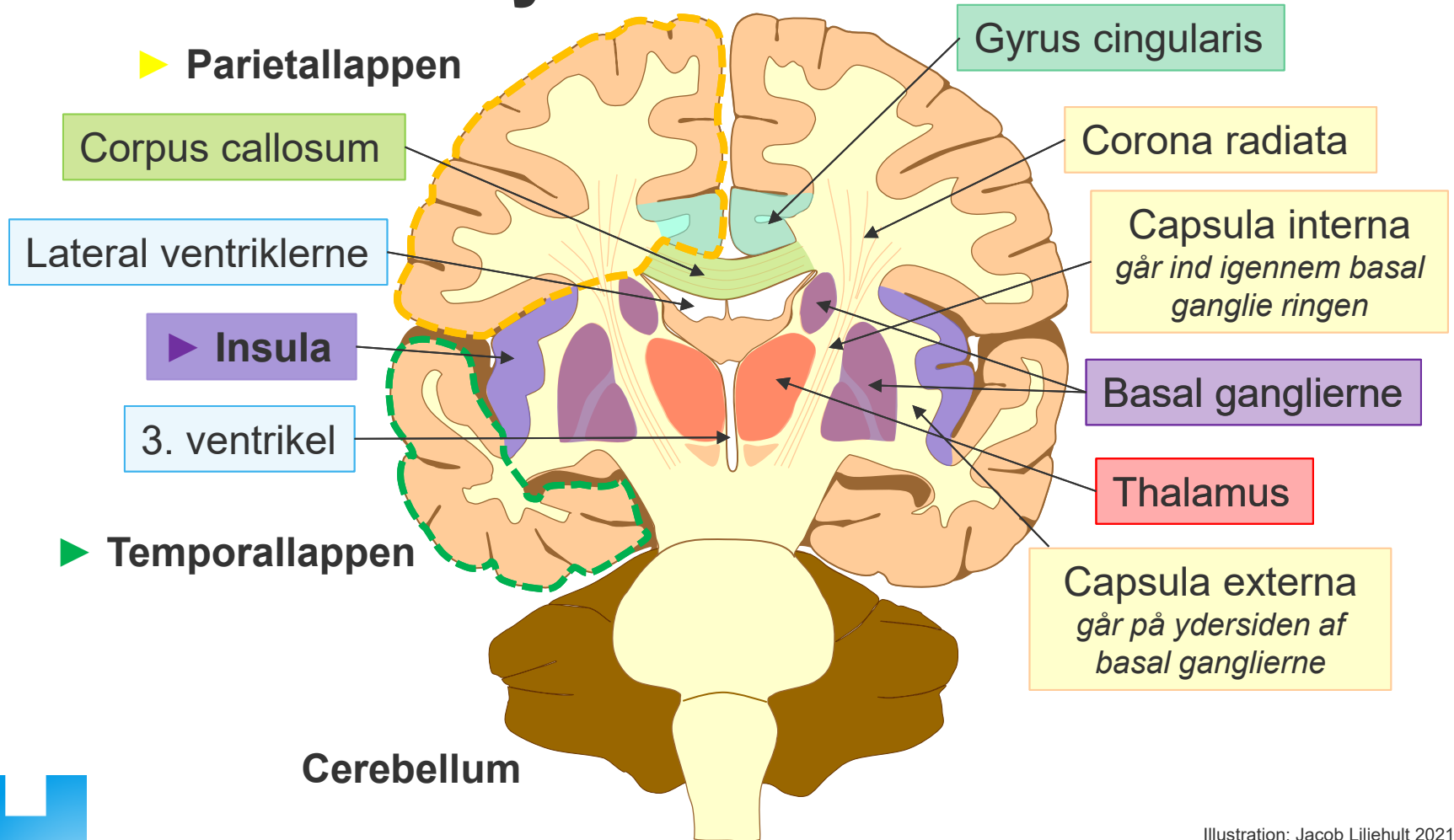


Illustration: Jacob Liljehult 2021

Eksekution

Hvad gør jeg?
Hvordan gør jeg det?

Perception

Hvor er jeg?
Hvad foregår der?

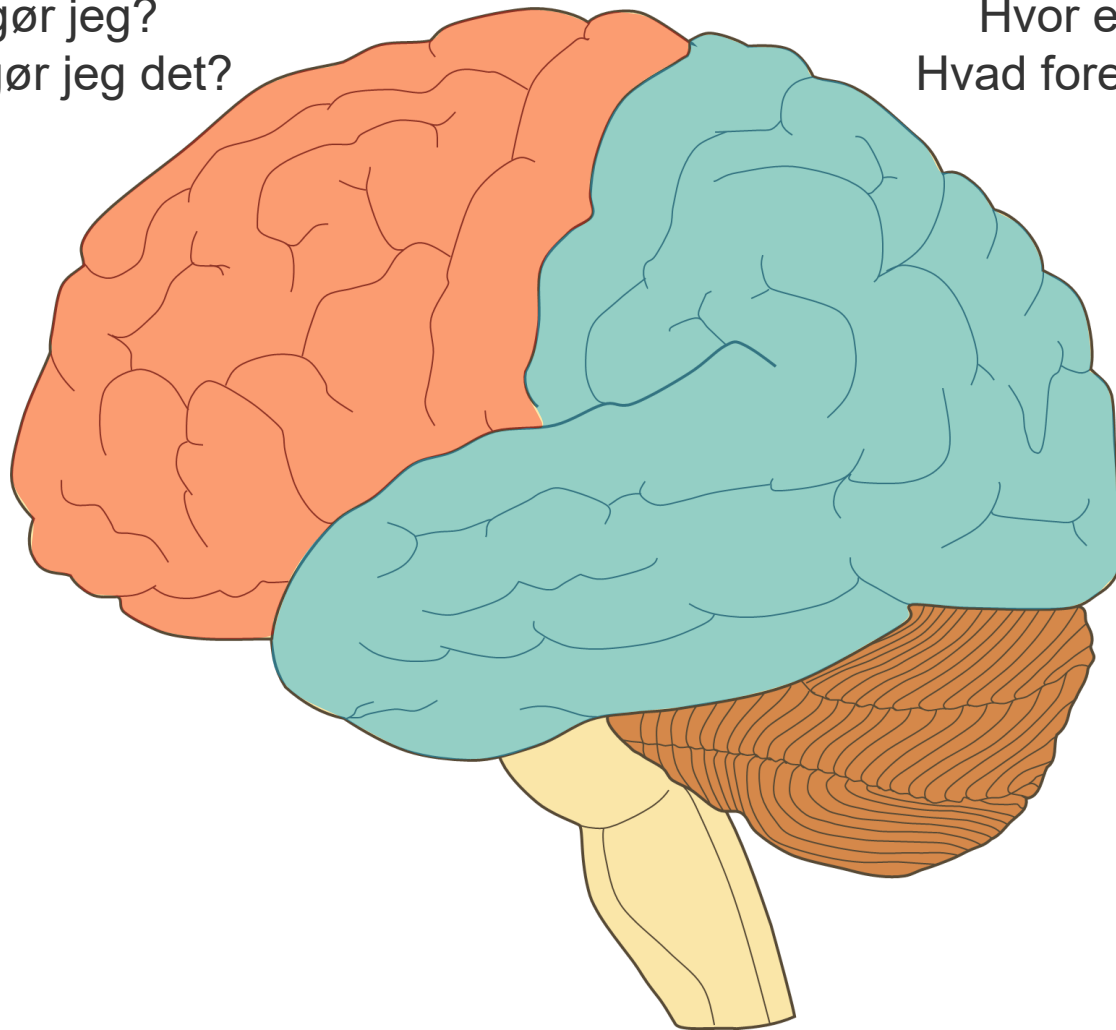
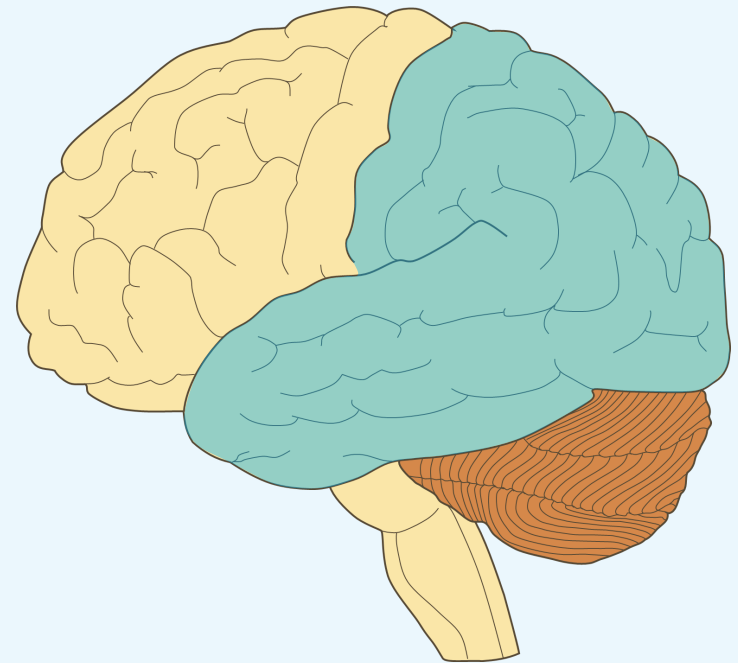


Illustration: Jacob Liljehult 2022

Sansning og perception

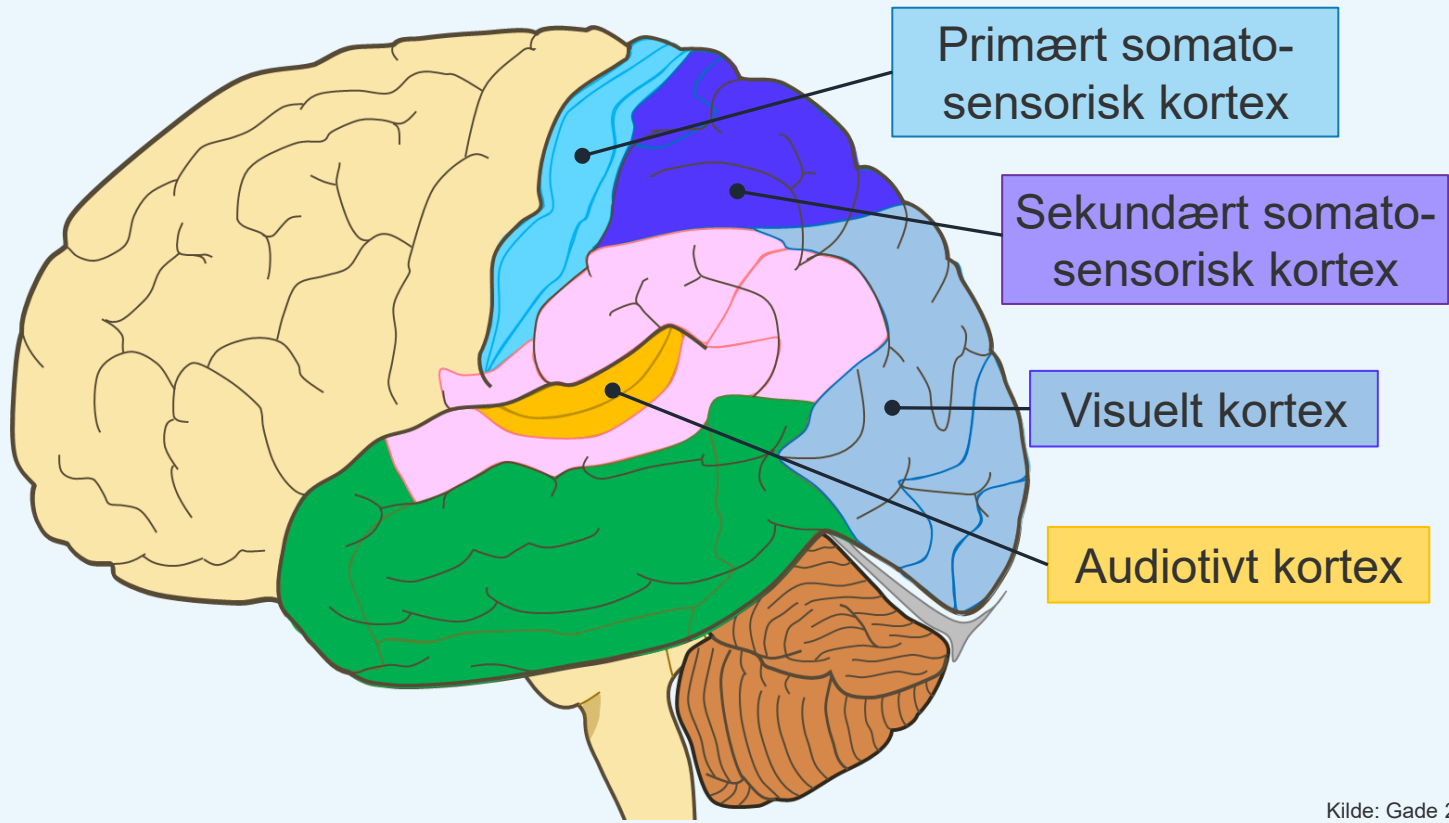
Sanser

- **Specialiserede sanser**
 - Lugt
 - Smag
 - **Syn**
 - Hørelse
 - **Balance**
- **Somato-sensoriske sanser**
 - Berøring
 - Smerte/temperatur
 - Proprioception



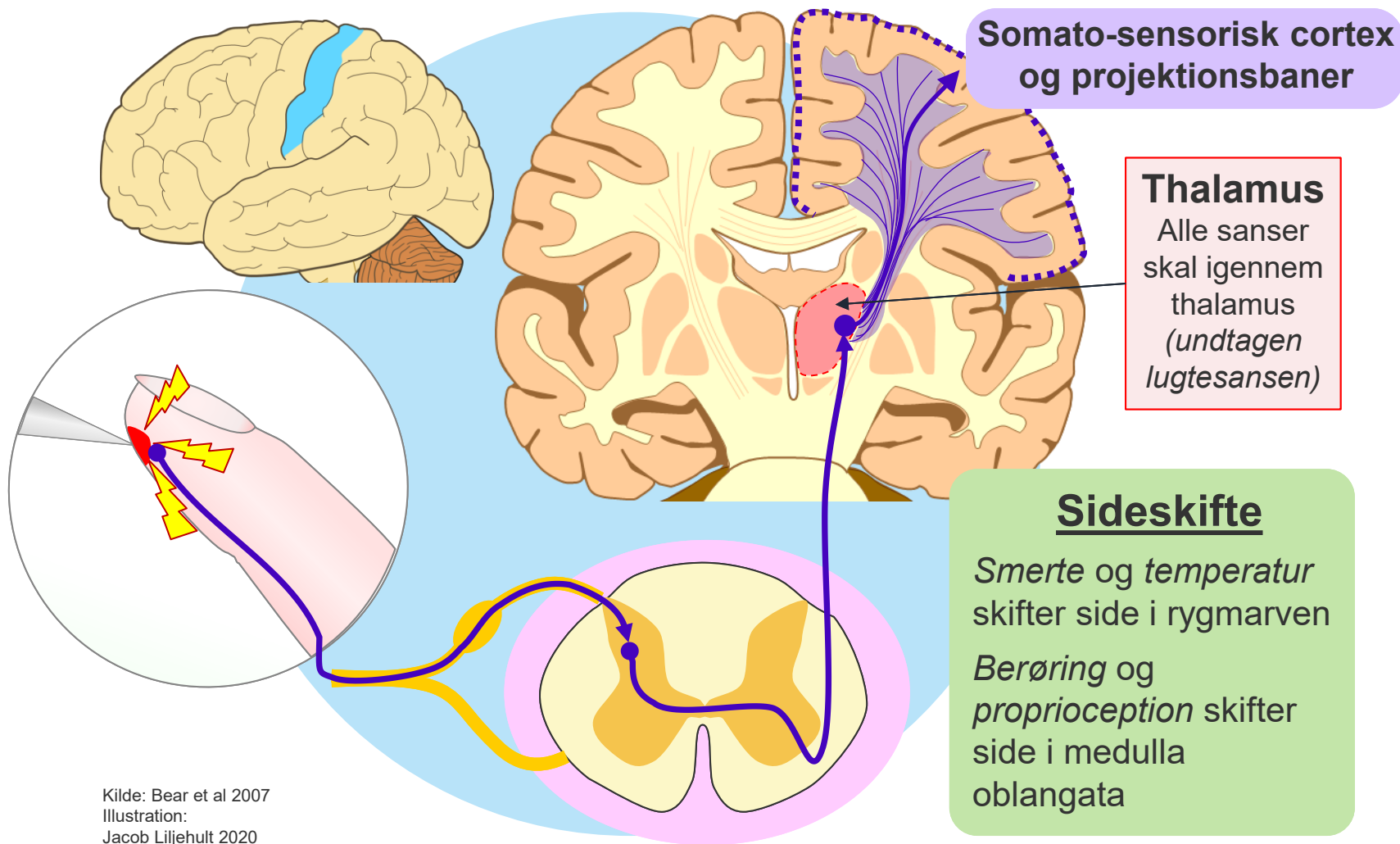
Kilde: Gade 2009
Illustration: Jacob Liljehult 2020

Perception



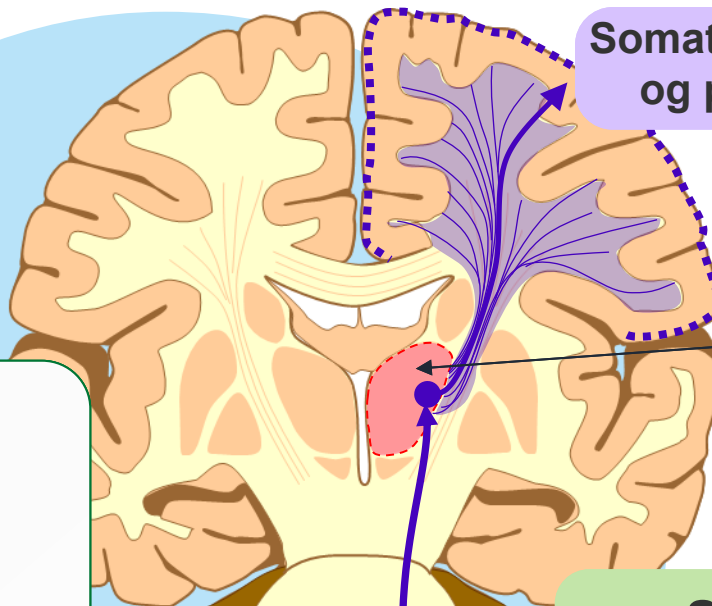
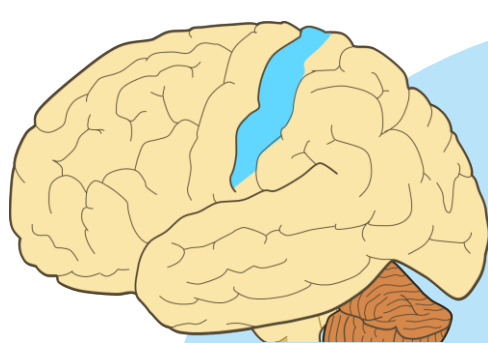
Kilde: Gade 2009
Illustration: Jacob Liljehult 2020

De somato-sensoriske baner



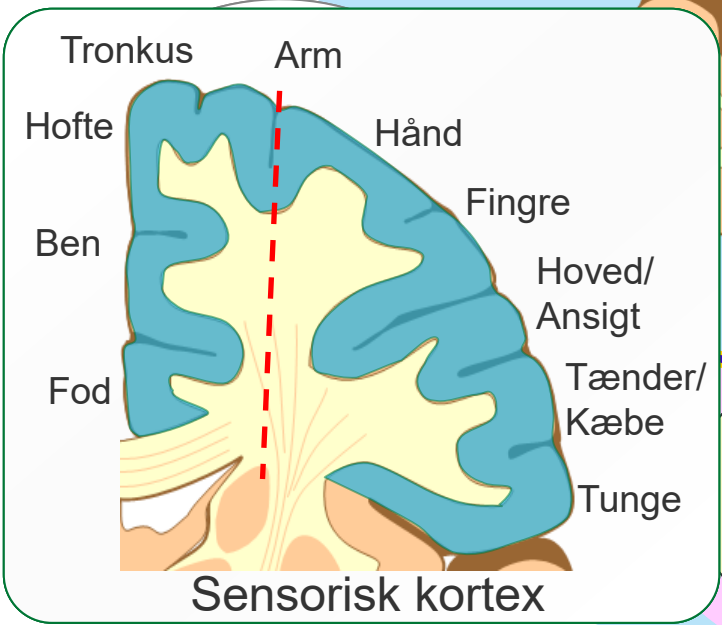
Kilde: Bear et al 2007
Illustration:
Jacob Liljehult 2020

De somato-sensoriske baner



Somato-sensorisk cortex og projektionsbaner

Thalamus
Alle sanser skal igennem thalamus (undtagen lugtesansen)



Sideskifte
Smerte og temperatur skifter side i rygmarven
Berøring og proprioception skifter side i medulla oblongata

Illustration:
Jacob Liljehult 2020

Synsbanerne

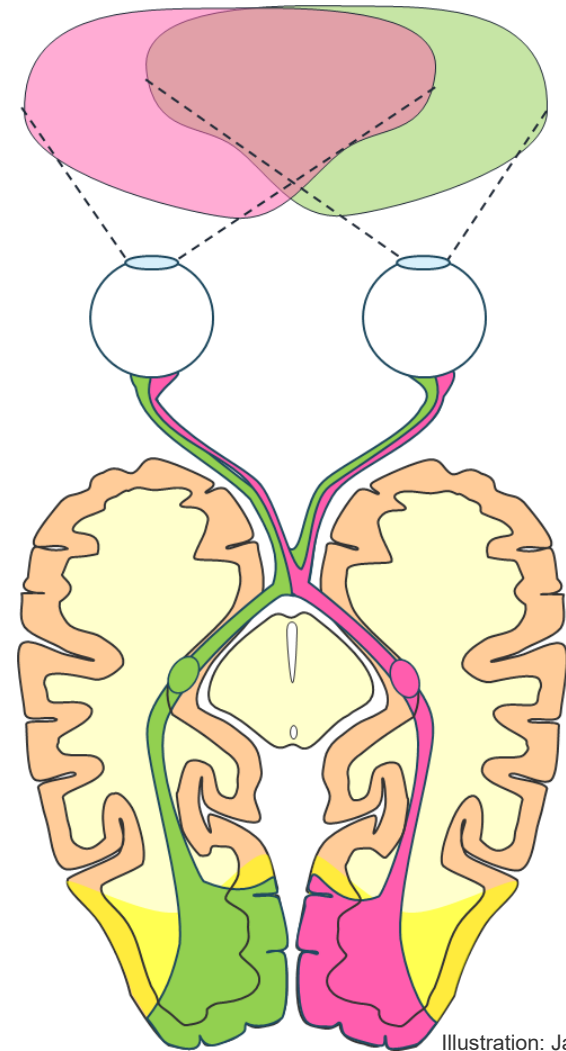
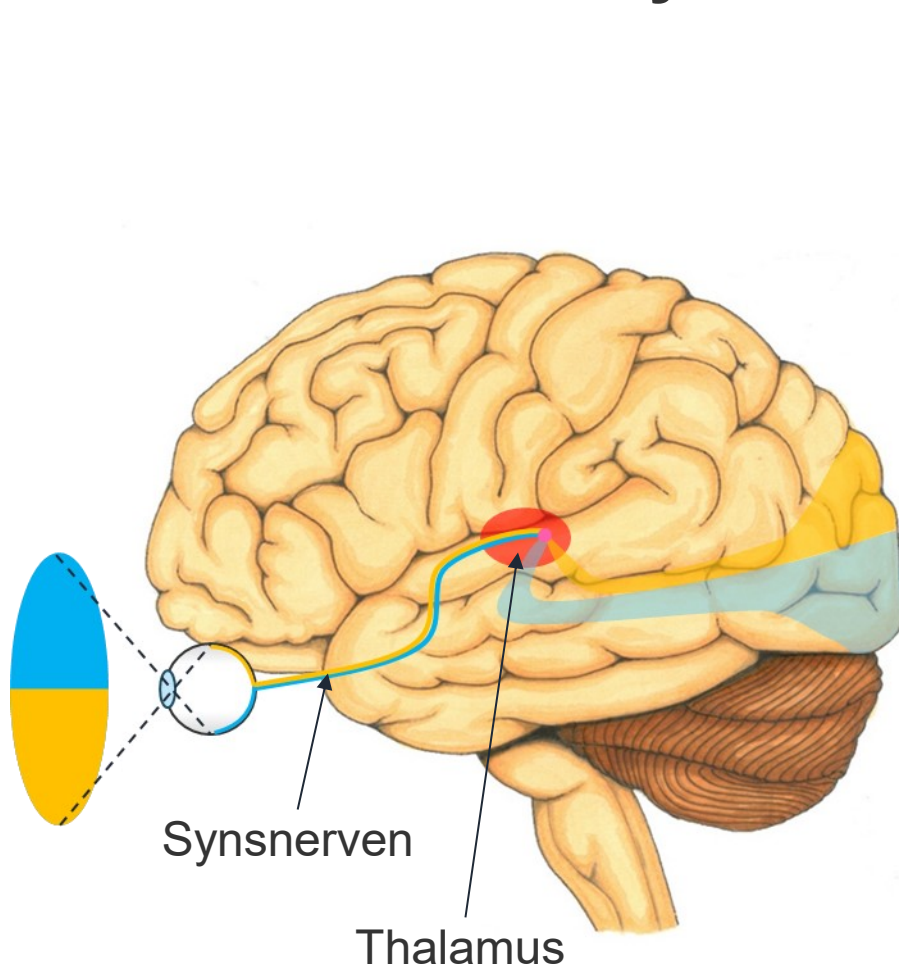


Illustration: Jacob Liljehult 2021

Visuelle forstyrrelser

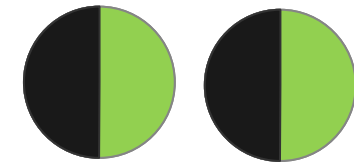
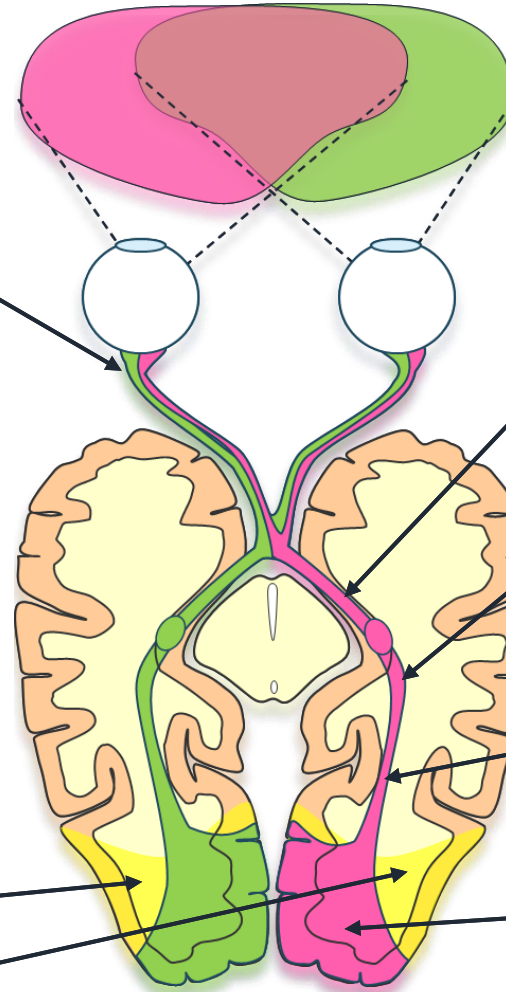
Ved apopleksi er
anopsi altid i
modsatte side

Amaurosis fugax

Hvis begge
occipitallapper
er ramt

Kortikal blindhed +
anosognosi
(Anton's syndrom)

Agnosi
Manglende evne til at
genkende ting
(Oftest bilaterale læsioner)



hemianopsi



Øvre kvadrant
anopsi



Nedre kvadrant
anopsi



hemianopsi

Illustration: Jacob Liljehult 2021

Visuo-spartielle forstyrrelser

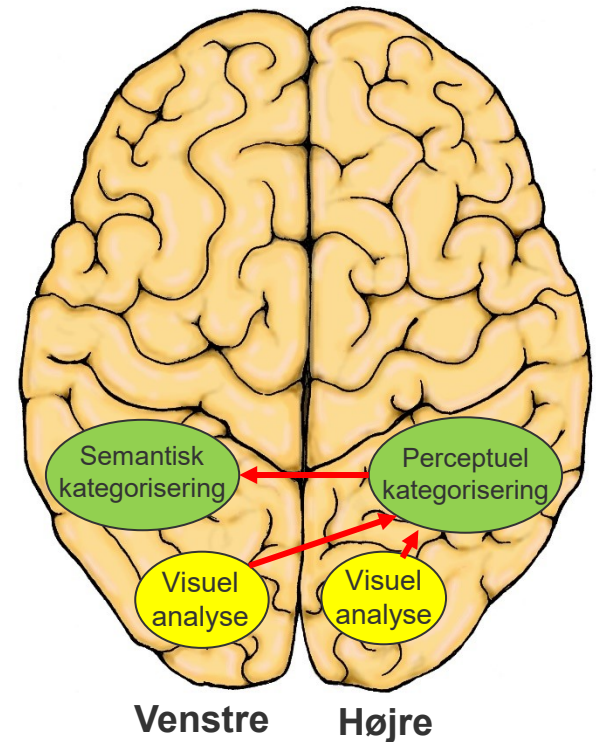
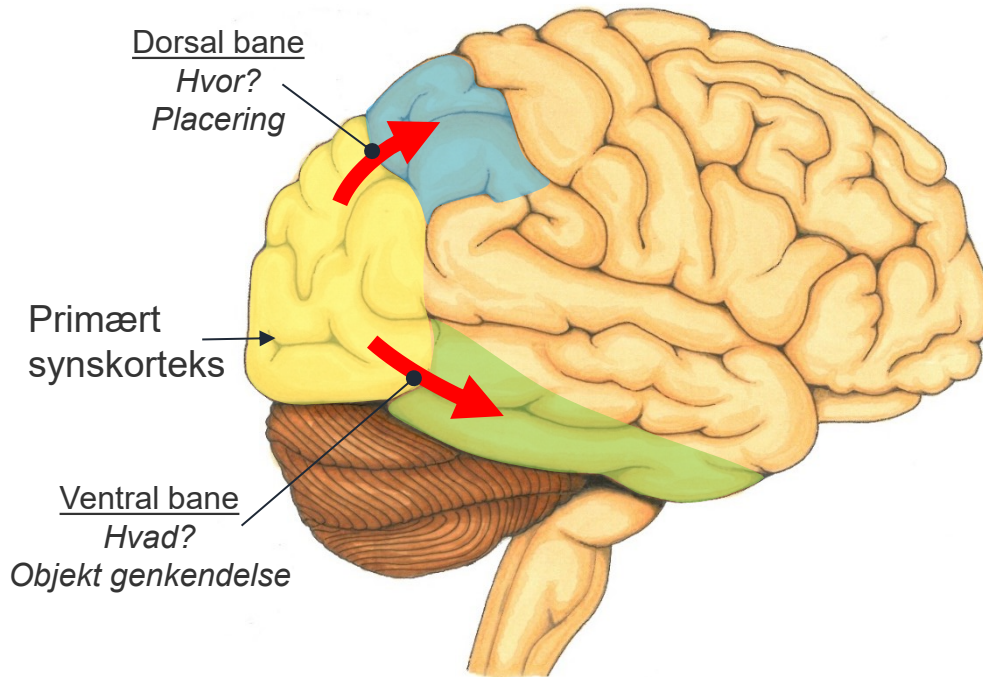


Illustration: Jacob Liljehult 2021

Visuo-spartielle forstyrrelser

Visuel agnosi

- *Problemer med at genkende/identificere genstande visuelt*
- *Højre → Apperceptiv agnosi*
 - *Manglende genkendelse af genstande; kan ofte beskrive detaljer, men ikke helhed*
 - *Påvirker ofte evnen til at genkende ansigter*
- *Venstre → Associativ agnosi*
 - *kan ikke identificere genstande eller deres funktion*

Simultan-agnosi

- *Kan kun identificere én ting ad gangen*

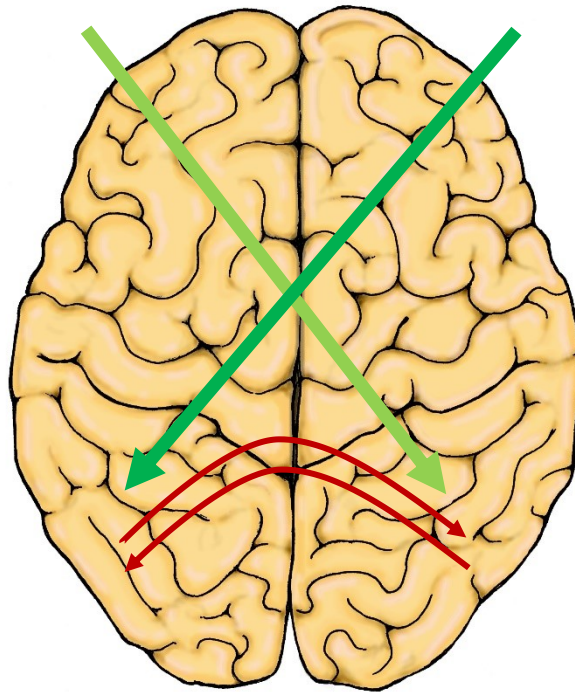
Visuo-spartiel disorientering

- *Problemer med at vurdere genstandes placering i rummet*

Topografisk amnesi/agnosi

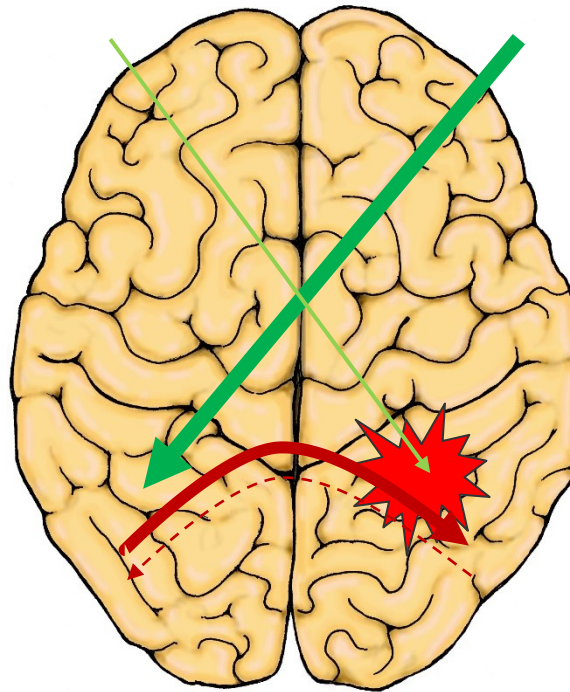
- *Problemer med at huske/genkende kendte omgivelser*

Neglekt og inattention



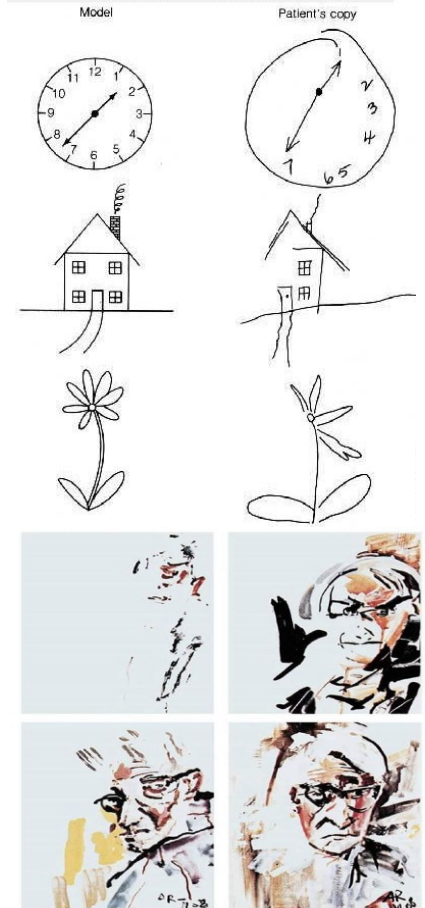
Normal hjerne

Illustration: Jacob Liljehult 2020
Kilde Gazzaniga et al 2009



Læsion i højre
parietallap

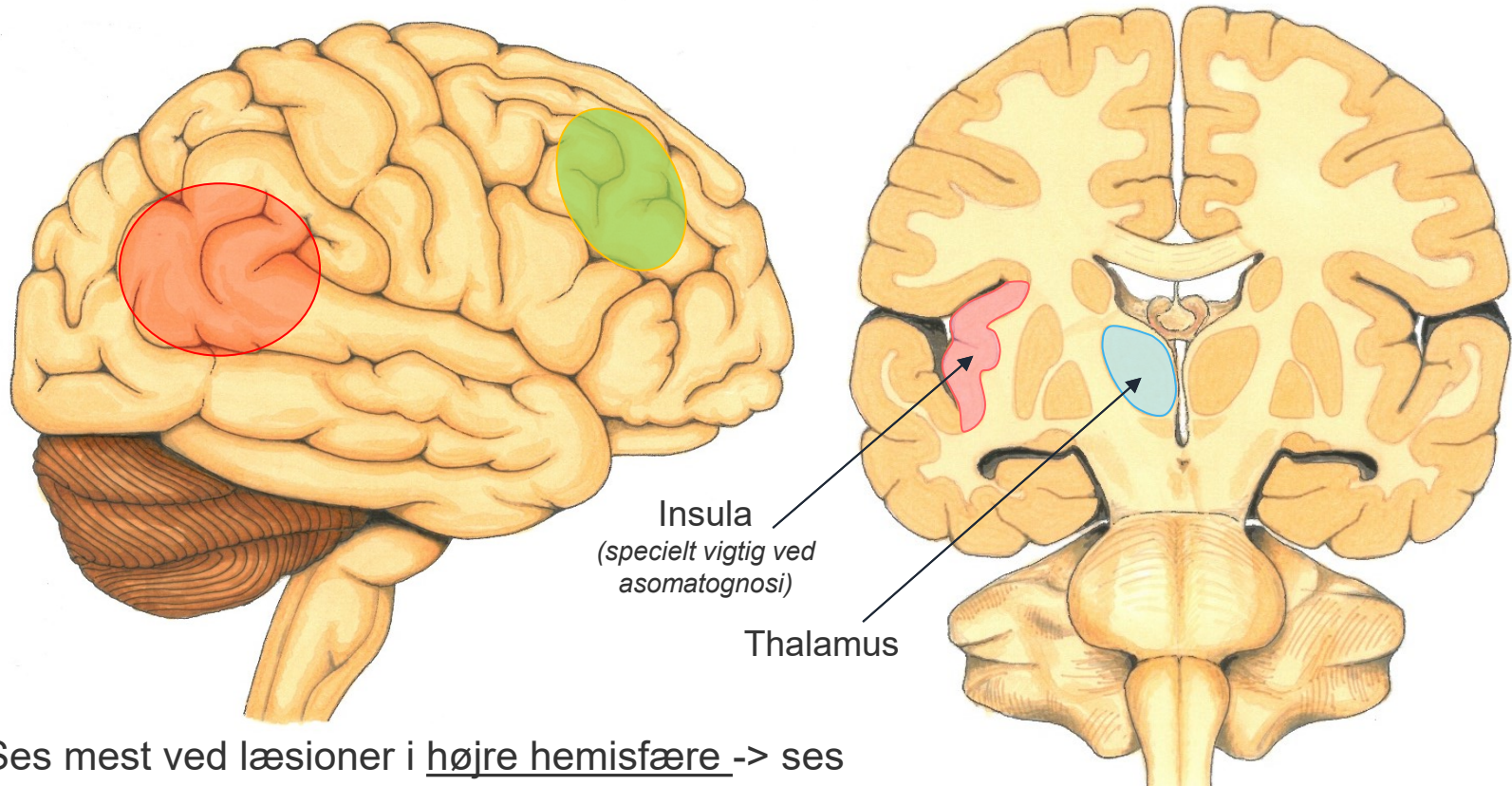
Figure 13. Drawings by a patient with left-side neglect.



Hvor er gafflen?



Anosognosi



Ses mest ved læsioner i højre hemisfære -> ses derfor ofte sammen med venstresidig hemiparese

Anosognosi

Manglende sygdoms- eller symptomerkendelse

Kategorier

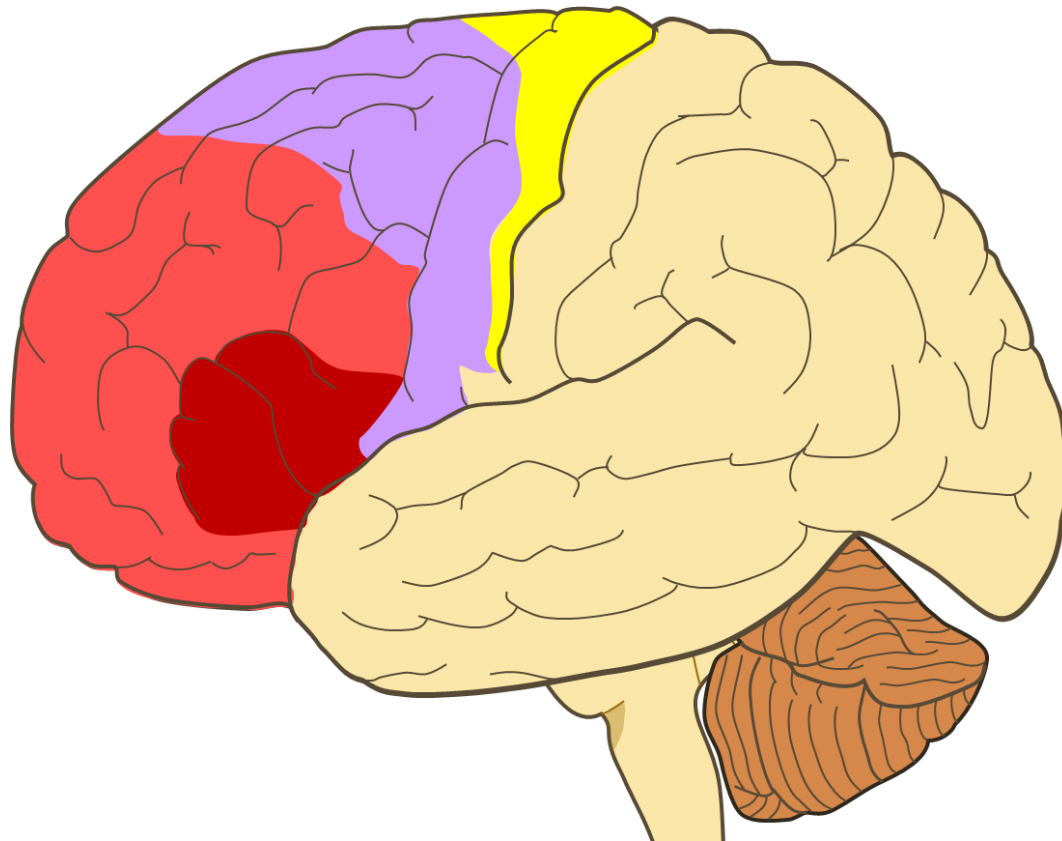
- Global anosognosi: Benægter generelt at være syg (hyppigt ved hovedtraumer og demens)
- Lokal anosognosi: Benægter et specifikt symptom – ofte hemiparese eller hemianopsi (hyppigt ved apopleksi)
- Asomatognosi: Vedkender sig ikke ejerskab over lammet ekstremitet
”Der ligger et fremmed ben i min seng” (hyppigt ved apopleksi)

Karakteristika

- Fortæller ikke spontant om symptomer
- Benægter eksplicit symptomerne
- Konfabulerer om årsagen til symptomerne (udenoms forklaringer)
- Tager ikke højde for symptomerne i sin adfærd
- Reagerer ikke følelsesmæssigt/ virker ligeglad (*anosodiafori*)
- Tager ikke højde for symptomerne i sine planer for fremtiden (urealistisk om fremtiden)

Eksikution

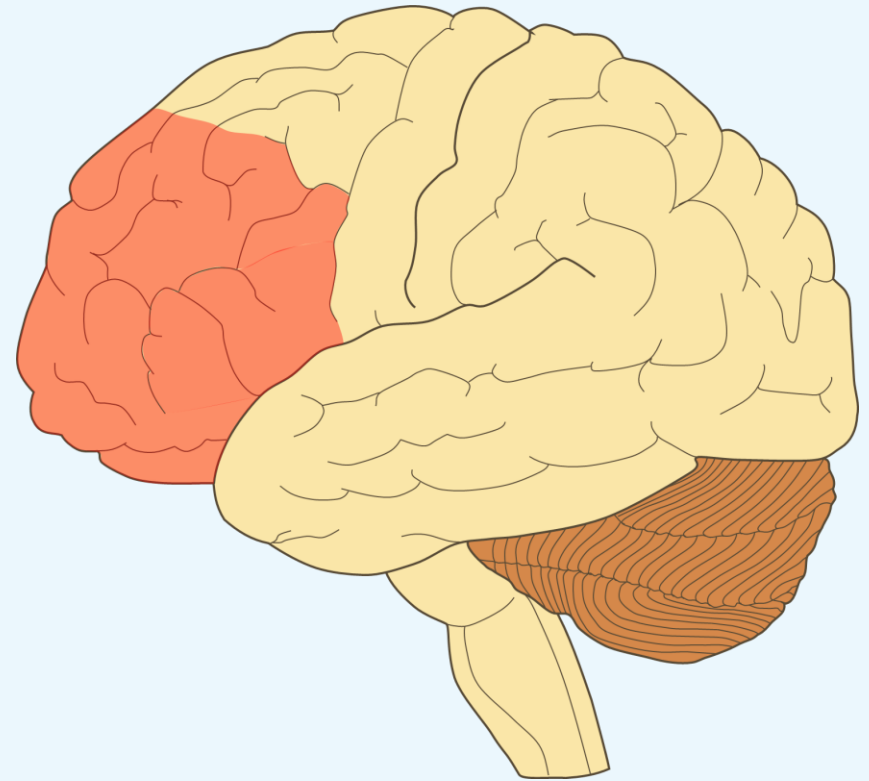
fra plan til handling



Præfrontale funktioner

Beslutninger om hvad man skal gøre og hvordan

- Apati, rigiditet, springende opmærksomhed
- Perseveration og stereotyp adfærd
- Manglende hæmning
- Nedsat dømmekraft, selvindsigt
- Ændret personlighed



Kilde: Gade 2009
Illustration: Jacob Liljehult 2020

Præfrontale funktioner

Dorso-lateralt

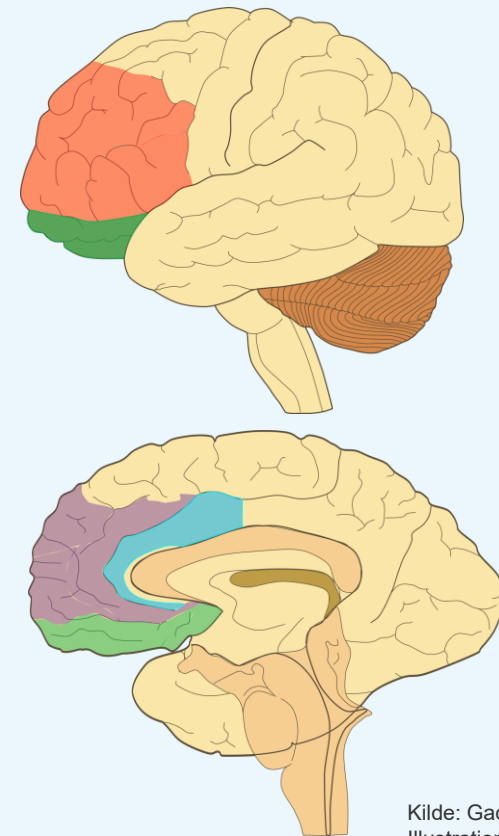
Nedsat energi, initiativløshed, nedsat evne til planlægning, ligegyldig

Orbitalt

Opstemt, rastløs, mangler hæmninger, ansvarsløse handlinger, hensynsløs, selvoptaget

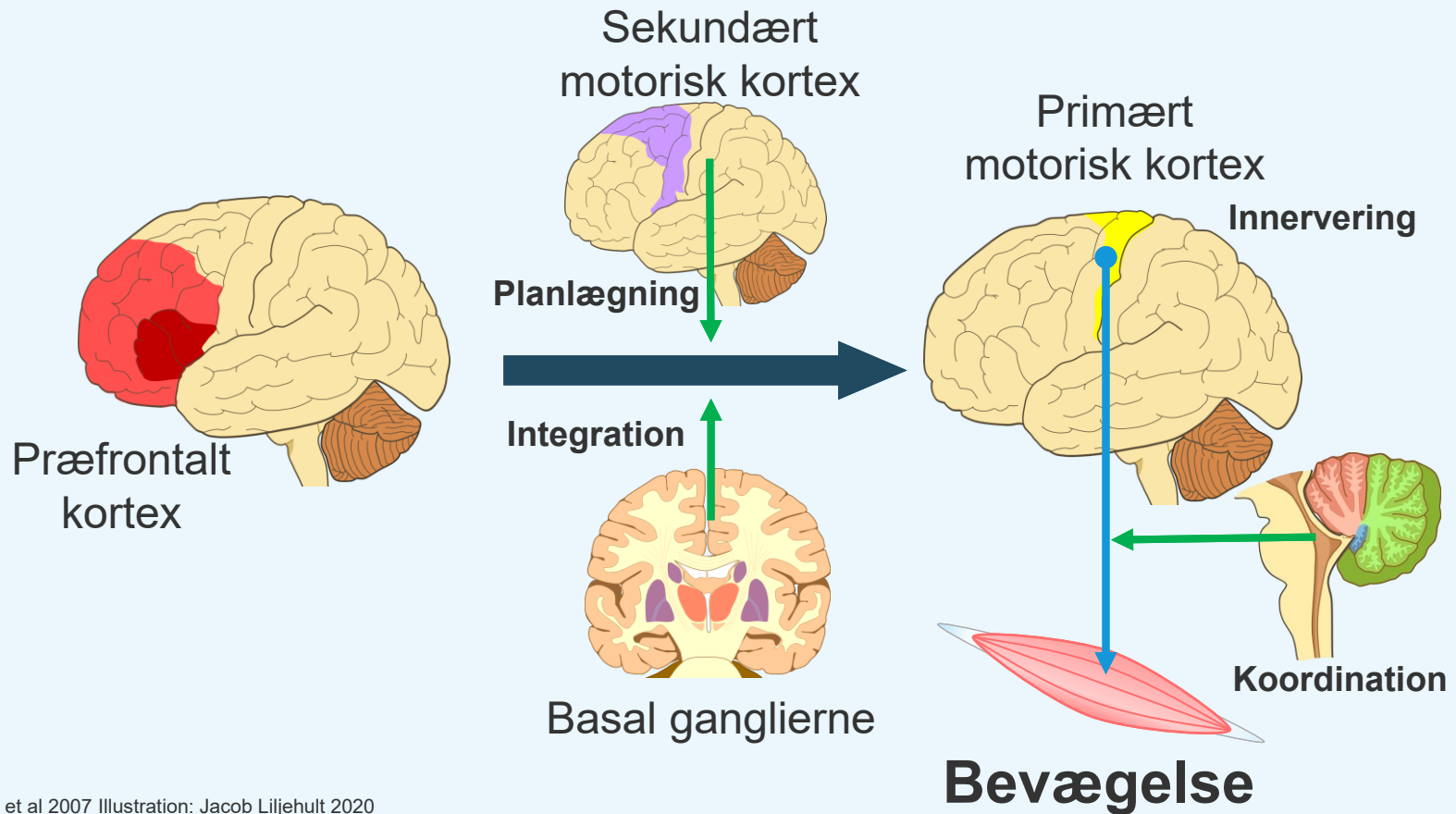
Medialt

Nedsat opmærksomhed, nedsat bevidsthed, desorienteret, konfabulerende



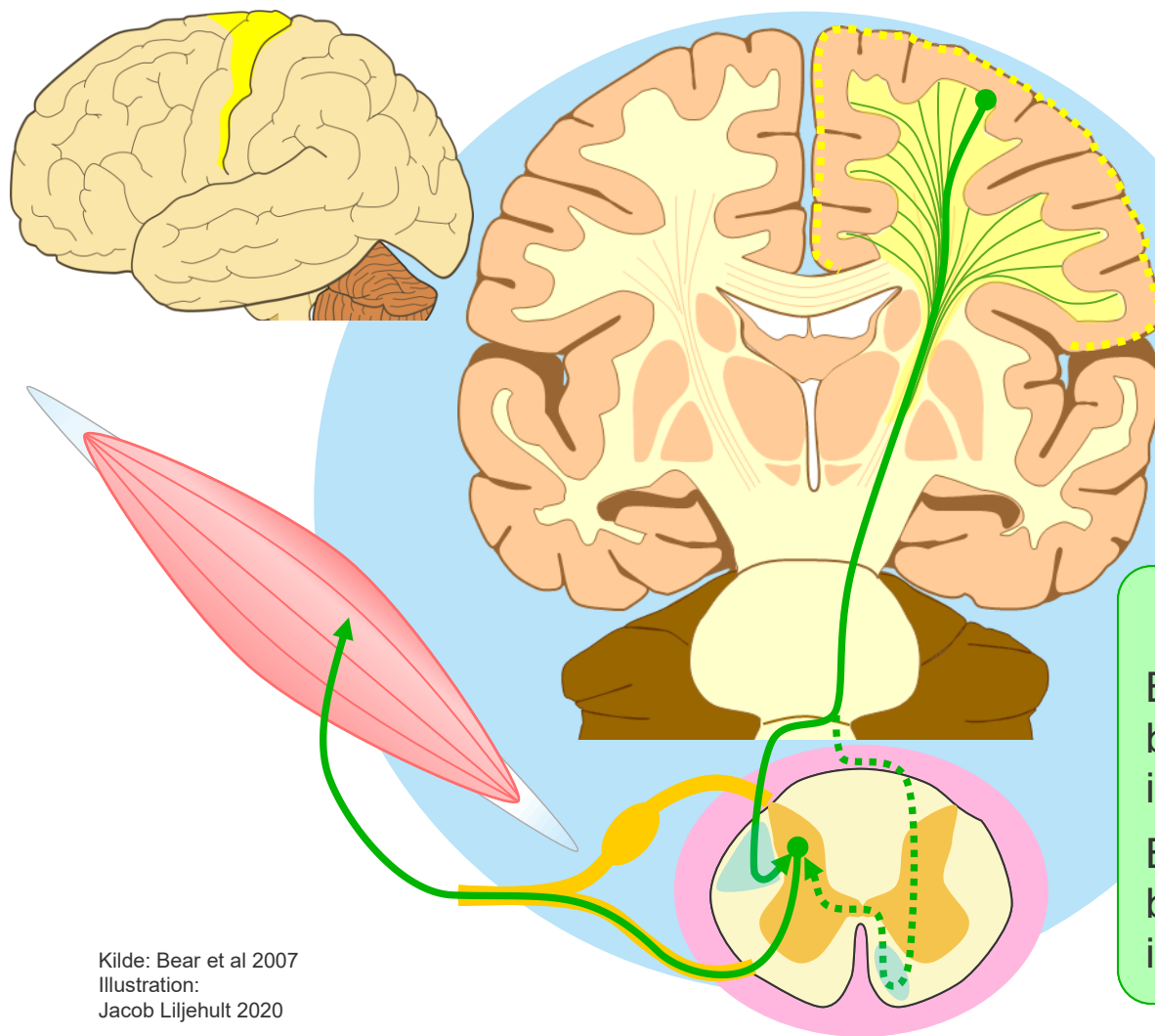
Kilde: Gade 2009
Illustration: Jacob Liljehult 2020

Det motorisk system



Kilde: Bear et al 2007 Illustration: Jacob Liljehult 2020

De motoriske baner



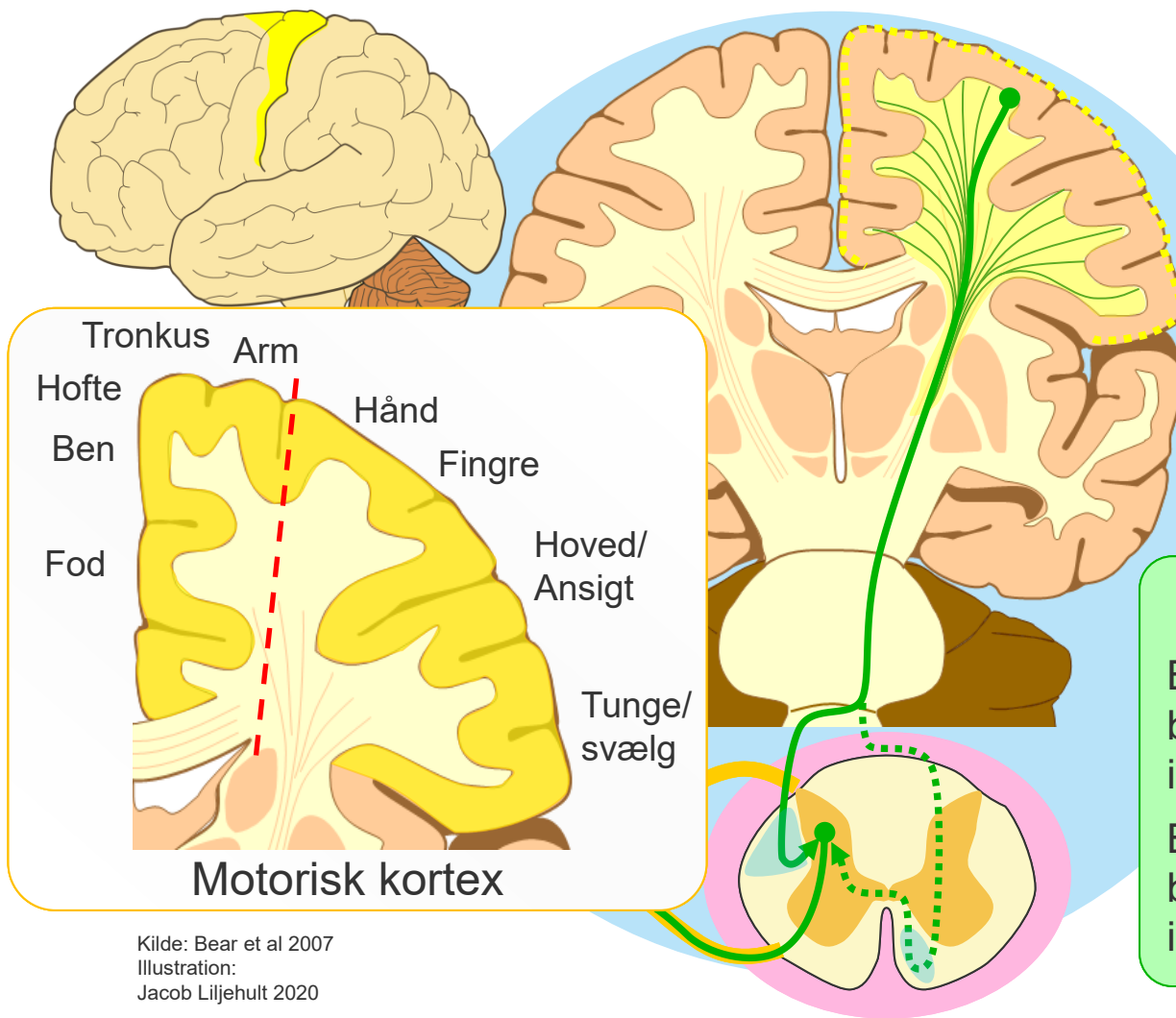
Motorisk cortex og
projektionsbaner

Sideskifte
 Baner for *voluntære*
 bevægelser skifter side
 i medulla oblongata
 Baner for *involuntære*
 bevægelser skifter side
 i rygmarven

Kilde: Bear et al 2007
 Illustration:
 Jacob Liljehult 2020

De motoriske baner

Motorisk cortex og
projektionsbaner



Sideskifte

Baner for *voluntære* bevægelser skifter side i medulla oblongata

Baner for *involuntære* bevægelser skifter side i rygmarven

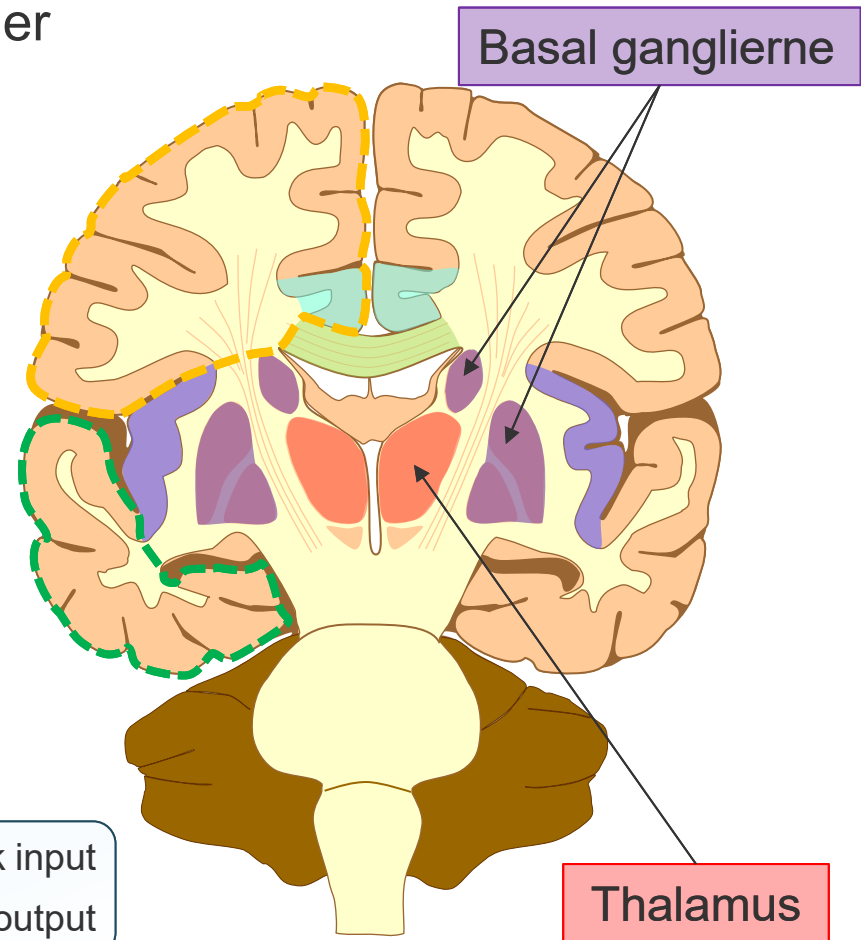
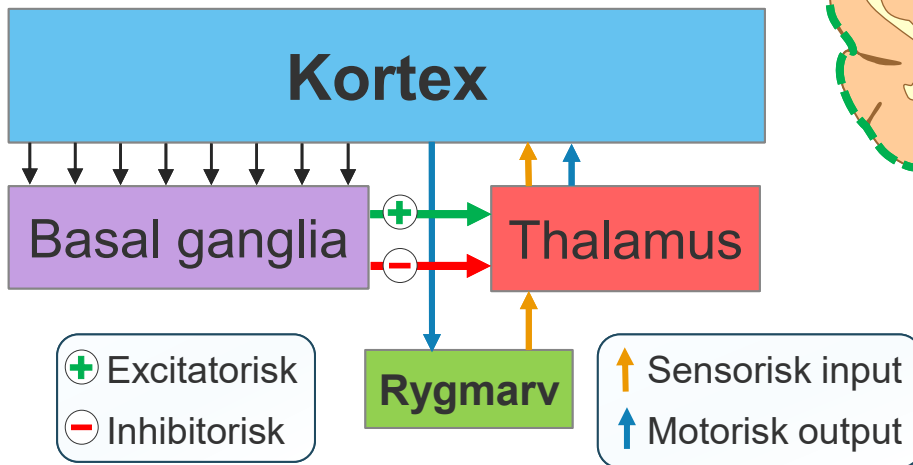
Kilde: Bear et al 2007
Illustration:
Jacob Liljehult 2020

Basal ganglierne

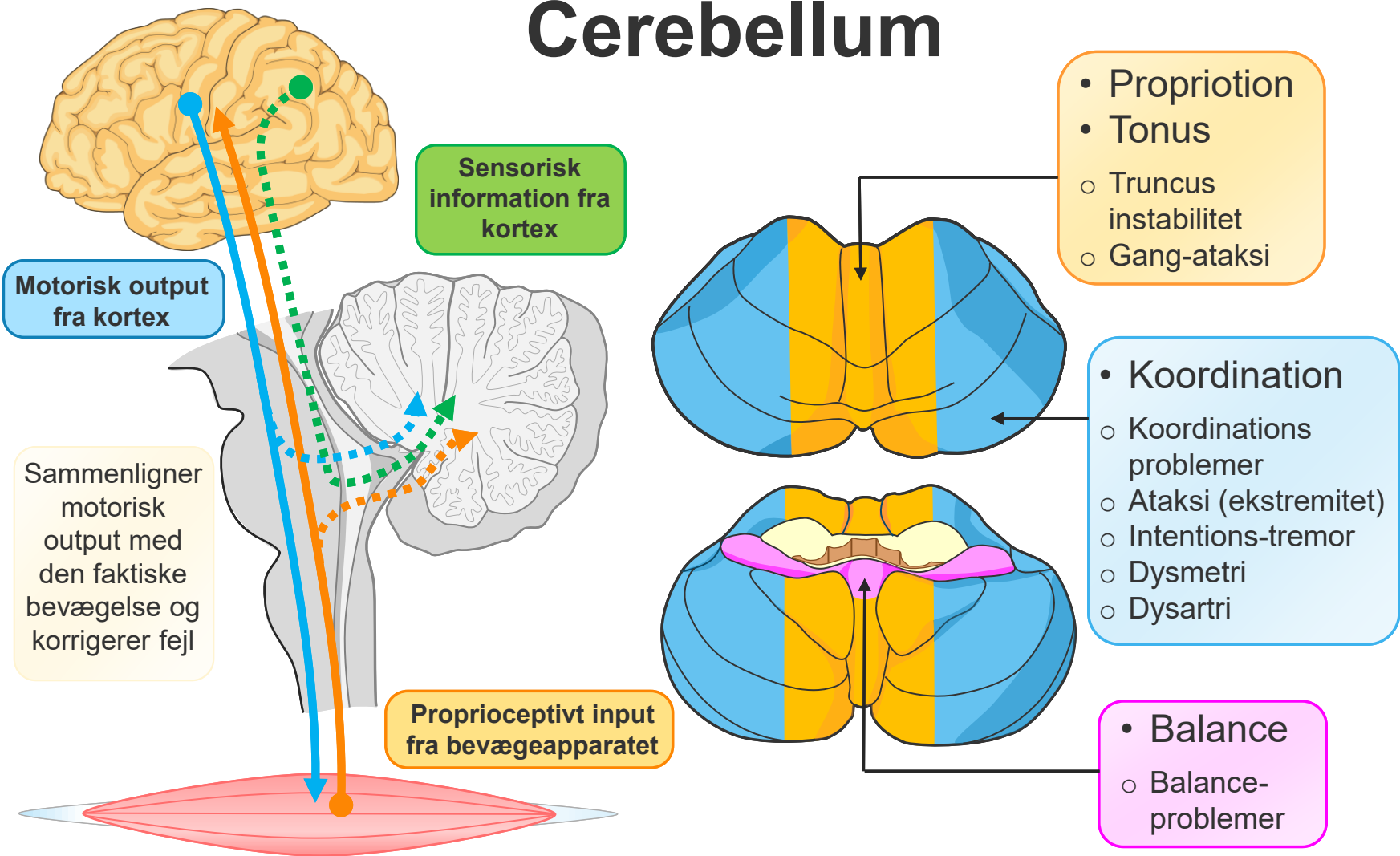
Tager input fra kortex og samordner det til et samlet adfærds-output

Tre kredsløb:

1. Integration af motorisk output
2. Læring og kognition
3. Regulering af emotionelle aspekter af adfærd



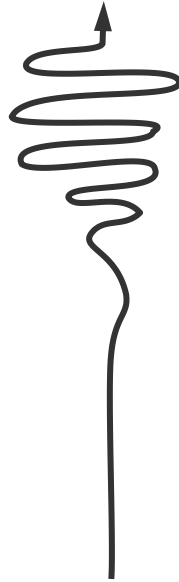
Cerebellum



Normal bevægelse



Cerebellar ataksi



Tremor forværres når
fingeren nærmer sig
målet

Parkinson Sygdom



Tremor i starten af
bevægelsen, men
flader ud

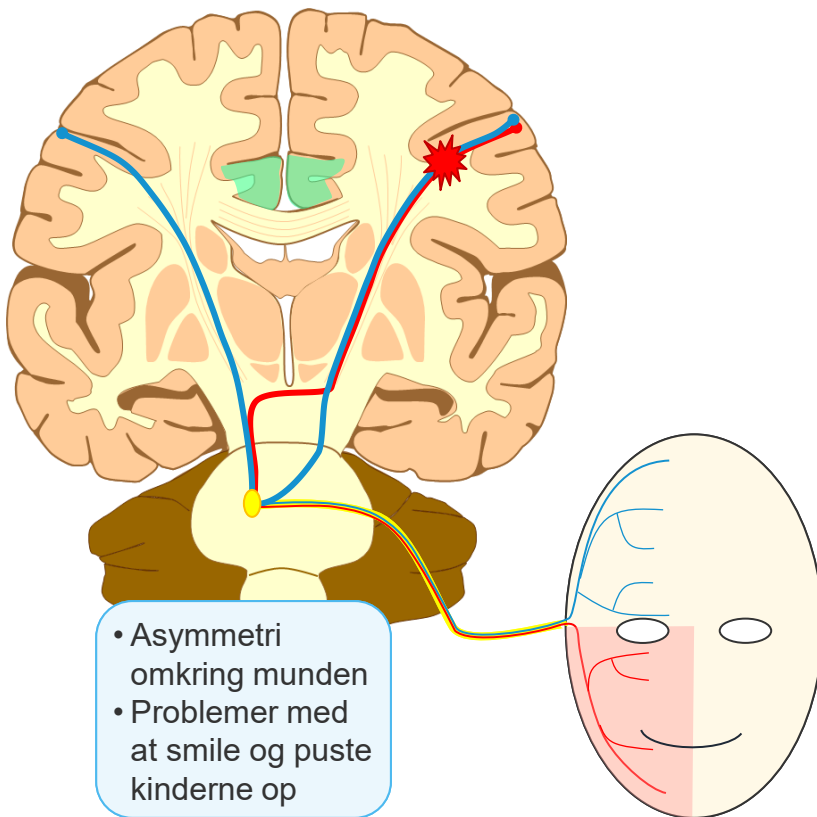
Essentiel tremor



Konstant tremor

Faciales parese

Central
(Supranuklear)



Perifer
(Infranuklear)

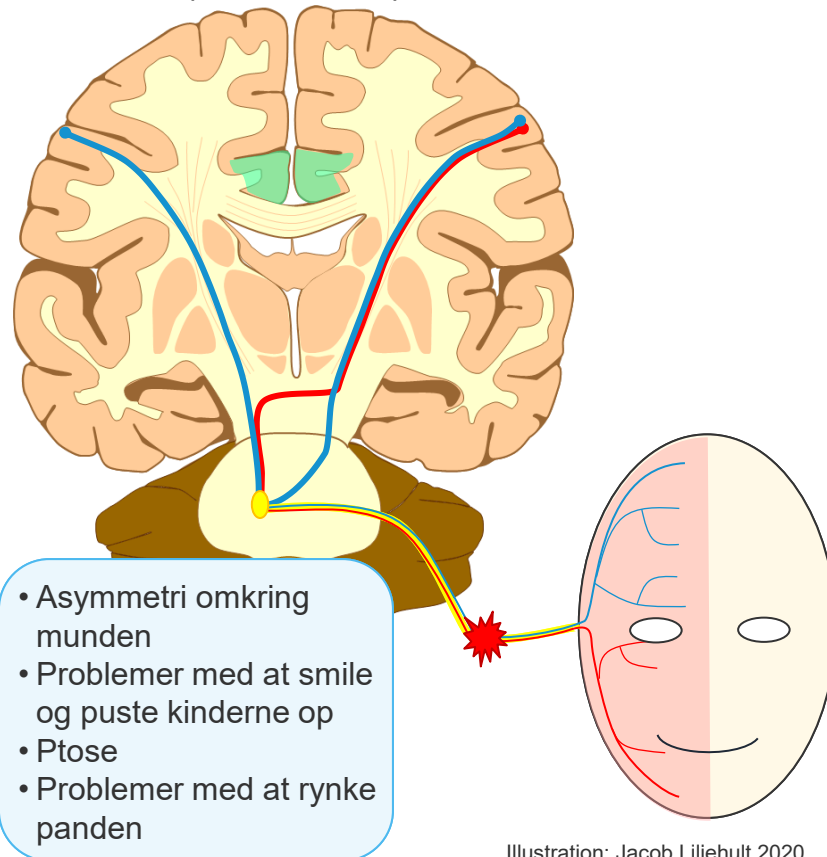


Illustration: Jacob Liljehult 2020

Apraksi

Generelt: Latenstid, tøvende og klodset, perseveration

Ideomotorisk

- Problemer med at lave de rigtige bevægelser der er knyttet til en handling
 - *Patienten kan ikke finde ud af at vinke på opfordring*

Ideatorisk

- Problemer med sekvensen af delelementer i en aktivitet
 - *Patienten begynder at bruse sig før han har taget tøjet af*

Konceptuel

- Problemer med at udvælge de rigtige genstande til handlingen
 - *Patienten prøver at børste tænder med tandpastatuben*

Ideomotorisk apraksi



<https://youtu.be/vTFdNk7Jloo>

Mund og svælg

Tungedeviation



Ganesejlsparese



Dysfagi (Synkeproblemer)

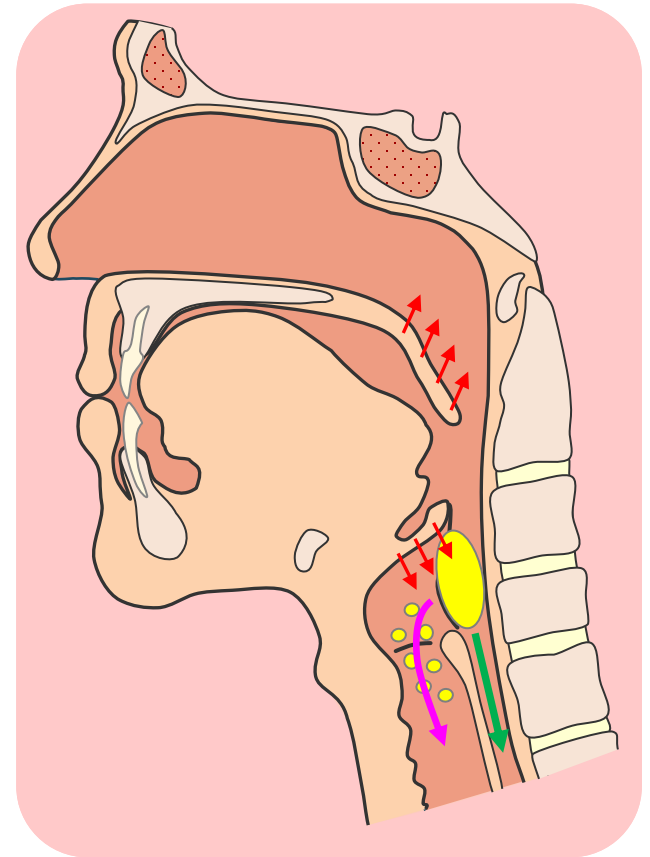
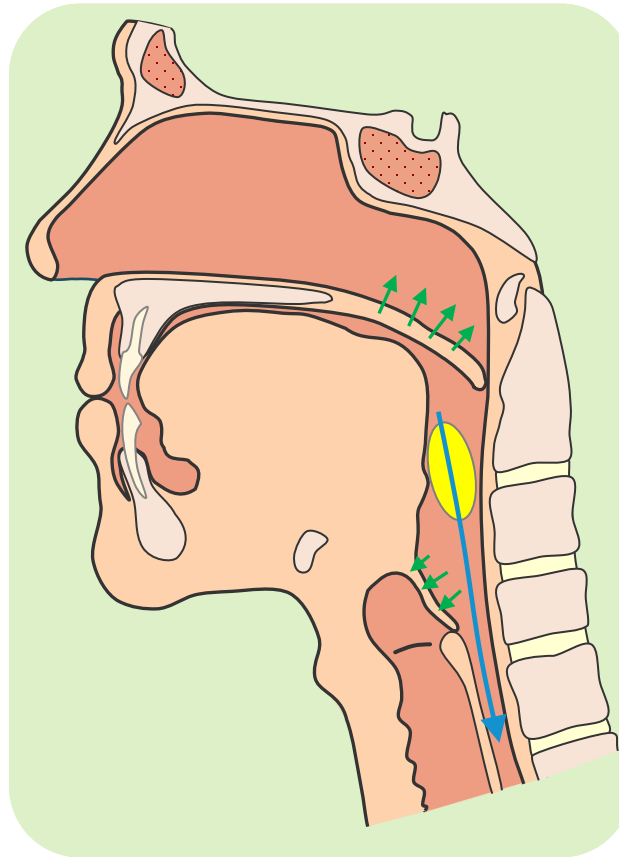
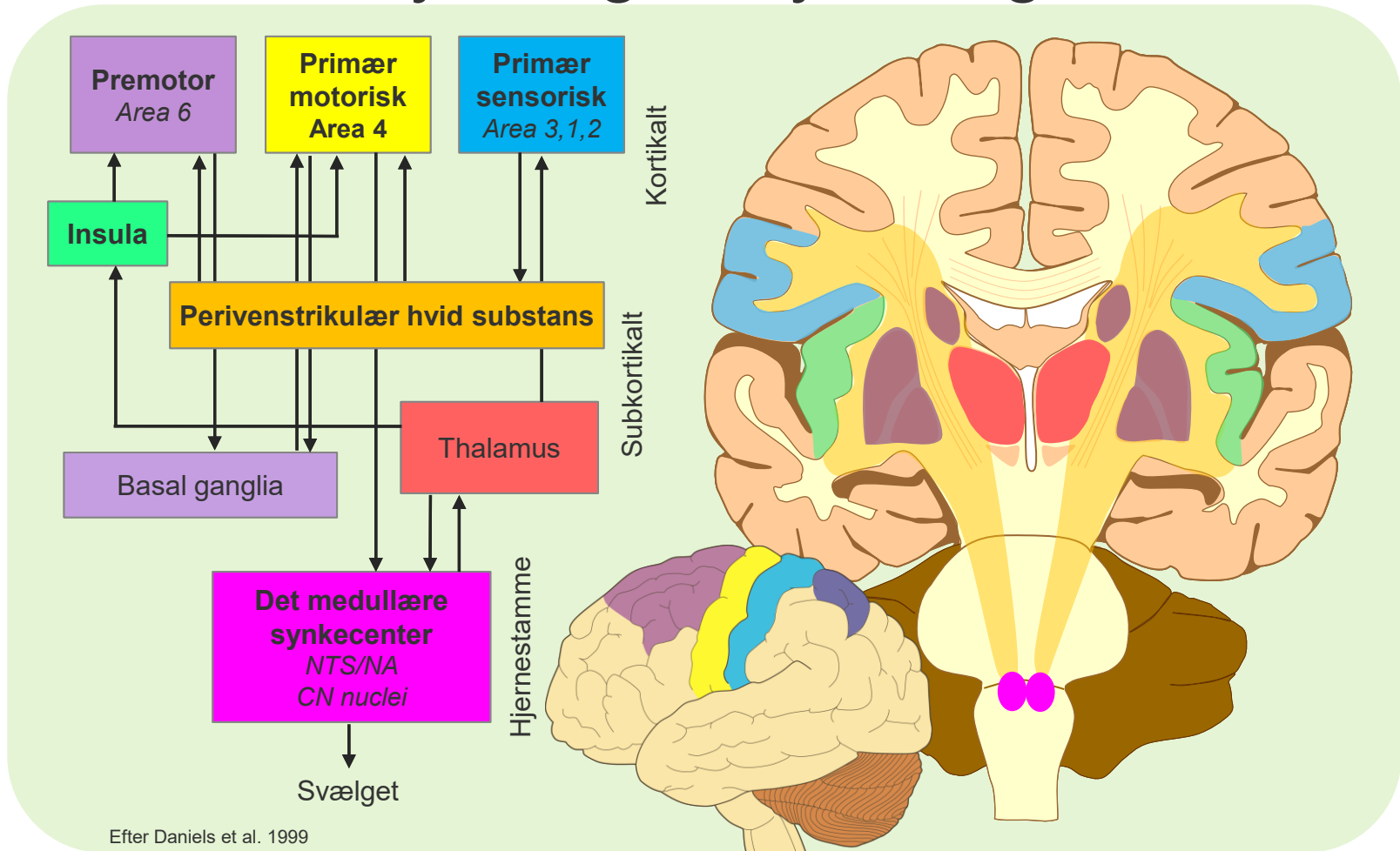


Illustration: Jones et al 2011 & Jacob Liljehult 2022

Fysiologisk synkning

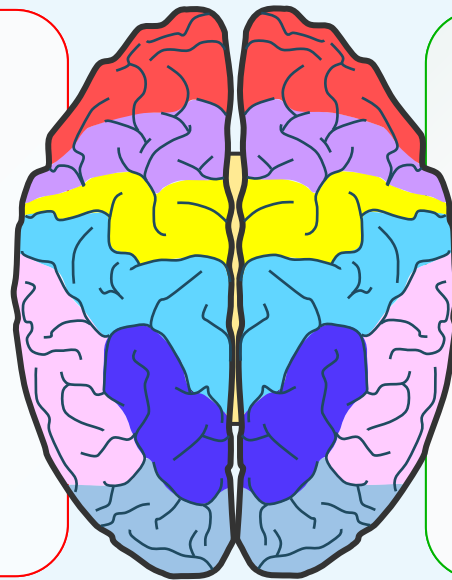


Efter Daniels et al. 1999

Sprog og kommunikation

Venstre side

- Afasi (Produktion, forståelse)
 - Flydende
 - Ikke-flydende
 - Global
- Agrafi (Skrivning)
- Aleksi (Læsning)

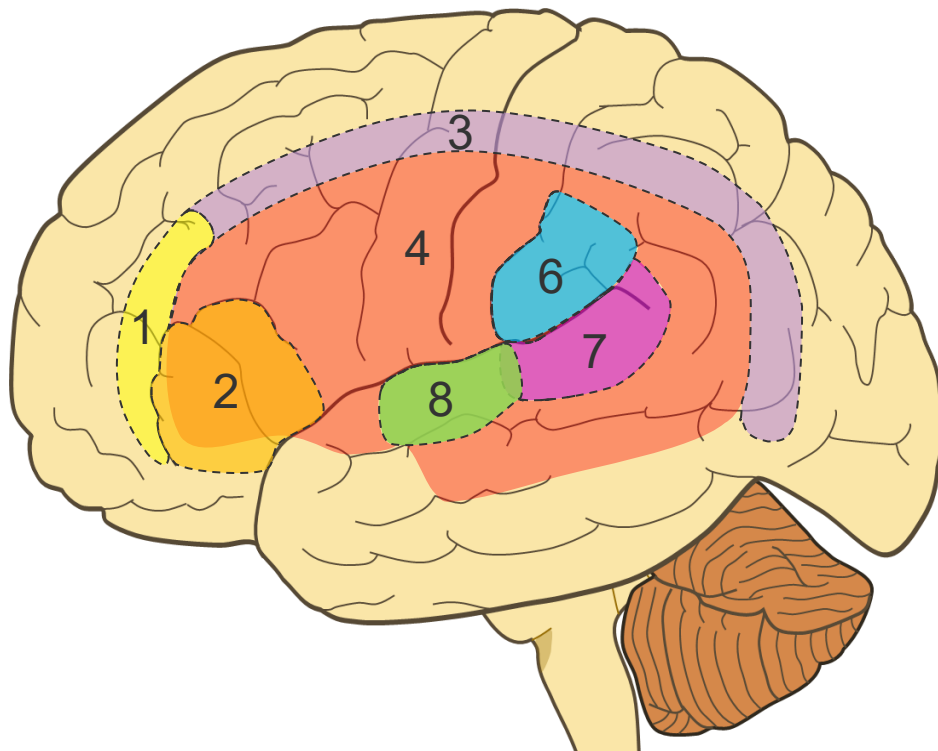


Højre side

- Intonation af sproget
- Giver sproget affekt
- Sproglig struktur

Begge sider

- Dysartri (Eksikution)



Ikke-flydende afasi

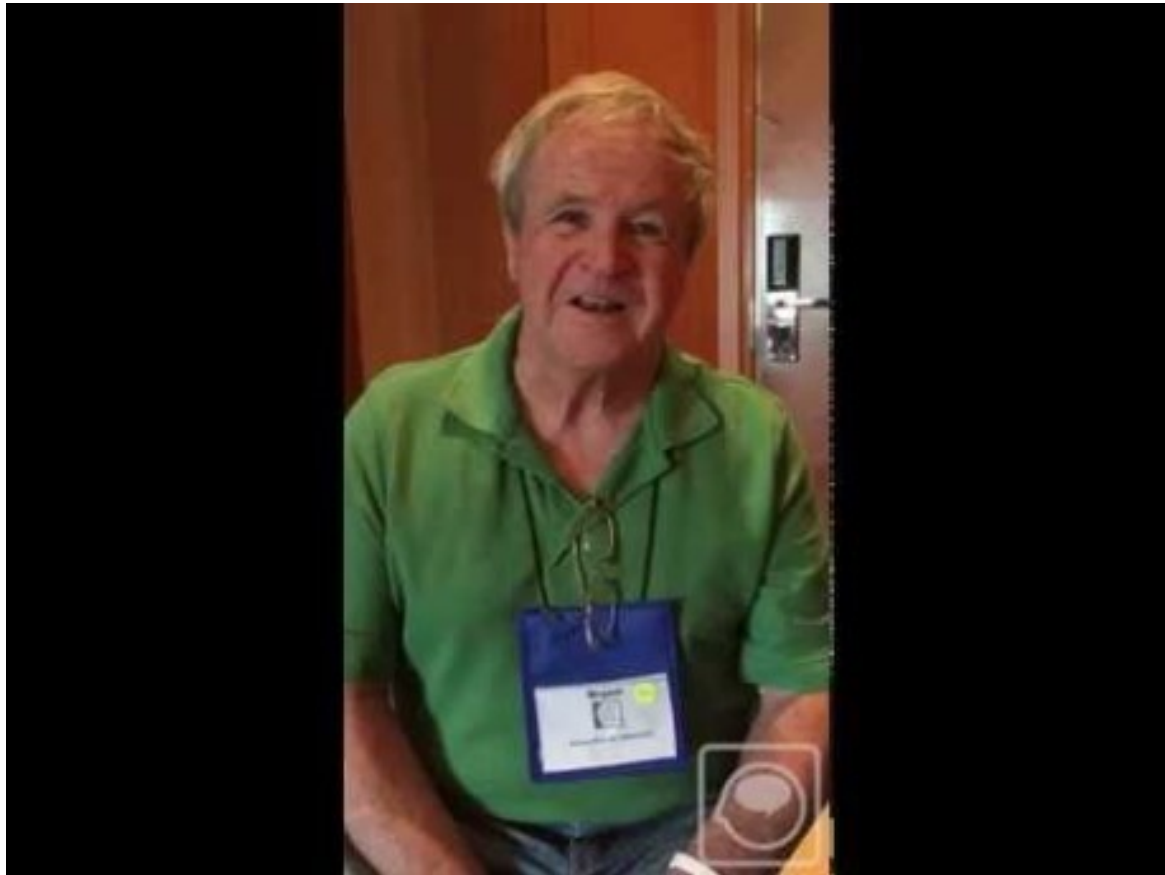
	Forståelse	Gentagelse	Afasitype
1	God	God	Transkortikal motorisk afasi
2	God	Dårlig	Broca-afasi
3	Dårlig	God	Blandet transkortikal afasi
4	Dårlig	Dårlig	Global afasi

Flydende afasi

	Forståelse	Gentagelse	Afasitype
5	God	God	Anomisk afasi
6	God	Dårlig	Konduktionsafasi
7	Dårlig	God	Transkortikal sensorisk afasi
8	Dårlig	Dårlig	Wernicke-afasi

Tekst efter Gade et al 2009; Illustration: Jacob Liljehult 2020

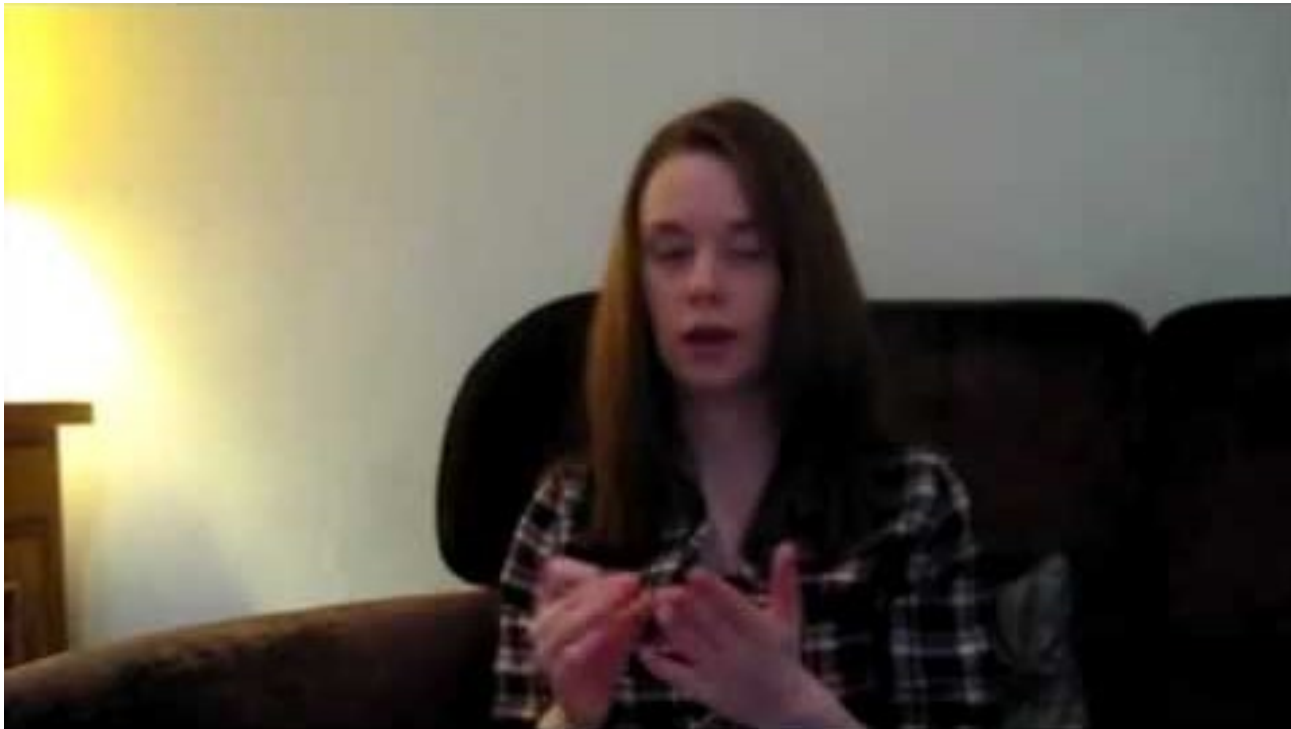
Flydende afasi



Manglende forståelse; flydende talestrøm uden relevant indhold

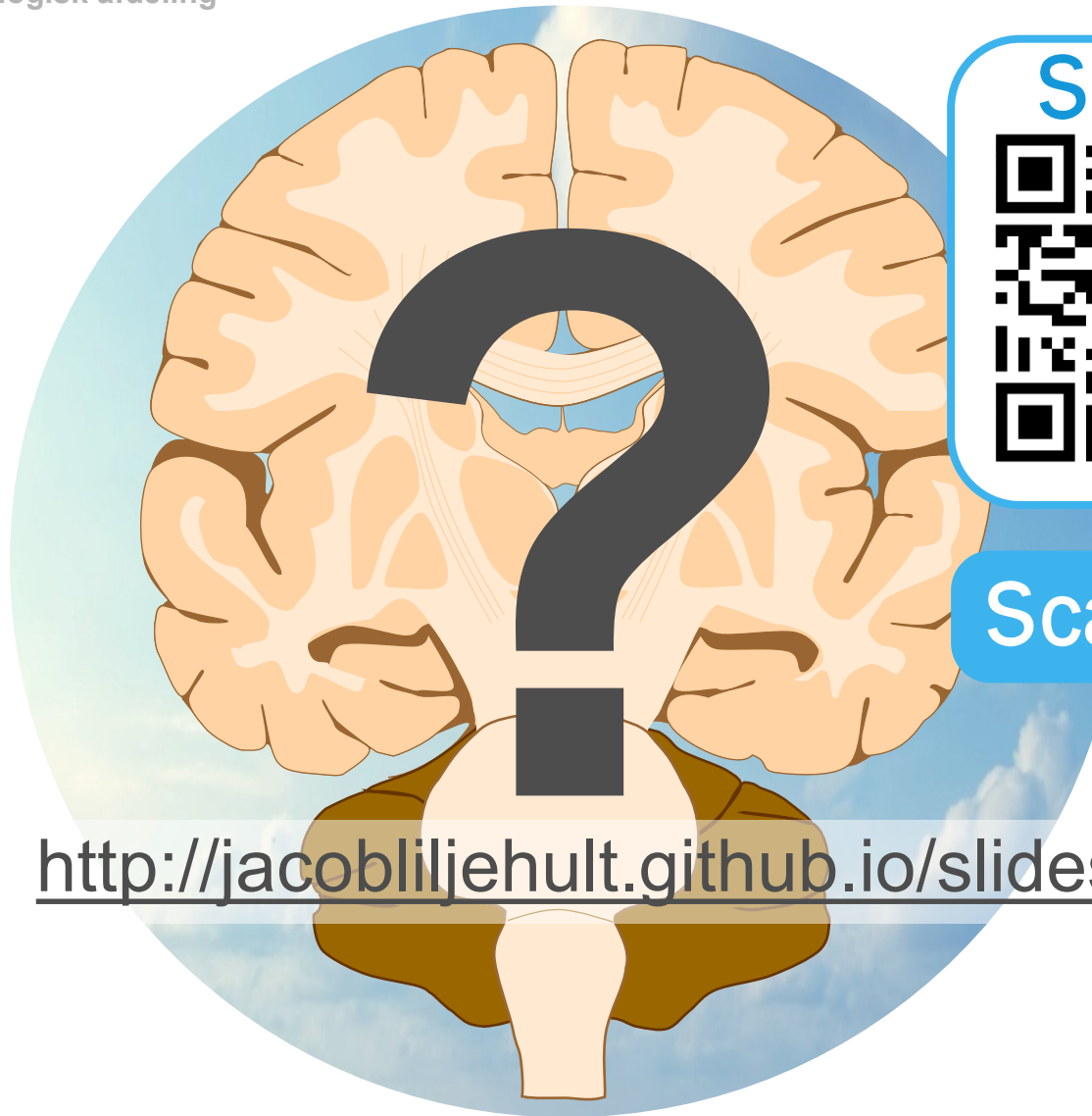
<https://youtu.be/3oef68YabD0>

Ikke-flydende afasi



God forståelse
Svarene er opbrudte og indeholder kun
meningsbærende enheder

<https://youtu.be/IP8hkopObvs>



Slides



Scan mig

<http://jacobliljehult.github.io/slides>