ZNS ERKRANKUNGEN

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Semmelweis Egyetem – I. Patológiai és Kísérleti Rákkutató Intézet

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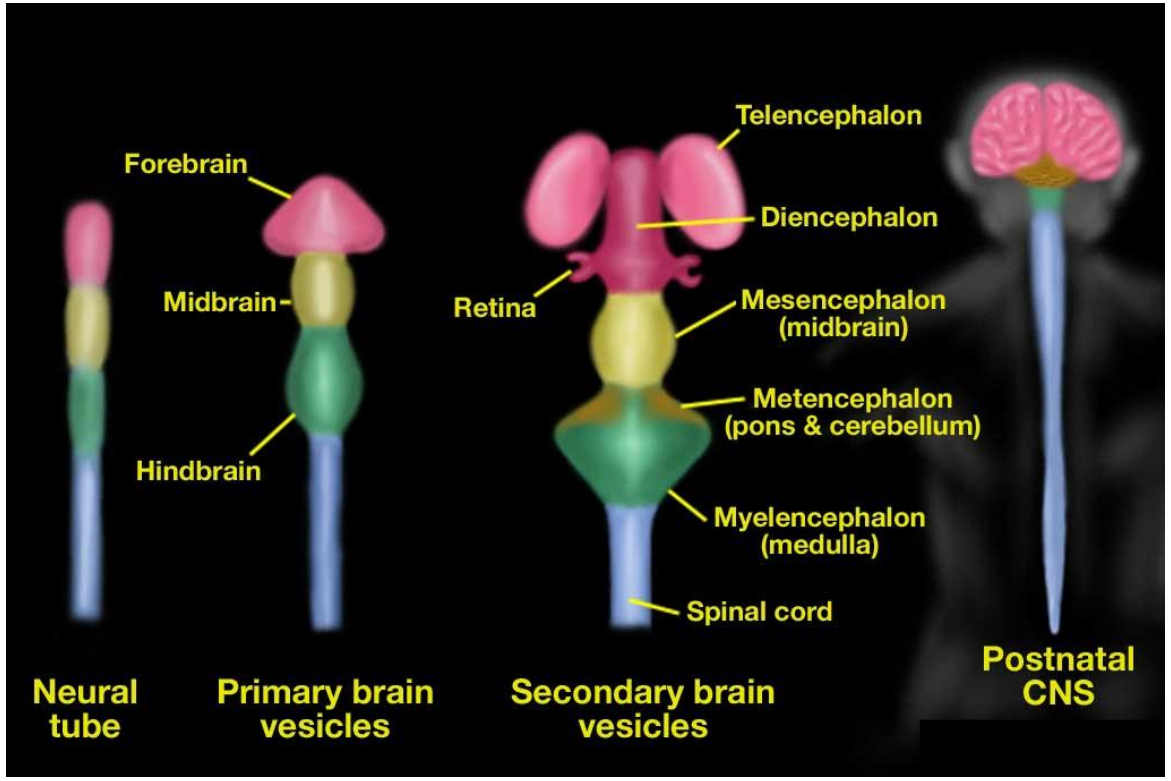
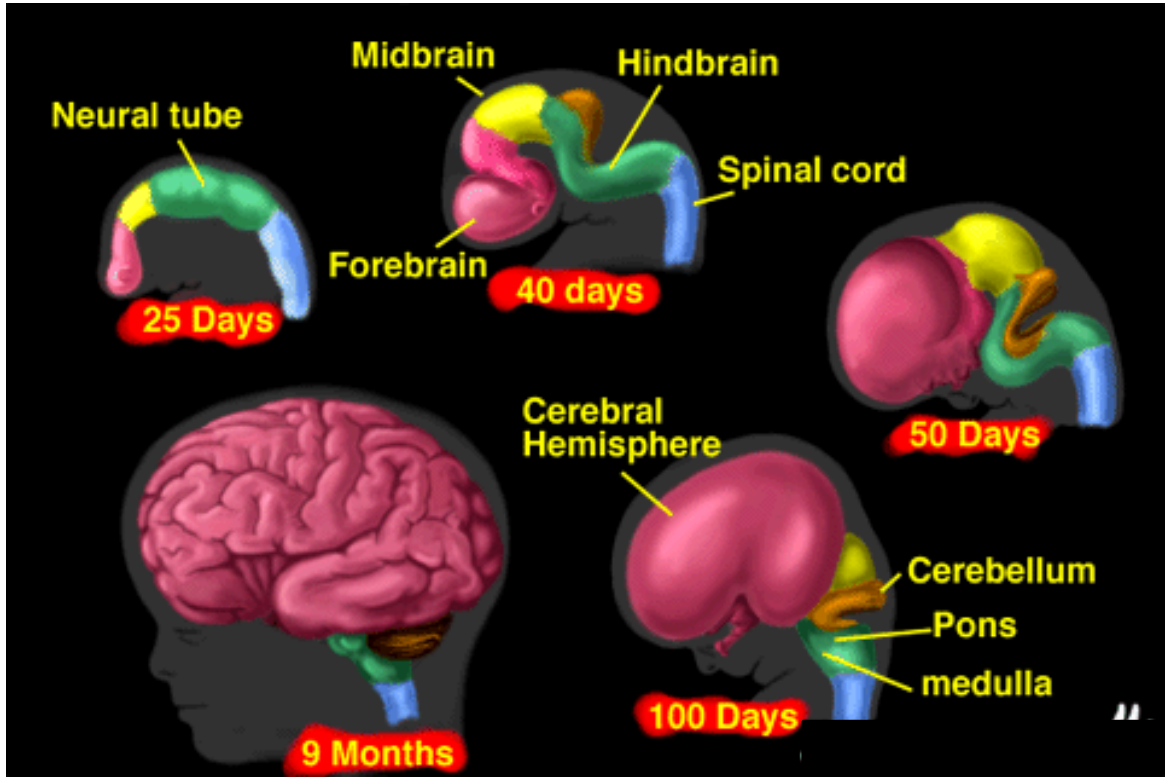
ENTWICKLUNG DES ZNS





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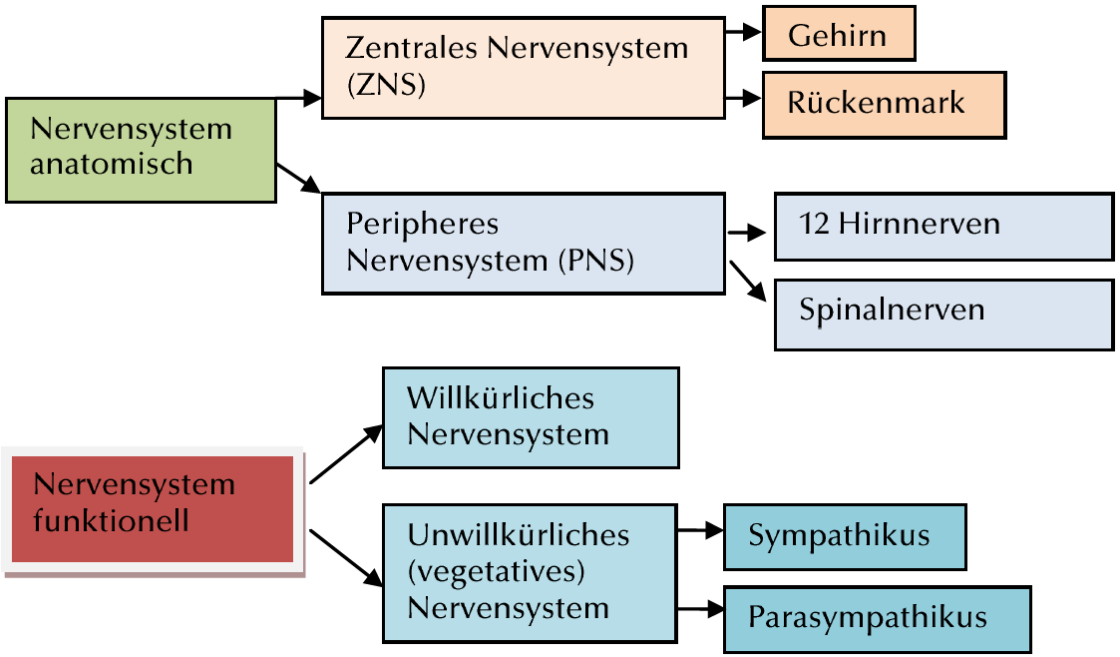
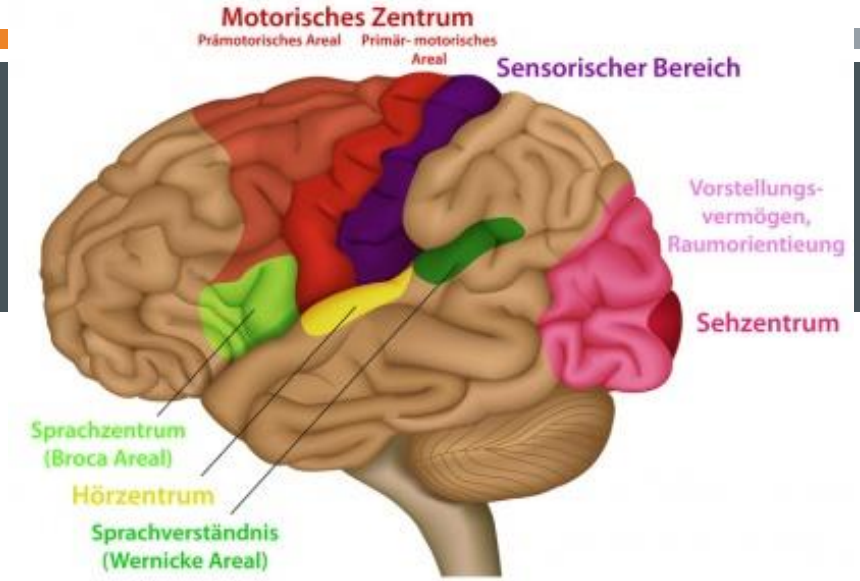
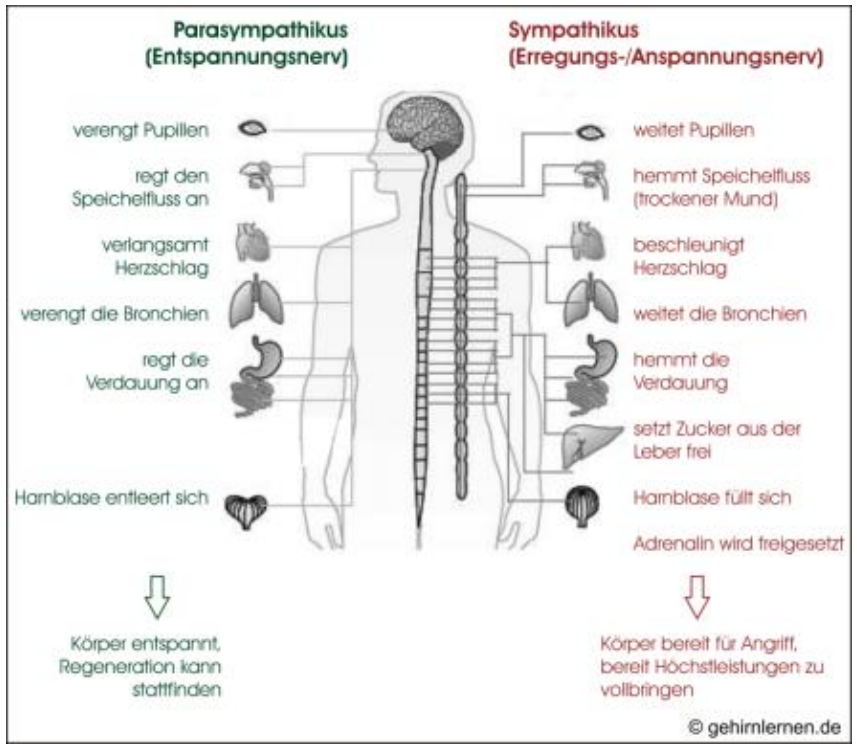
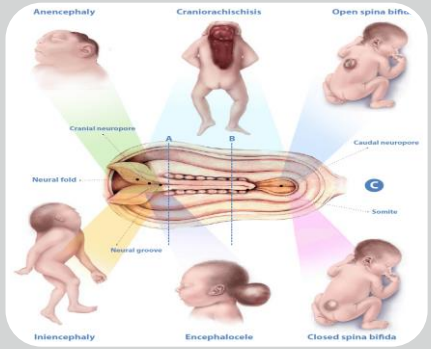
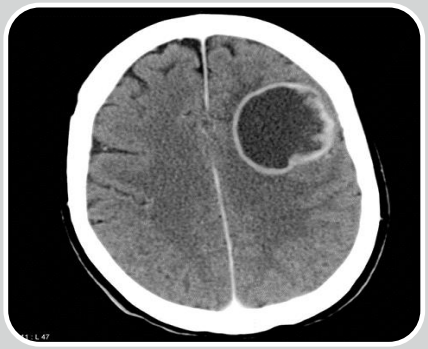


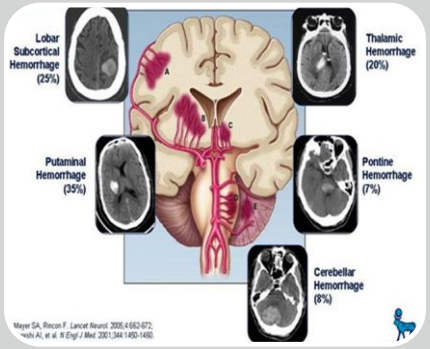


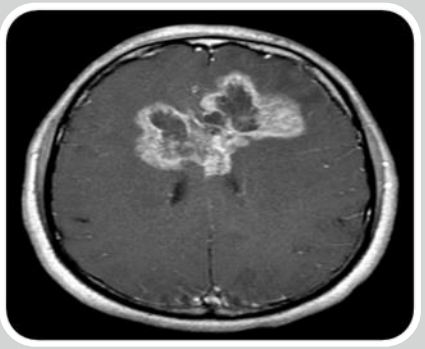


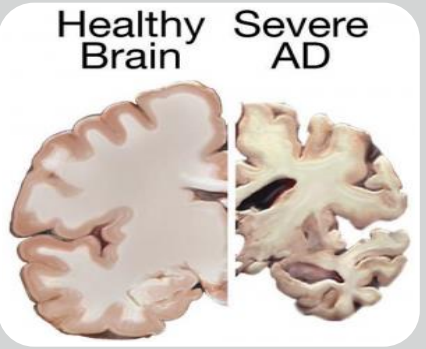
ERKRANKUNGEN DES ZNS











Angeborene Fehlbildungen des ZNS

Entzündliche Erkrankungen des ZNS

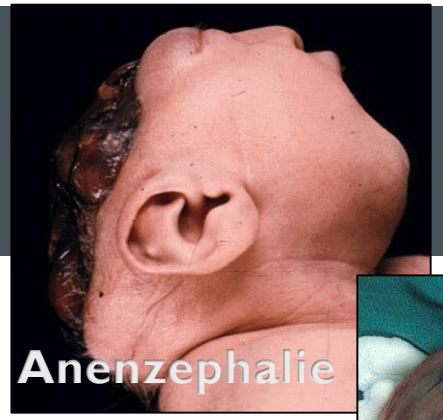
Zirkulations-störungen des ZNS

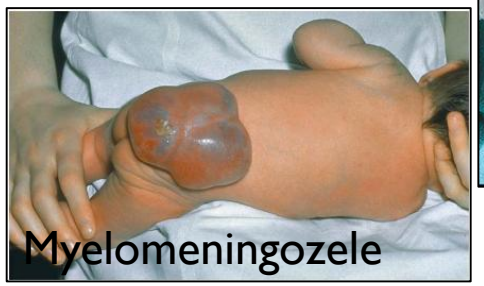
Tumoren des ZNS

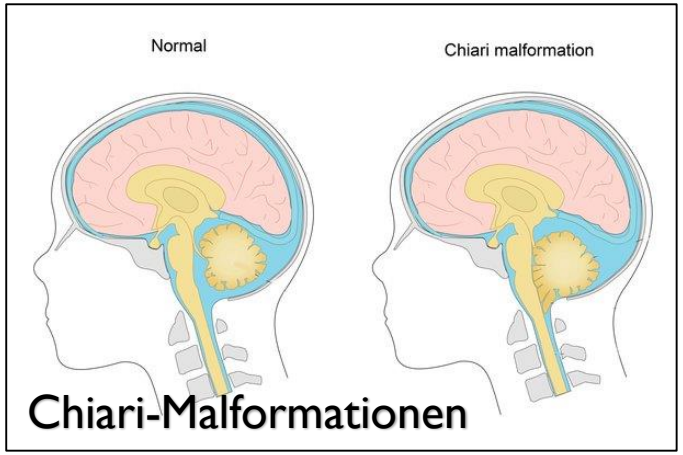
Neurodegenerative Erkrankungen des ZNS

ANGEBORENE FEHLBILDUNGEN DES ZNS

- **Dysraphische Störungen**
  - Anenzephalie
  - Enzephalozele
  - Spina bifida – Meningozele, Myelomeningozele, Rachischisis
- **Chiari-Malformationen**
- **Telenzephalisationsstörungen**
  - Holoprosenzephalie
  - Agenesie des Corpus callosum
- **Neuronale Migrationsdefekte**
  - Agyrie
  - Polymikrogyrie



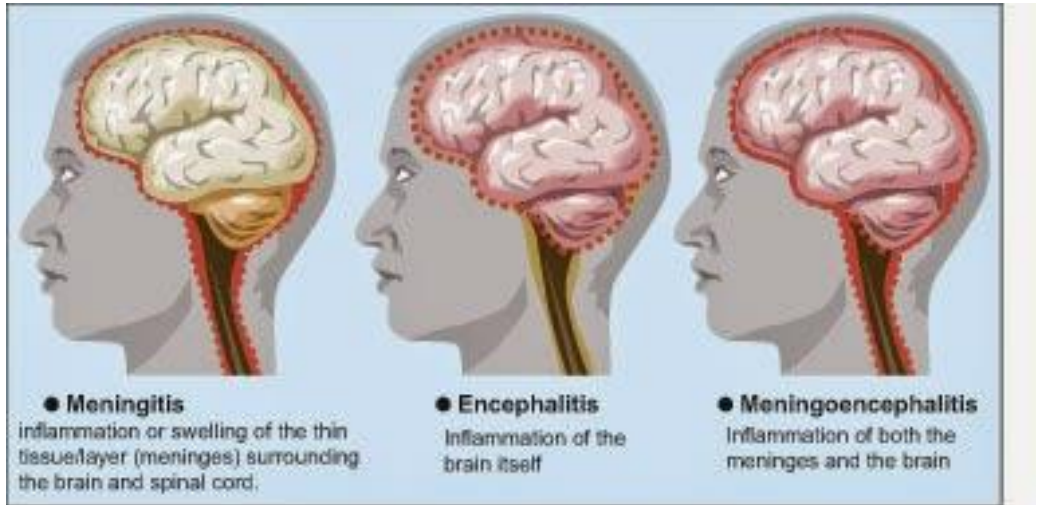




ENTZÜNDLICHE ERKRANKUNGEN DES ZNS

Lokalisation:

1. Gehirn, Rückenmark: Enzephalitis, Myelitis, Enzephalomyelitis.
2. Hirnhaute: Meningitis, Pachymeningitis.
3. Parenchyma und Hirnhaute: Meningoenzephalitis.



ENTZÜNDLICHE ERKRANKUNGEN DES ZNS

Infektionsweg

1. **Hämatogen - Sepsis oder bei einer infektiösen Endokarditis**
2. **Direkte implantation – Trauma, Iatrogen**
3. **Fortgeleitete Infektion – Otitis media, Zahnabszess, Congenitale malformation**
4. **Retrograd - Peripherale Nerven**

ENTZÜNDLICHE ERKRANKUNGEN DES ZNS

Erreger

1. Bakterielle Infektionen

- Bakterielle meningitis
- Gehirn abcess
- Tuberculosis
- Neurosyphilis

2. Virus Infektionen

- Viral meningitis
- Herpesvirus
- Cytomegalovirus
- Poliovirus
- Rabies
- HIV
- Progressive multifocal leukoencephalopathia

3. Mykotische Infektionen

- Candida
- Mucormycosis
- Aspergillus
- Cryptococcus

4. Protozoen Infektionen

- Toxoplasma

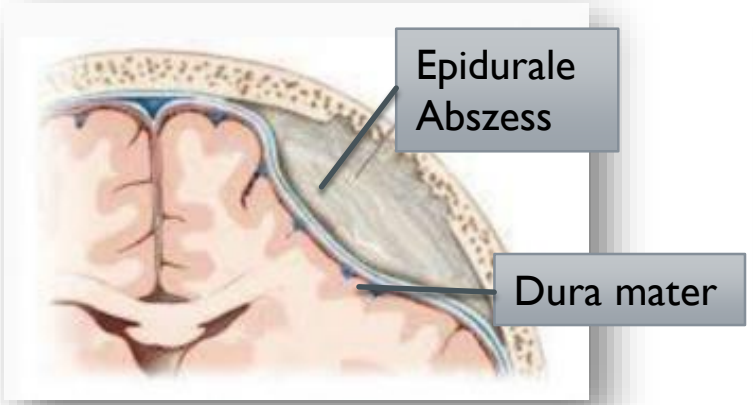
5. Parazitäre Infektionen

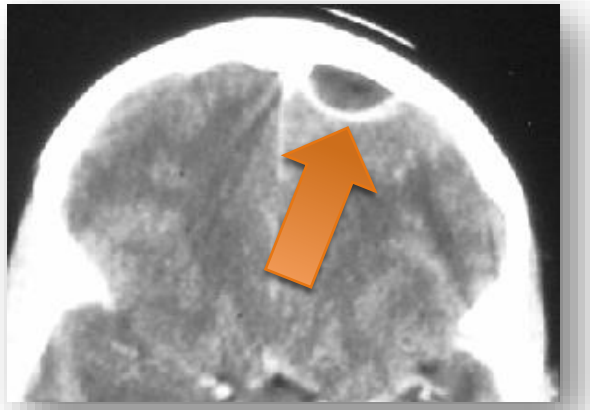
- Cystercosis
- Echinococcus

EPIDURALE UND SUBDURALE INFEKTIONEN

I. Epiduralabszess

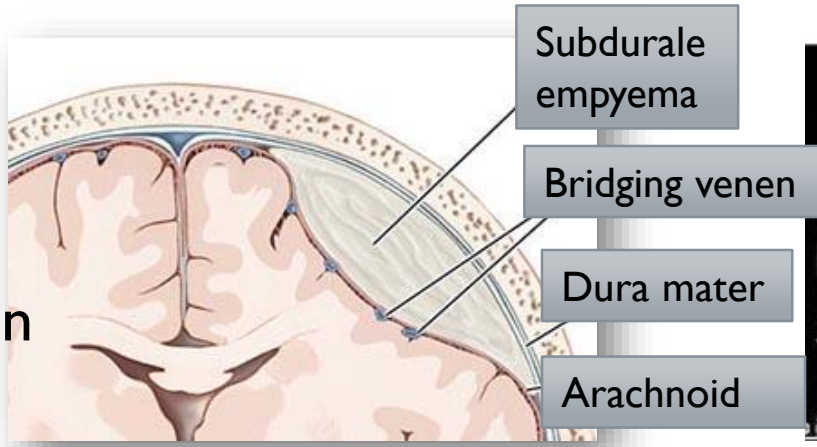
- Fortgeleitete Infektion – sinuitis, osteomyelitis
- Bacterielle, Mykotisch
- Rückenmark- Kompression

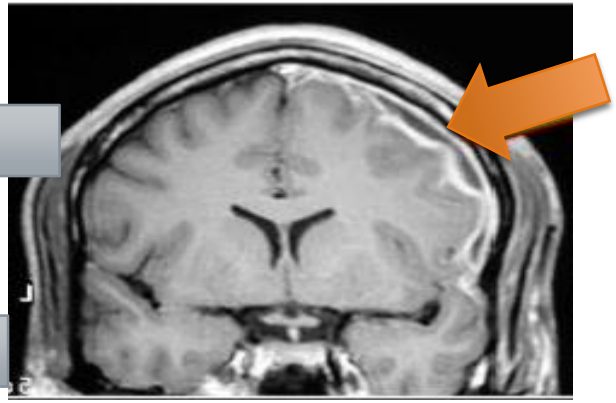




II. Subdurale Empyema

- Fortgeleitete Infektion – sinusitis
- Arachnoidealraum und Subarachnoidealraum nicht betroffen
- Thrombophlebitis –Septische Thrombosen

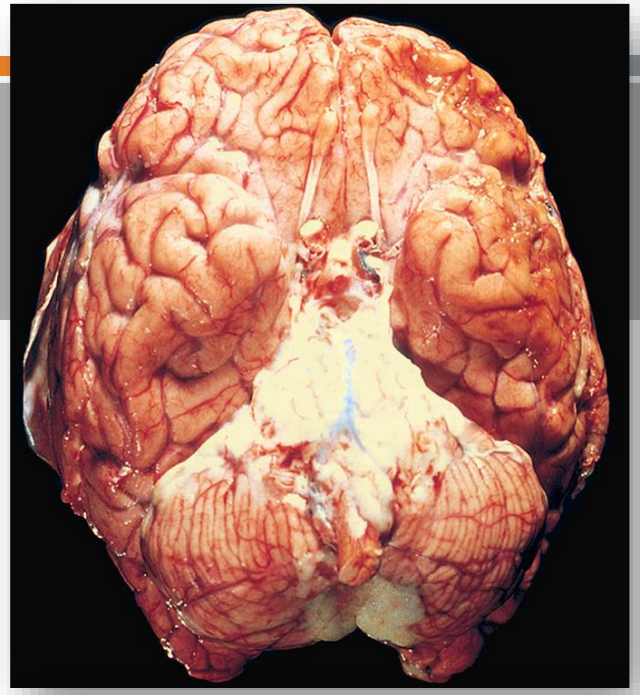


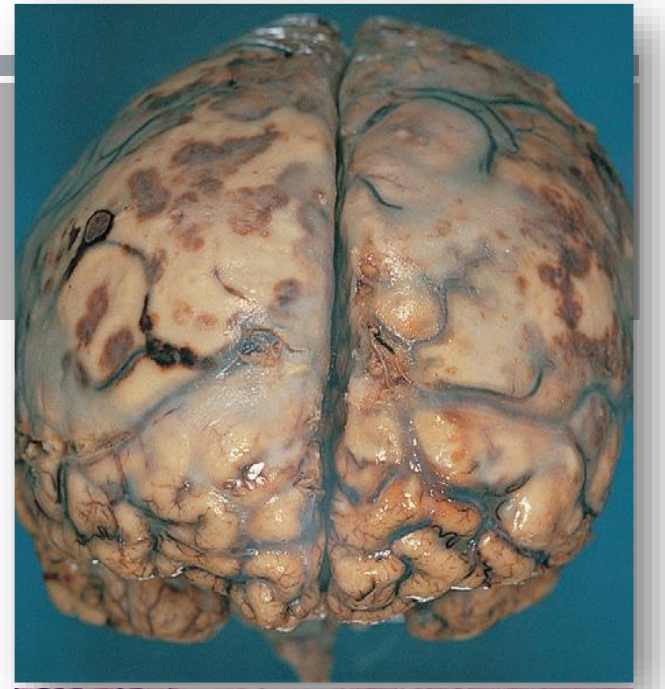


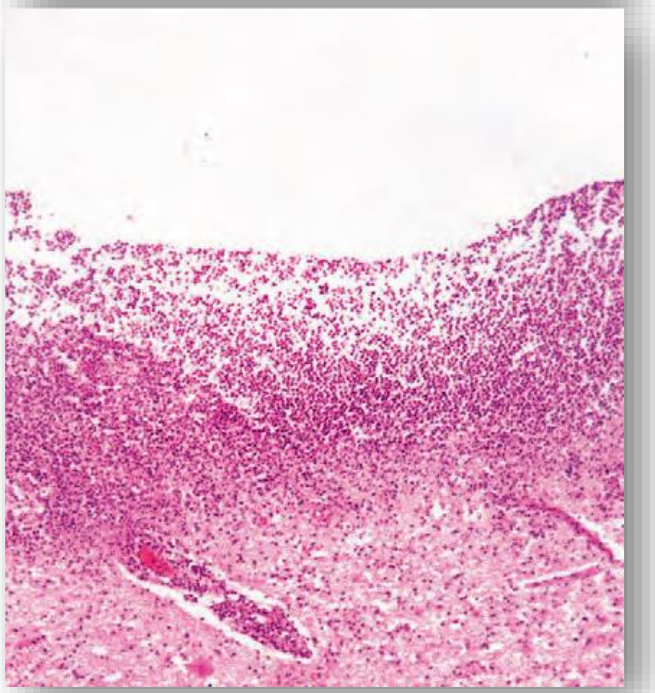
MENINGITIS

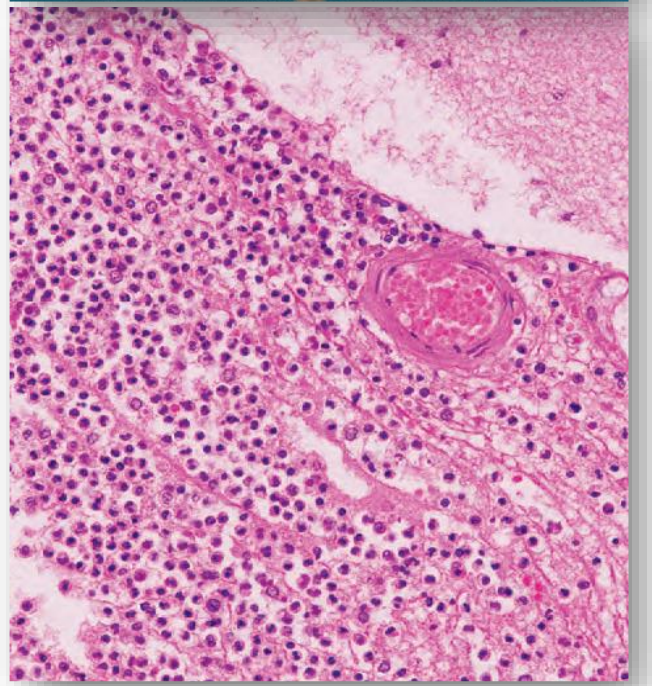
I. Bakterielle Meningitis

1. Neronatale
   - Escherichia coli
   - B Streptococcus
2. Kindliche Infektionen
   - Neisseria meningitidis
   - Streptococcus pneumoniae
3. Infektionen des Erwachsenenalters
   - Streptococcus pneumoniae
   - Listeria monocytogenes



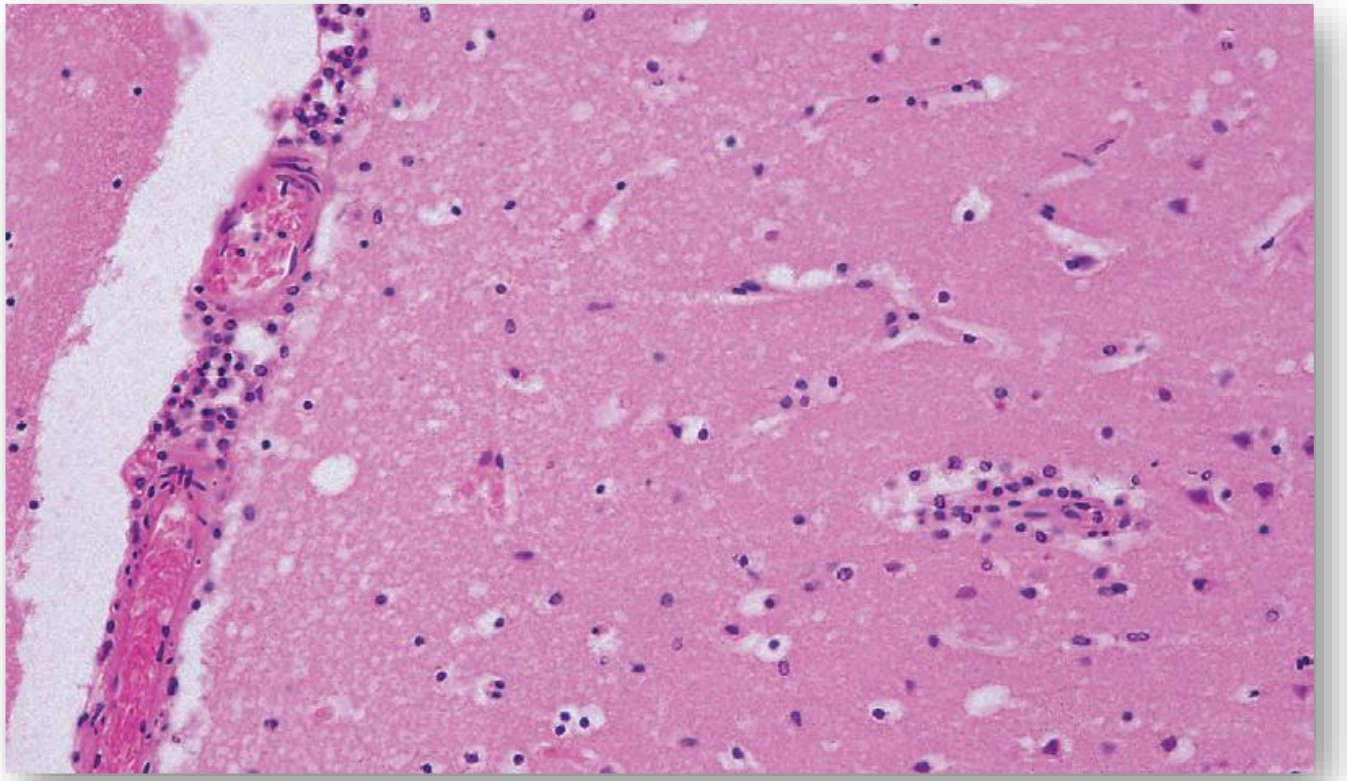






II. Aseptische / Akute virale Meningitiden

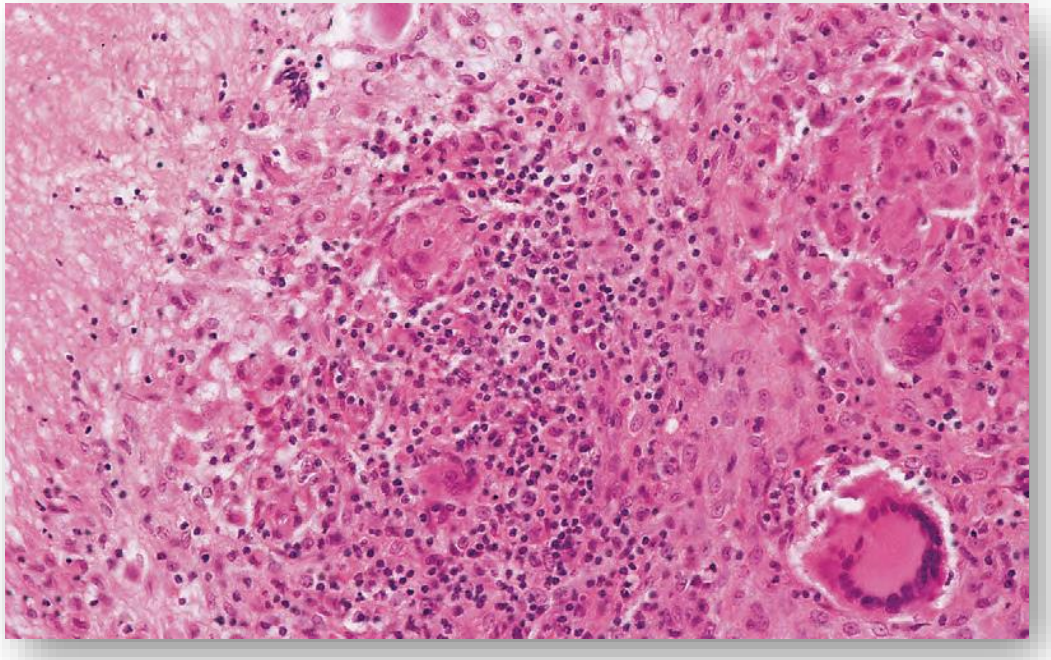
Echovirus
Coxsackie B
Coxsackie A
Herpes simplex virus (HSV)-2
Mumps
Human immunodeficiency virus (HIV)
Lymphochoriomeningitis virus
Arbovirus
Rubeola
Parainfluenza virus
Adenovirus

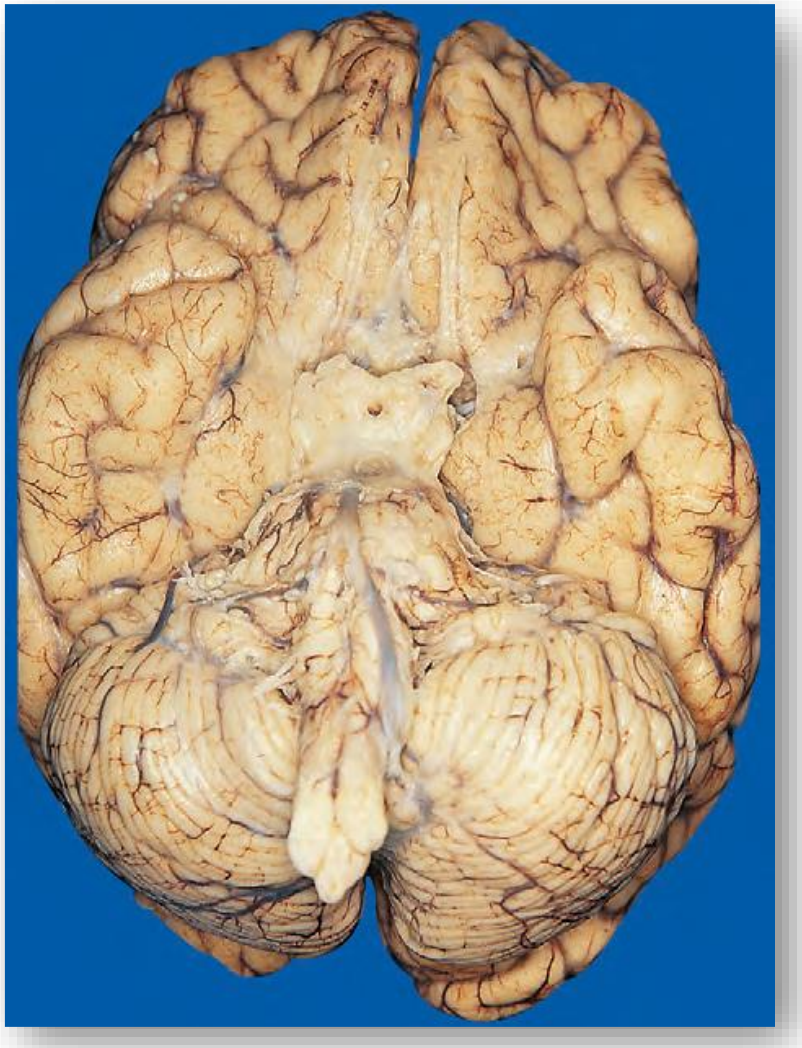


III. Chronic meningitis

III.I. Mycobacterium tuberculosis

- Basale Meningitis – Fibrinöse Exudat
- Intraparenchymale Masse (tuberculoma)
- Chronische tuberkulöse Entzündung - arachnoideale Fibrose - hydrocephalus



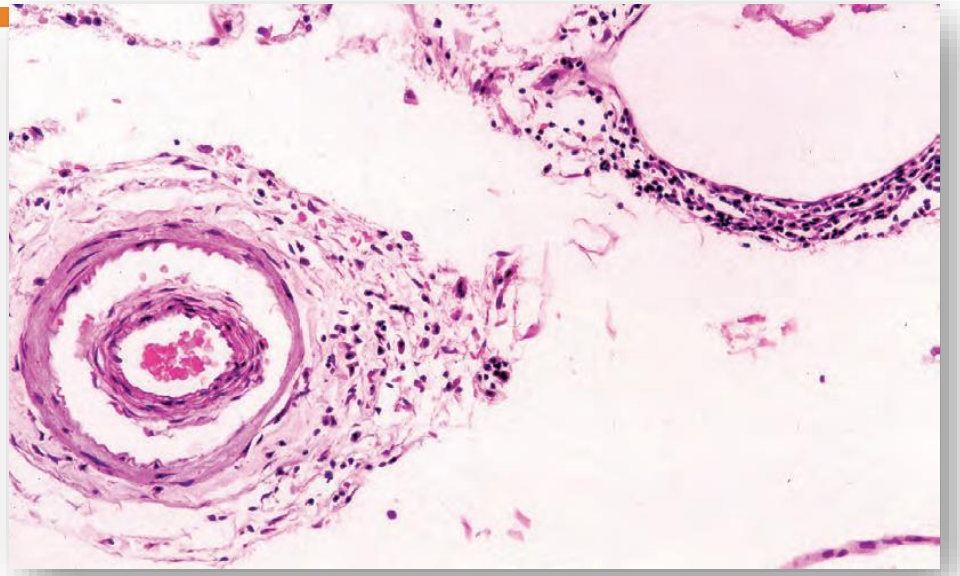


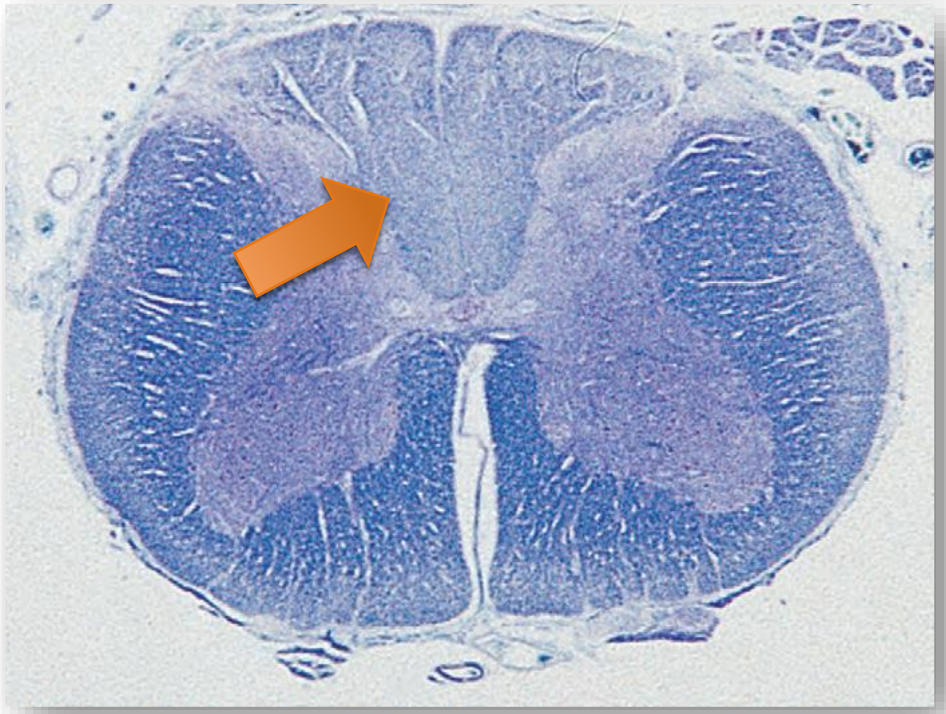
III.II. Spirochaeten Infektionen

A. Neurosyphilis (Tertiärstadium) – Treponema pallidum

- 10% nach unbehandelter Erstinfektion

1. Chronische Meningitis/Meningovaskuläre Neurosyphilis
   - Basale meningitis
2. Progressive Paralyse
   - Neuron Verlust– frontotemporal-betonten Enzephalitis
3. Tabes dorsalis
   - Degeneration der Hinterstränge im Bereich der Hinterwurzeln
   - Ataxia

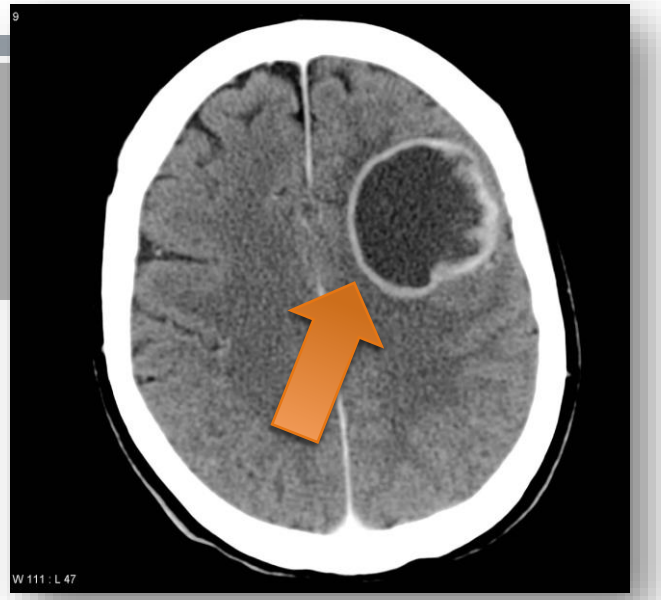




B. Neuroborreliose – Borrelia burgdorferi

- Aseptische Meningitis
- Nervus Facialis Lähmung
- Milde Encephalopathie
- Polyneuropathie

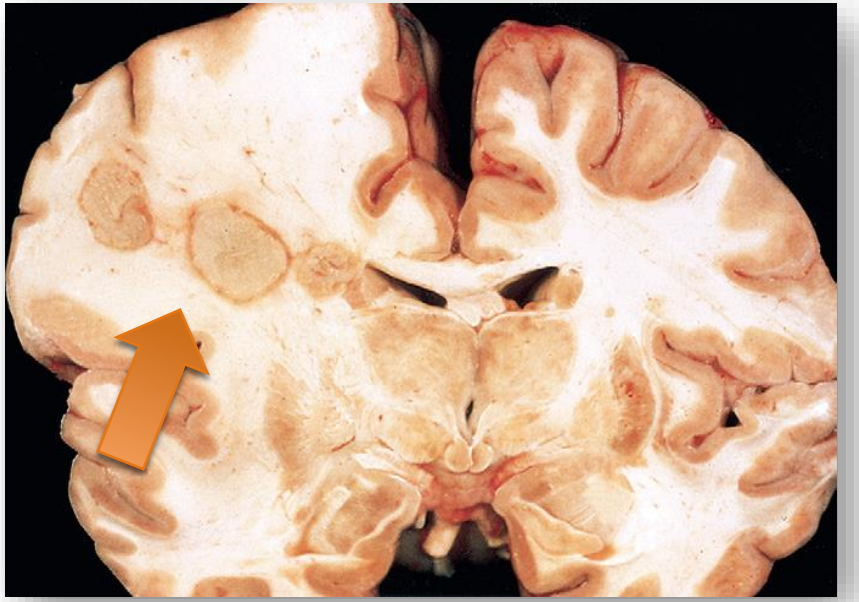
ENTZÜNDUNGEN DER HIRNSUBSTANZ

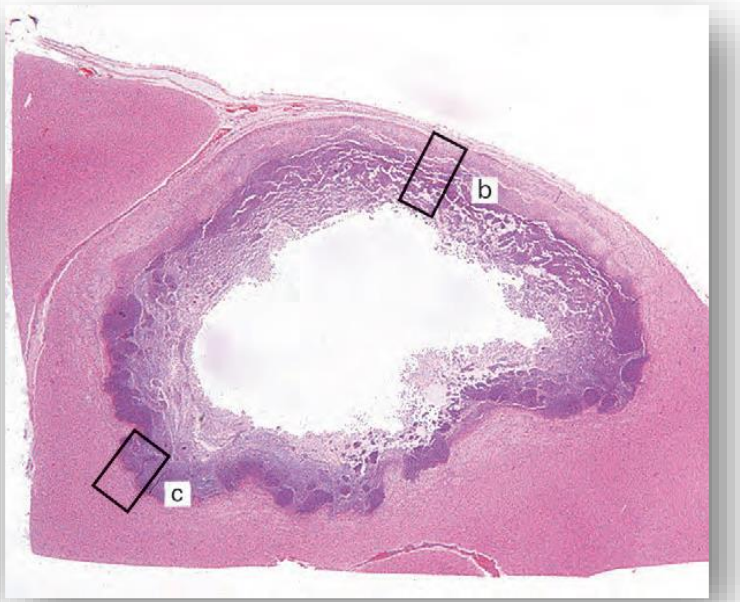


I. Hirnabszess

- Bakterielle Infektionen

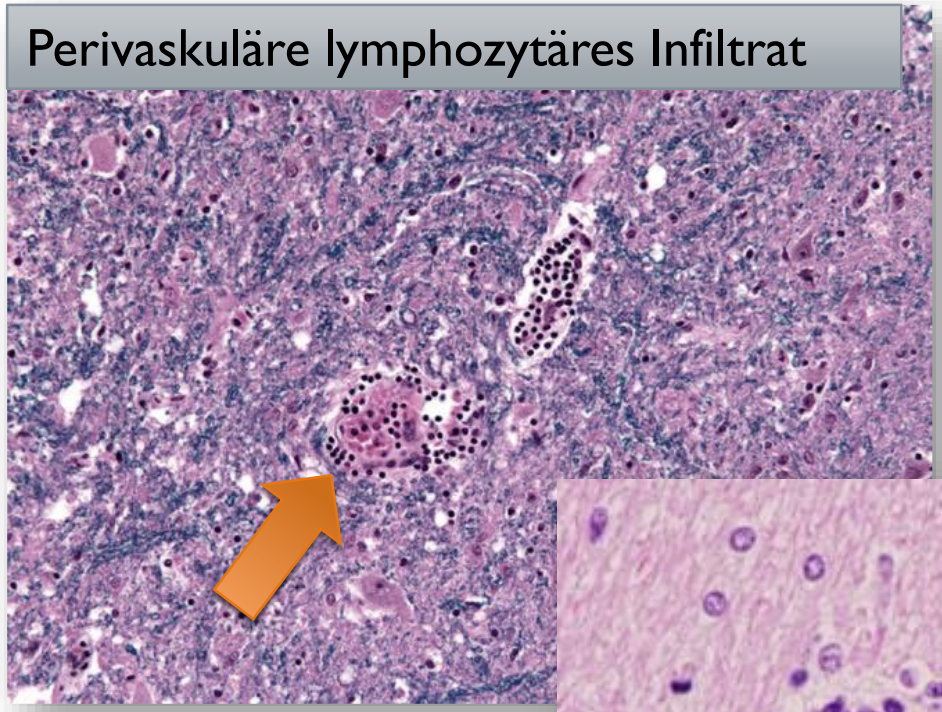
- Infektionsweg:
  - Fortgeleitete Infektion
  - Direkte
  - Hematogän
- Symptomen - Fokale



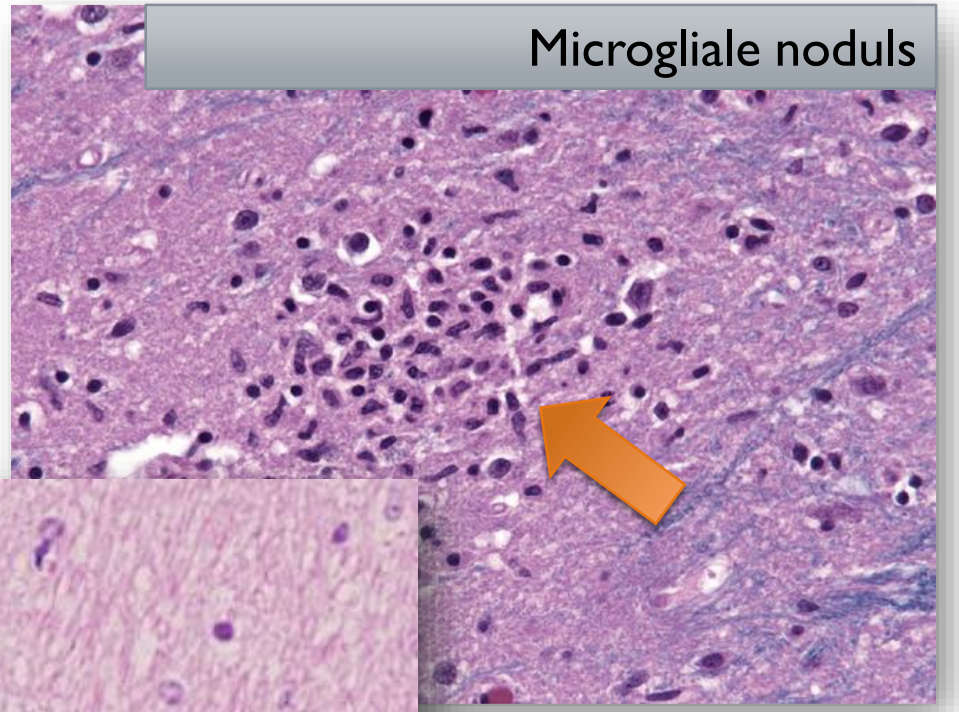


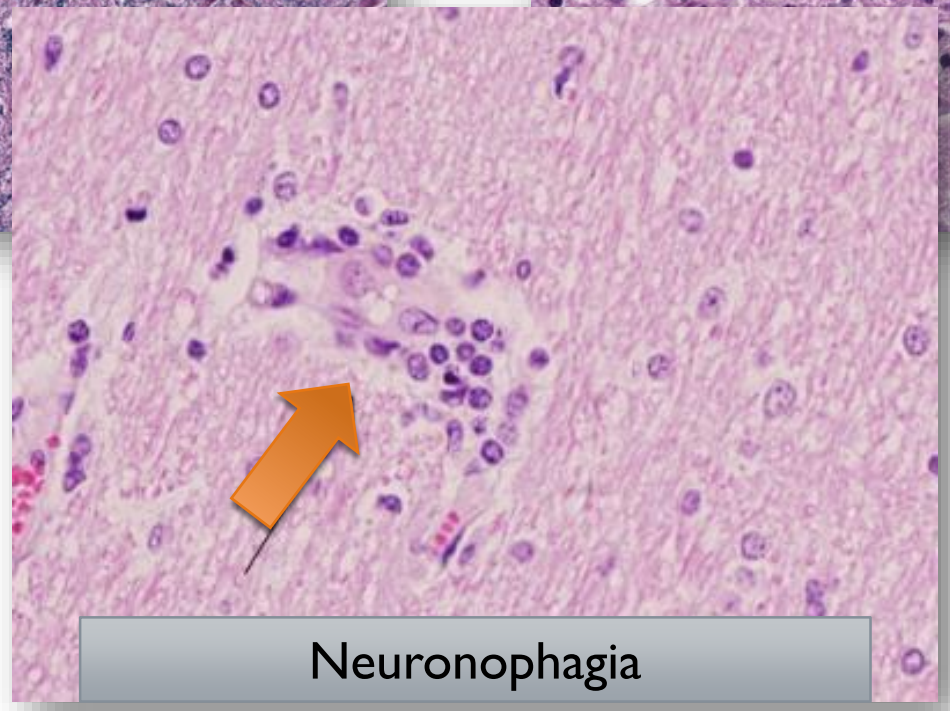
II. Virale Encephalitis

Perivaskuläre lymphozytäres Infiltrat



Microgliale noduls





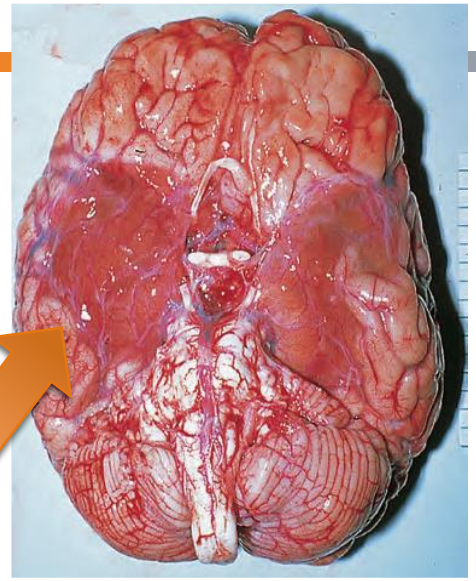
Neuronophagia

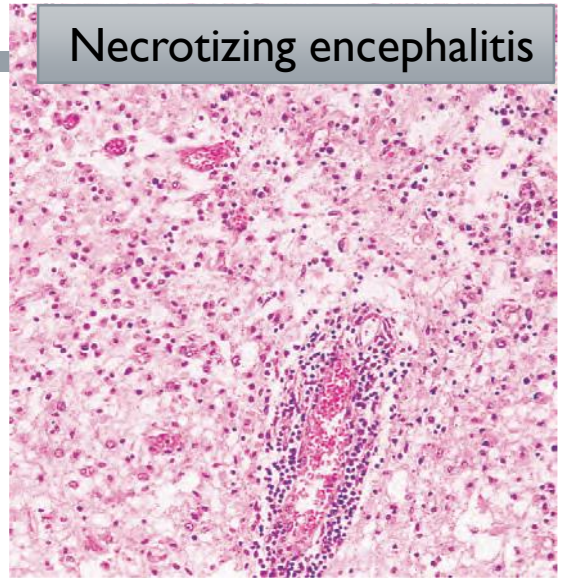
II. I. Herpes virus

A. Herpes simplex-1
- Kinder, Jugendliche
- Frontale, temporale Lappen
- Nekrotisierende encephalitis

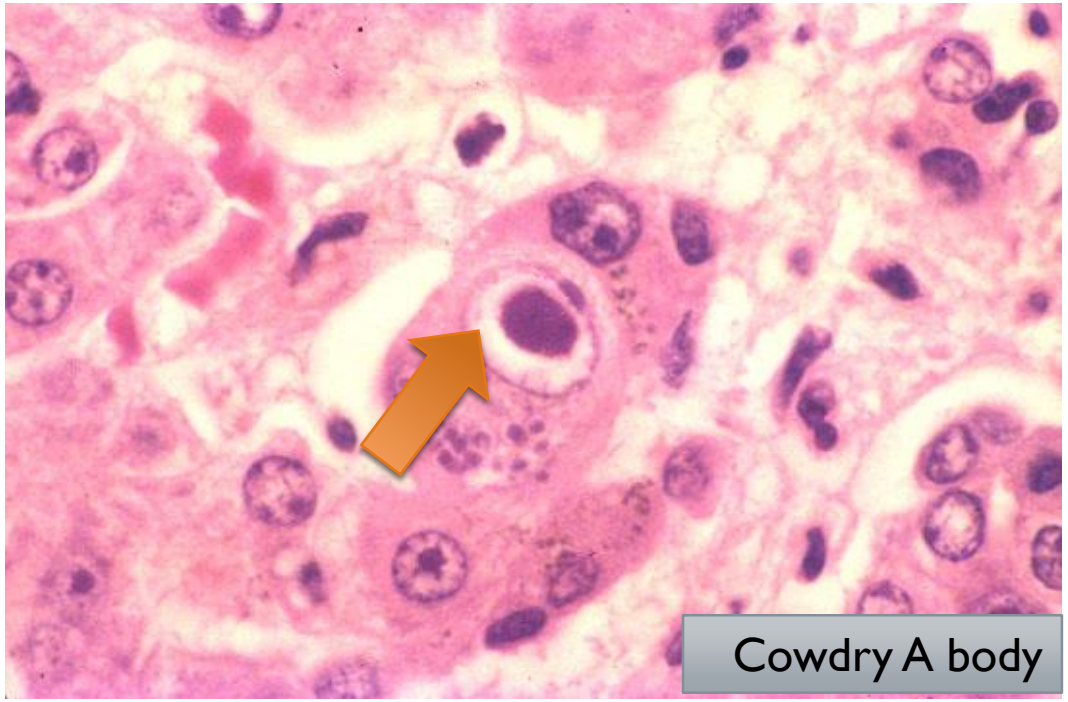
B. Herpes simplex-2
- Erwachsene
- Virale Meningitis
- Primär HSV genitale inf - Neugeborene

C. Varicella zoster
- Im Rahmen einer Immunsuppression
- HZV Encephalitis





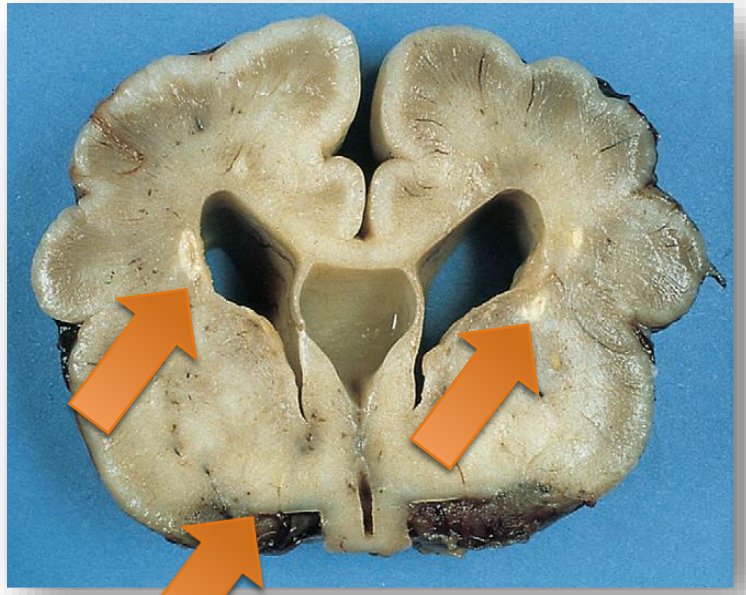
Necrotizing encephalitis

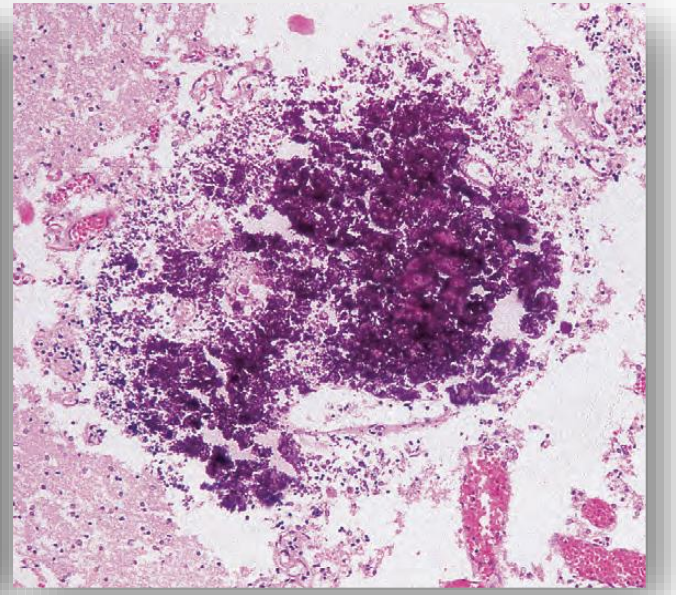


Cowdry A body

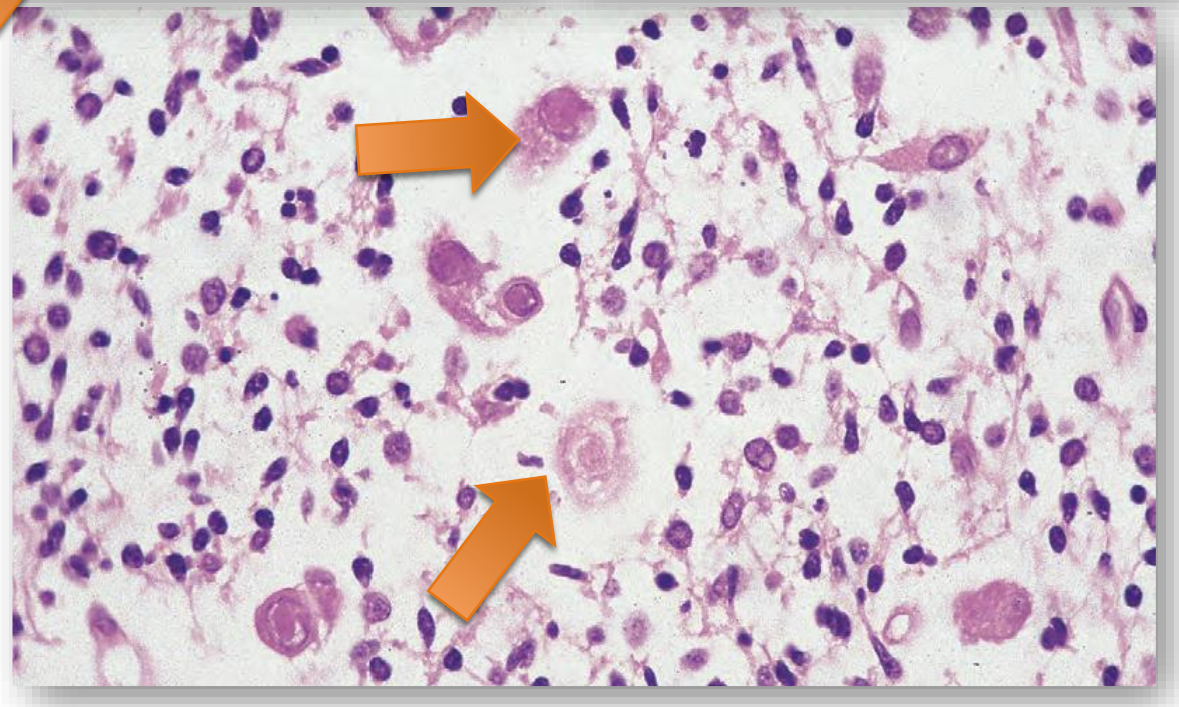
II. II. Zytomegalovirus

A. Fötus
   - Periventriculare Nekrose
   - Microcephalia
   - Periventriculare Kalzifikation

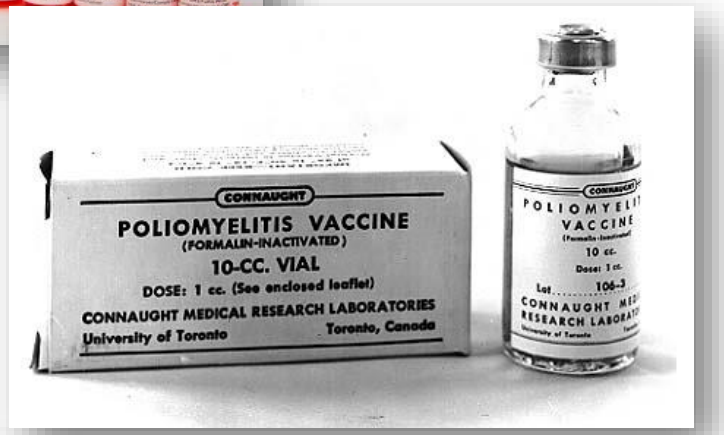




B. Erwachsene
   - Im Rahmen einer Immunsuppression
   - Periventriculare
   - Subacute Enzephalitis



II. III. Poliovirus

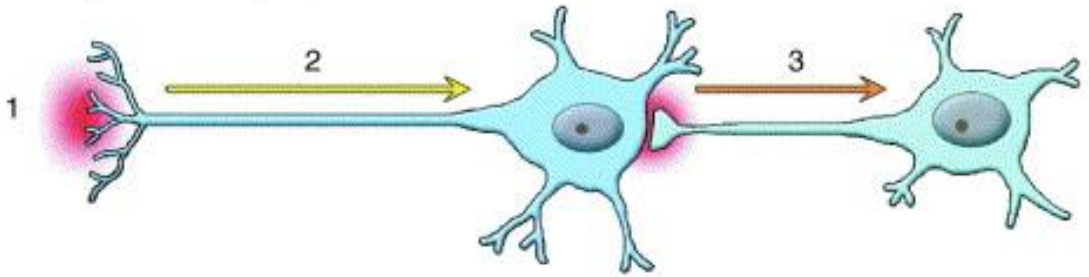


- Gastroenteritis – Sekundärer ZNS Verbreitung
  - Poliomyelitis anterior acuta /Paralytische poliomyelitis
  - Befall von Motorneuronen des Rückenmarks, auch von motorischen Kerngebieten des Hirnstammes
  - Achlaffe Paresen und Lähmungen, Hyporeflexia

- 25-35 Jahren – Postpolio Syndrom
  - Progressive Schwachheit, Schmerz

II. IV. Rabies Virus

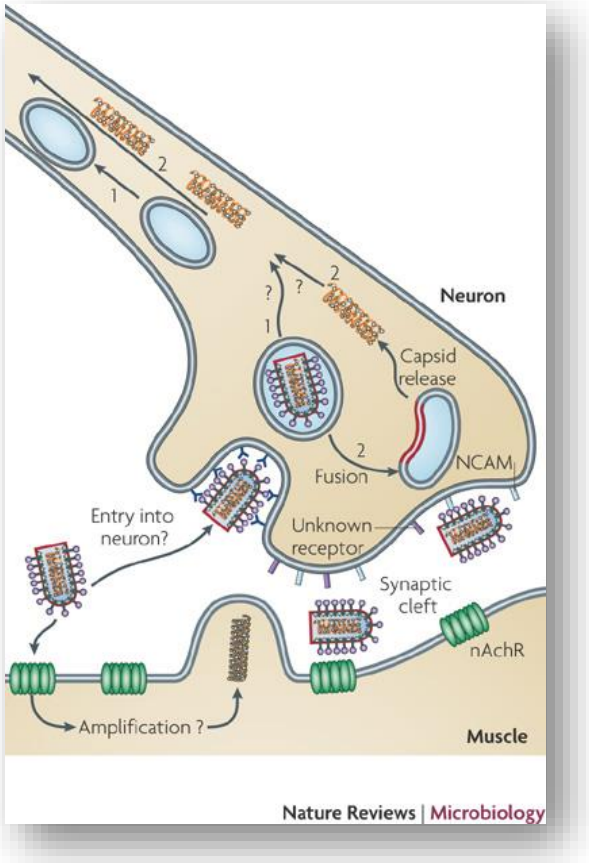
Retrograde trans-synaptic spread



- Tollwut
- Bisswunde

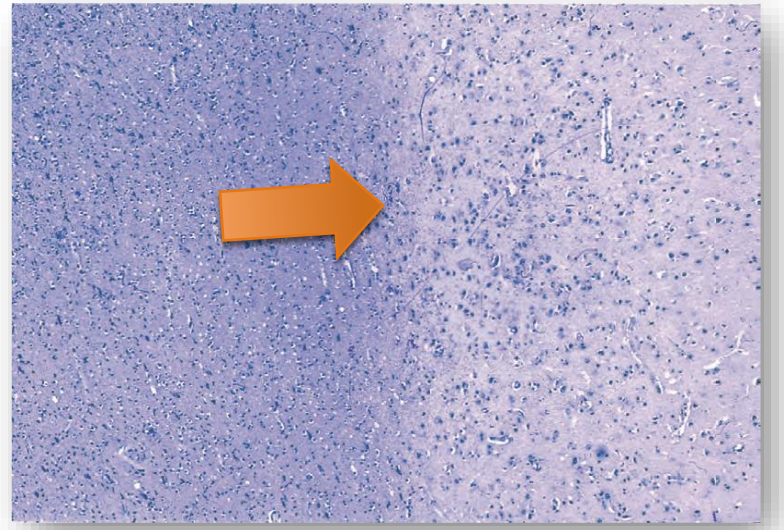
- Aufstiegende Infektion über periphere Nerven
- Speichel- und Tränendrüsen

- Symptomen:
  - Nicht Specifisch
  - Exzitationsstadium
    - Atemmuskel- und Schlundkrämpfen
  - Paralytisches Stadium
    - Lähmungen

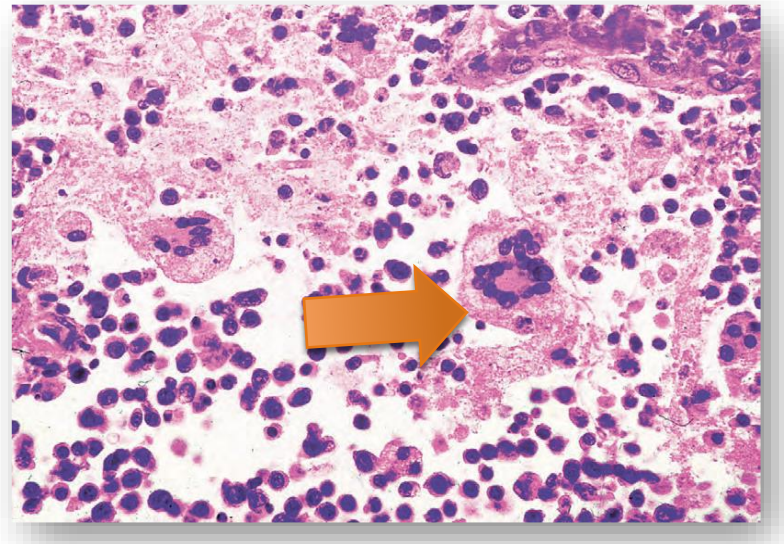


II.V. HIV-assoziierte ZNS-Erkrankungen

A. Aseptische Meningitis
   - 1 -2 wochen 10% der Patienten



B. HIV Enzephalitis (HIVE)
   - Perivasculäre lymphozytäres Infiltrat
   - Myelin Verlust in die Hemispheren (Leukoencephalopathia)
   - Microgliale Knötchen
   - Riesen Zelle



C. Opportunistischer Infektionen

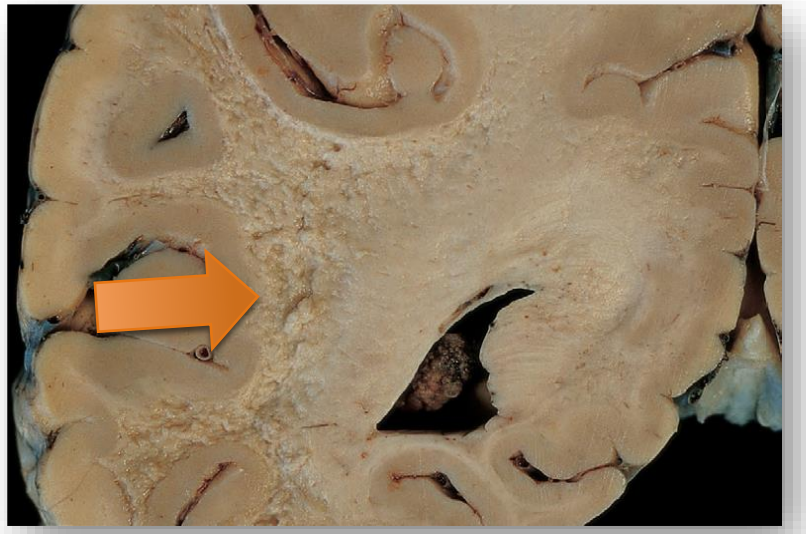
D. Primär ZNS lymphoma

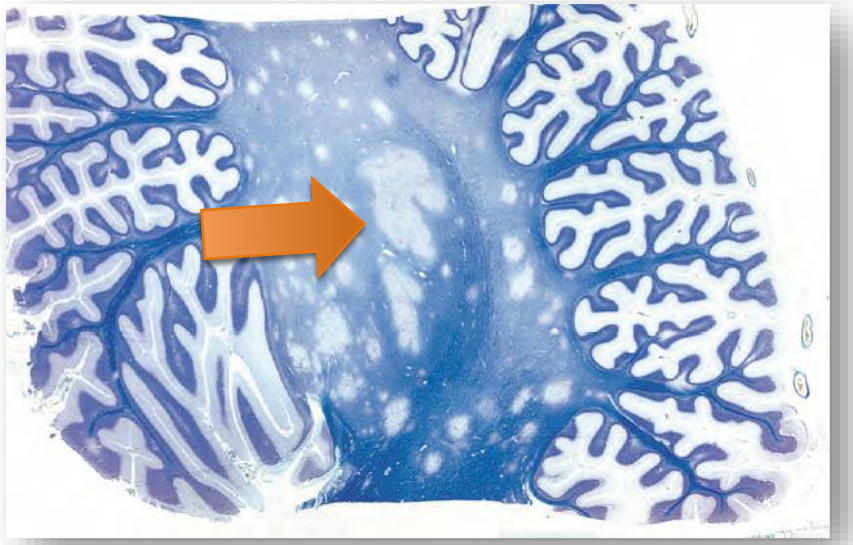
II.VI. JC virus / Progressive multifokale leukoenzephalopathie

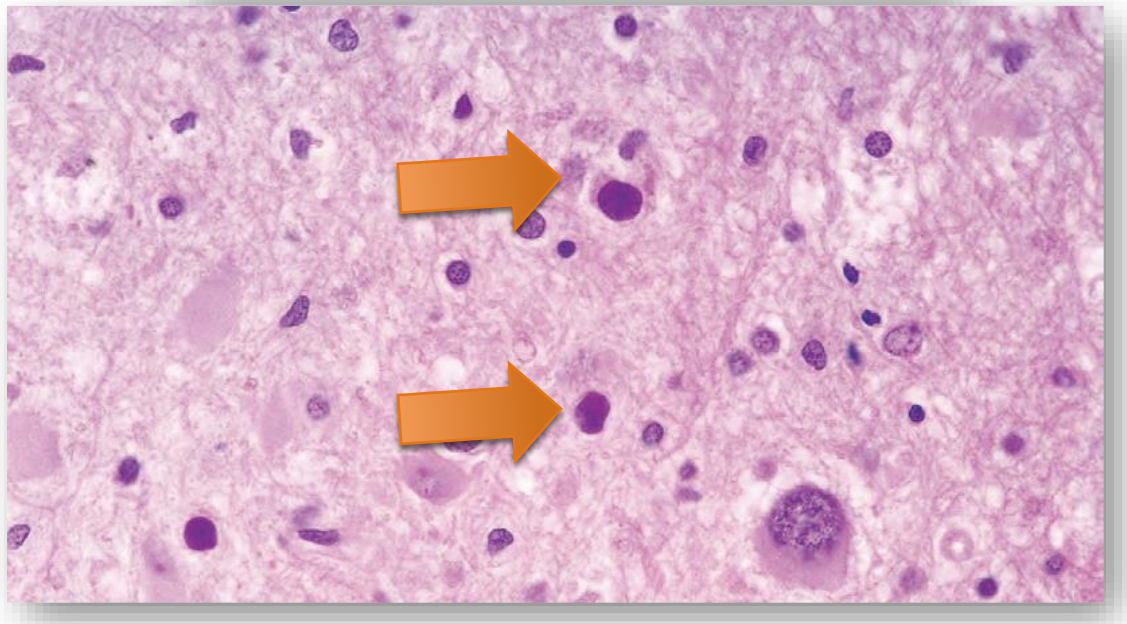
- Polyoma virus

- Infiziert oligodendrozyten
  - Demyelinisierung
  - Weisse Substanz – Hemispheren, Cerebellum

- Progressive neurologische symptomen

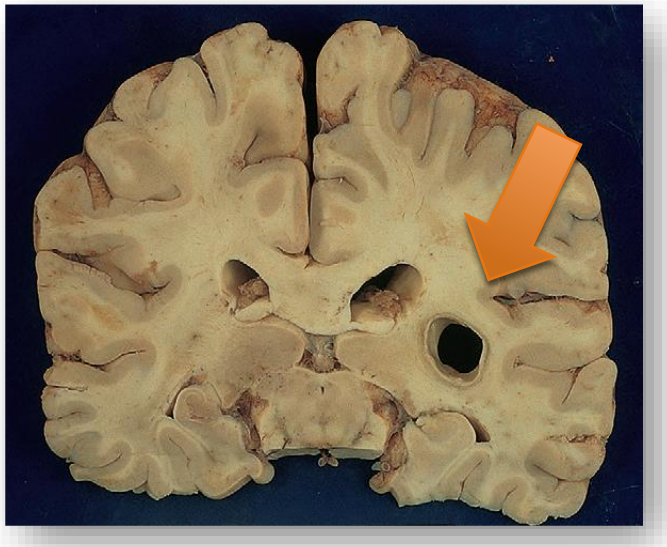


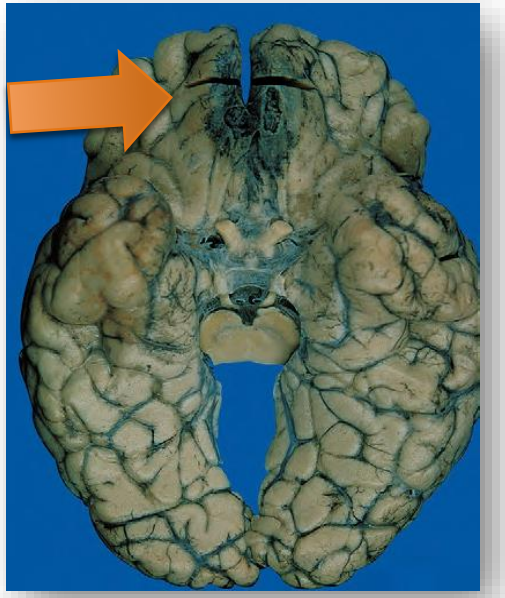




III. Mykotisch bedingte ZNS-Infektionen

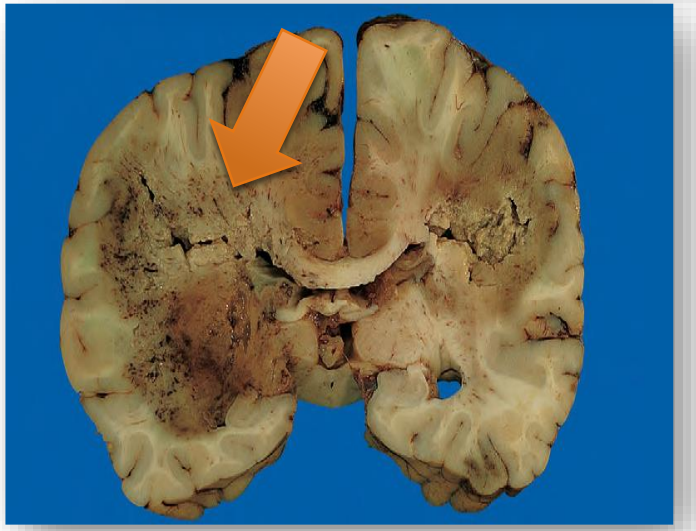
A. Candida Albicans
   - Multiplex microabscessen

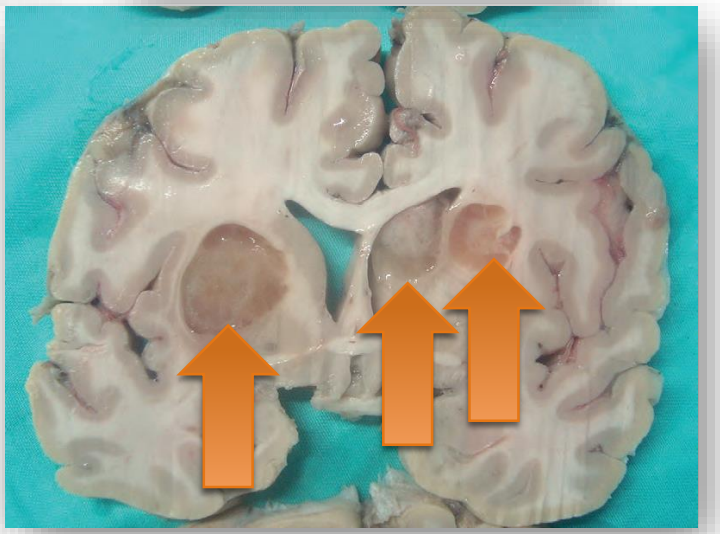




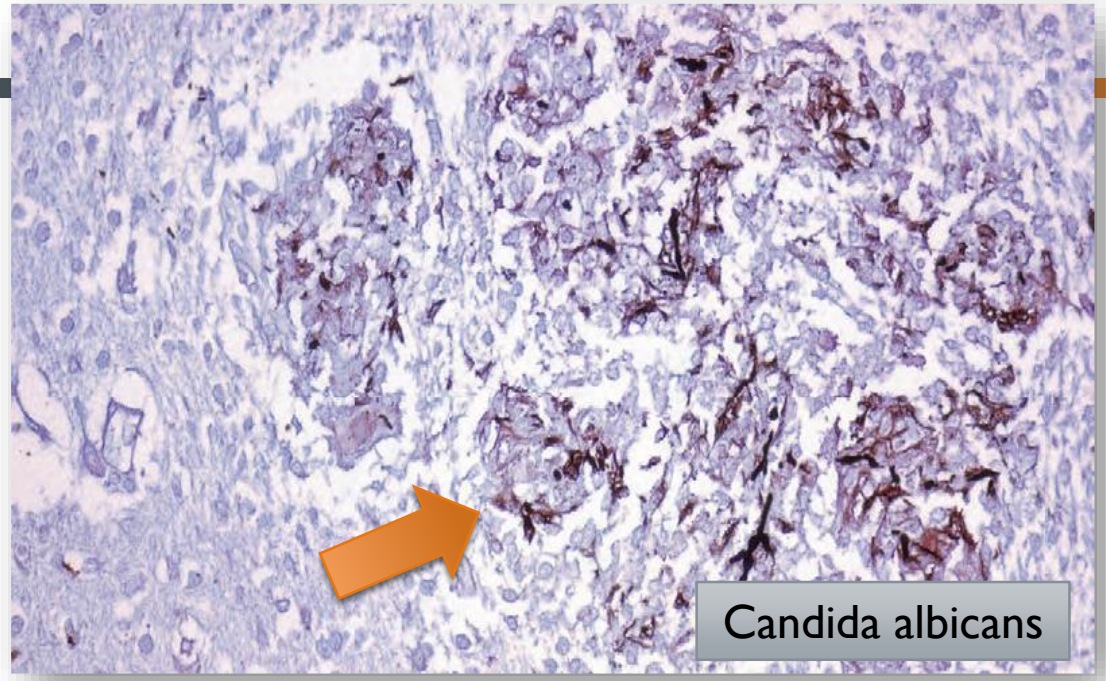
B. Mucormycose
   - Nasenhöhle, sinus Infektion
   - Fortgeleitete Infektion ,Vaskuläre Invasion

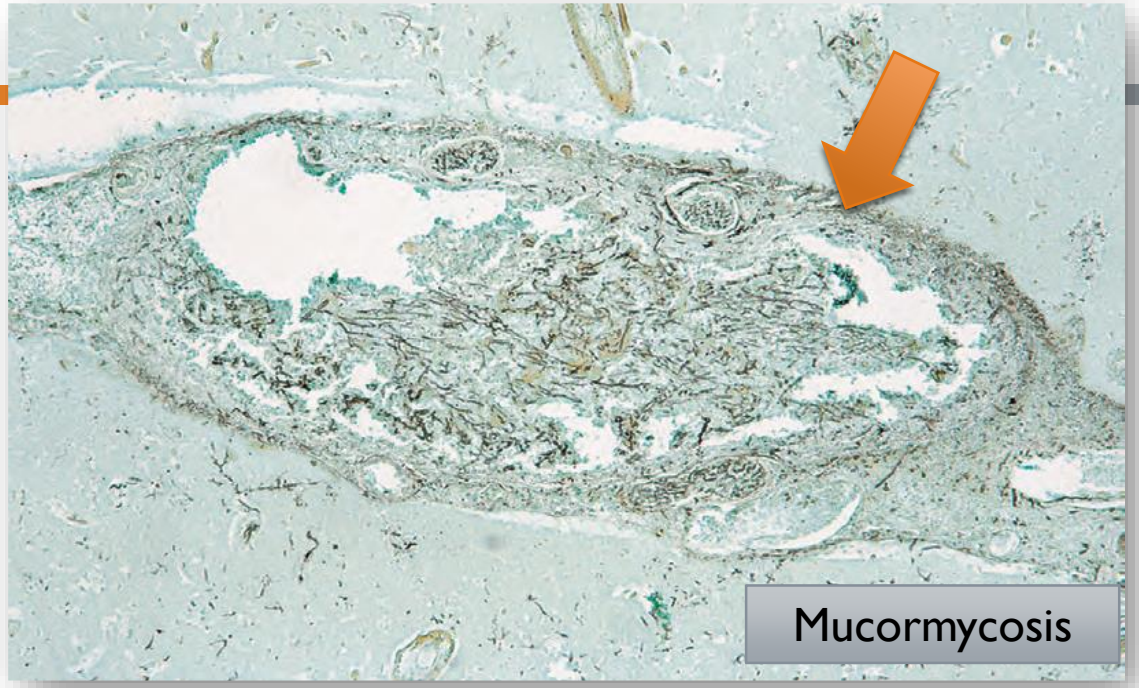
C. Aspergillus fumigatus
   - Hemorrhagische Infarkts
   - Vaskuläre Invasion

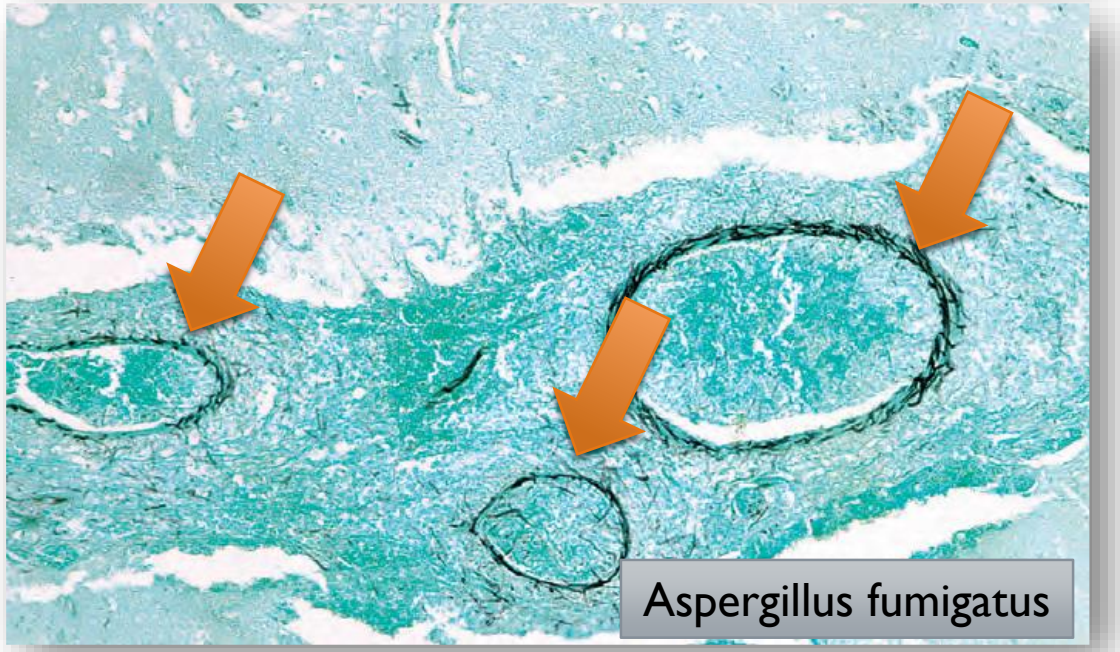


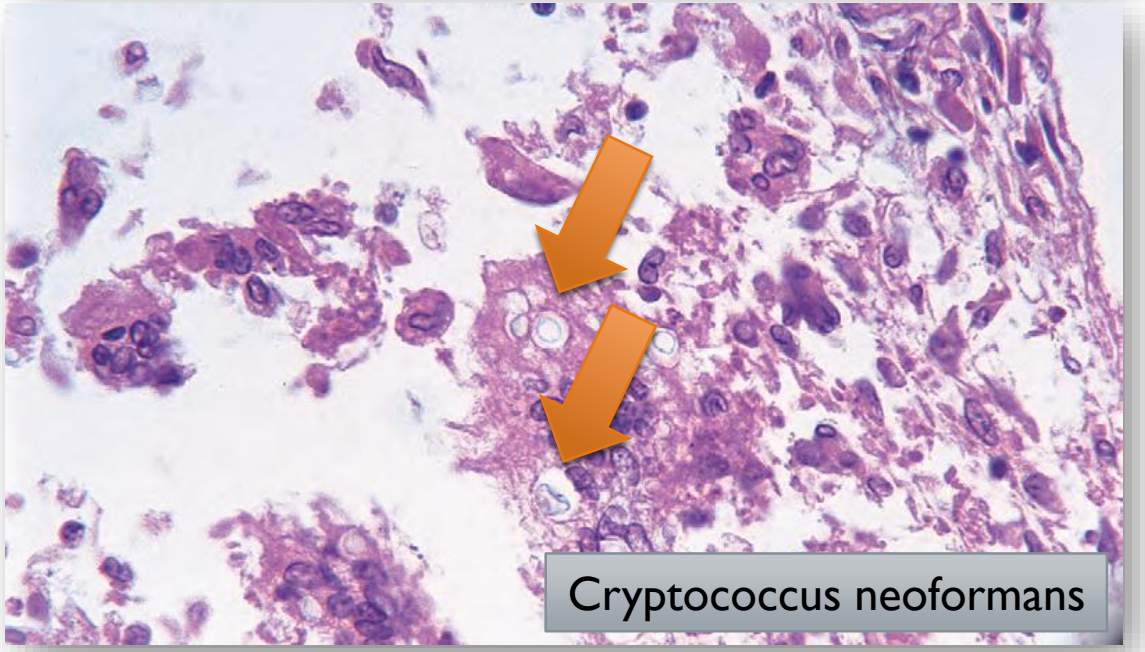


D. Cryptococcus neoformans
   - Meningitis, Meningoencephalitis
   - Fulminante


Candida albicans


Mucormycosis

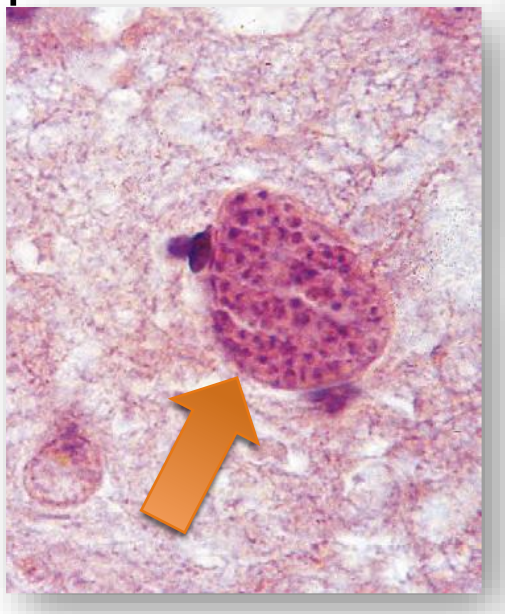

Aspergillus fumigatus

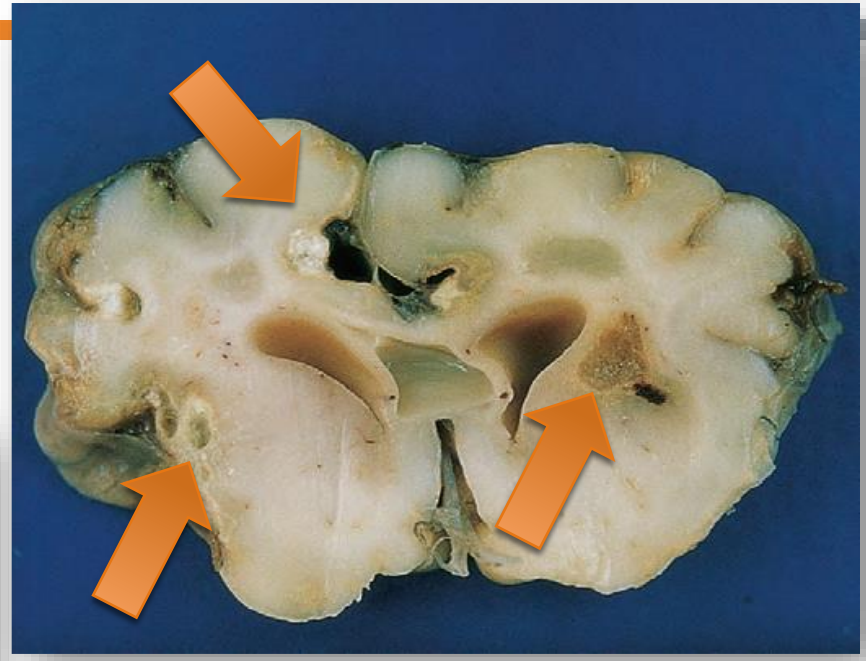

Cryptococcus neoformans

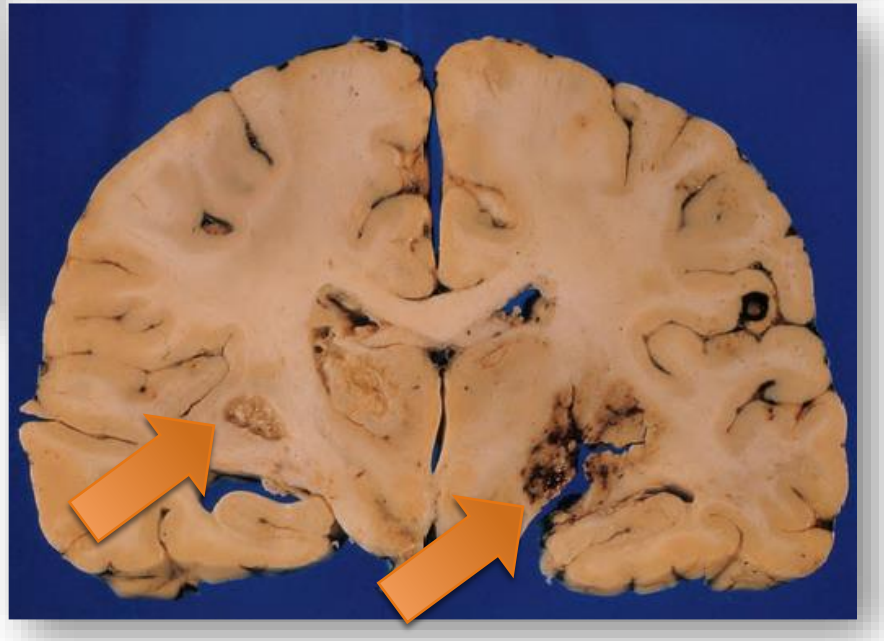
IV. protozoenbedingte ZNS-Infektionen- Toxoplasmose

- Toxoplasma gondii
  - Intermedier hosts - Menschen
  - Definitive host - Katze

A. Konnatale Toxoplasmose
  - Chorioretinitis
  - Hyprocephalus
  - Zerebrale Verkalkungen

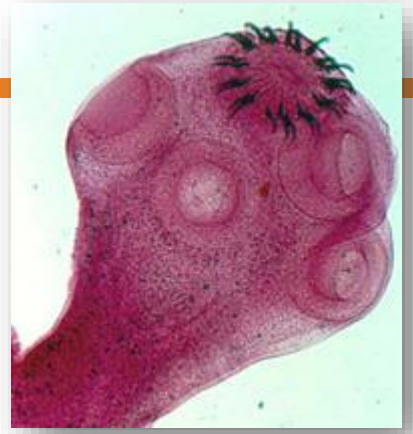






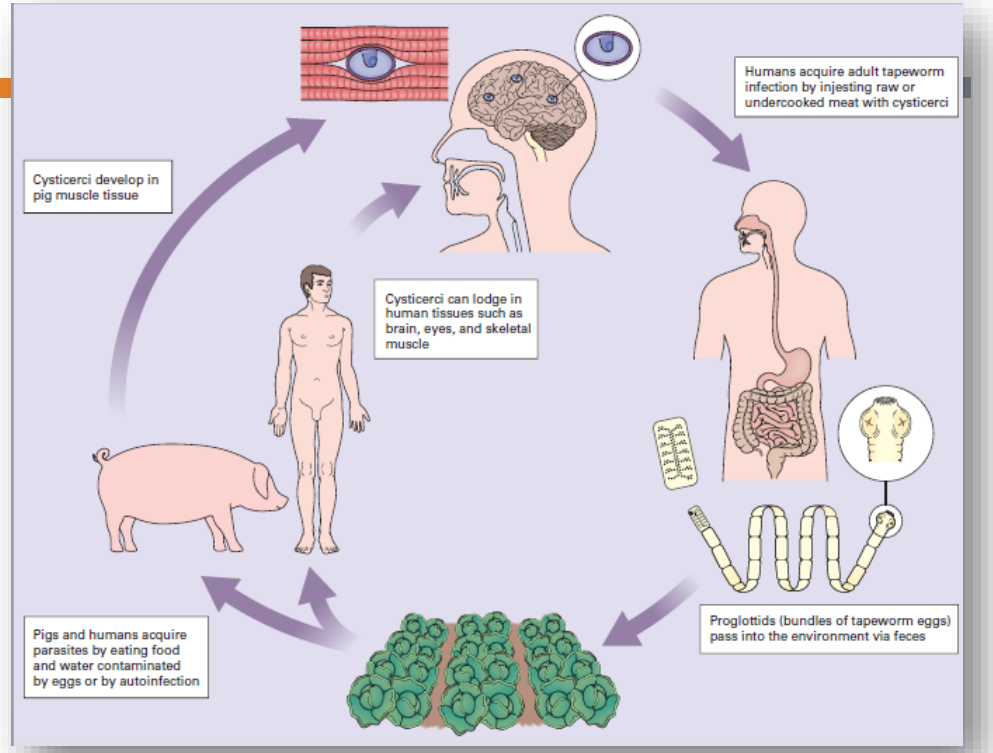
B. Infektionen im Erwachsenenalter
  - Immunsupprimierten Patienten
  - Subacute symptomen
  - Fokale-diffuze

IV. Parasitäre Infektionen

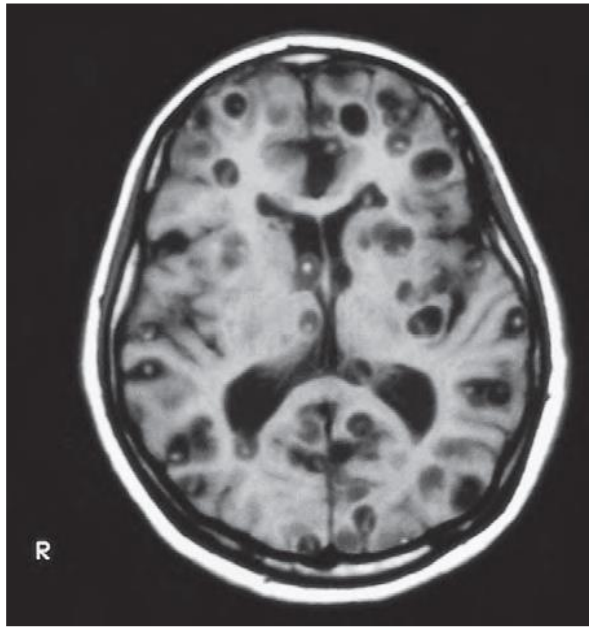


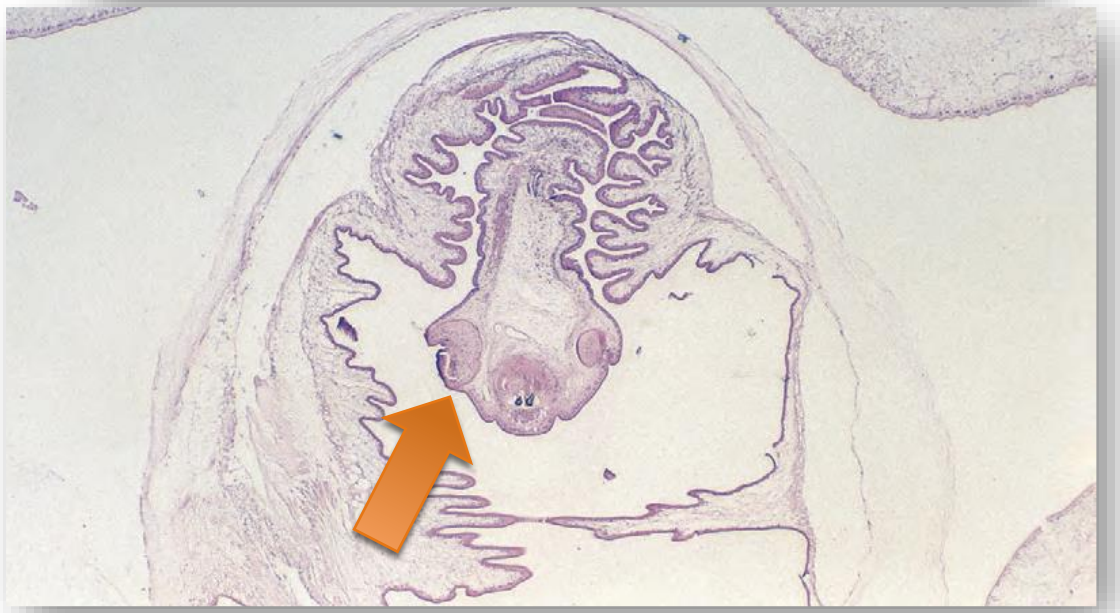
I. Tenia solium - Zystizerkose

- End-stadium
  - Larven verlassen den Gastrointestinal tract
  - Einkapseln – Hirn– Subarachnoideale Raum

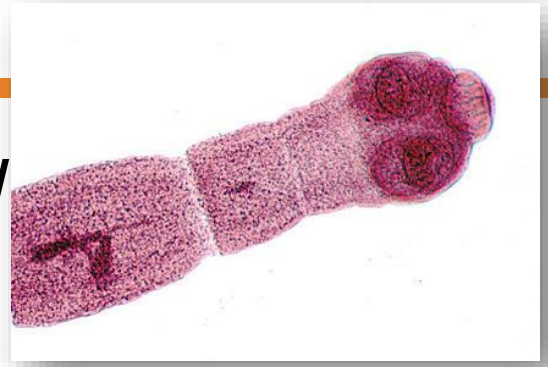


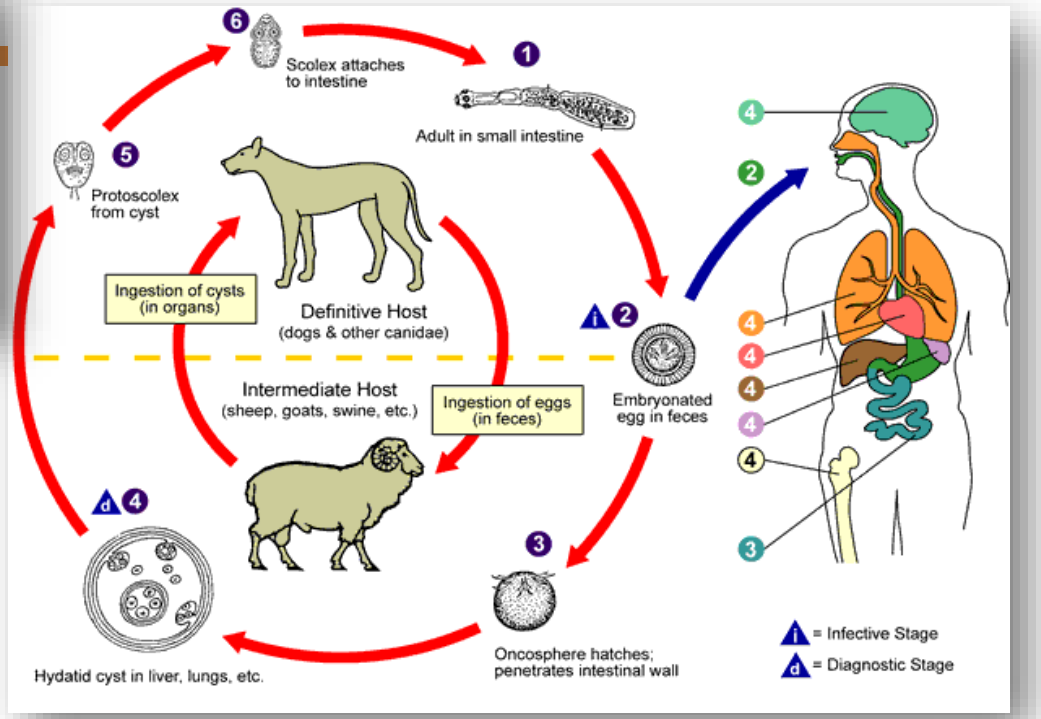
- Symptomen
  - Fokale
  - Epilepsy





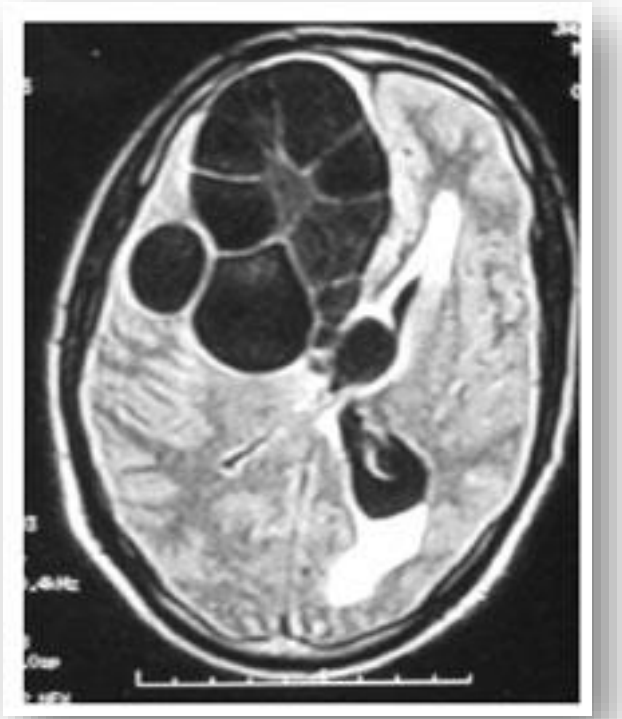
2. Echinococcus /Hydatidosis/

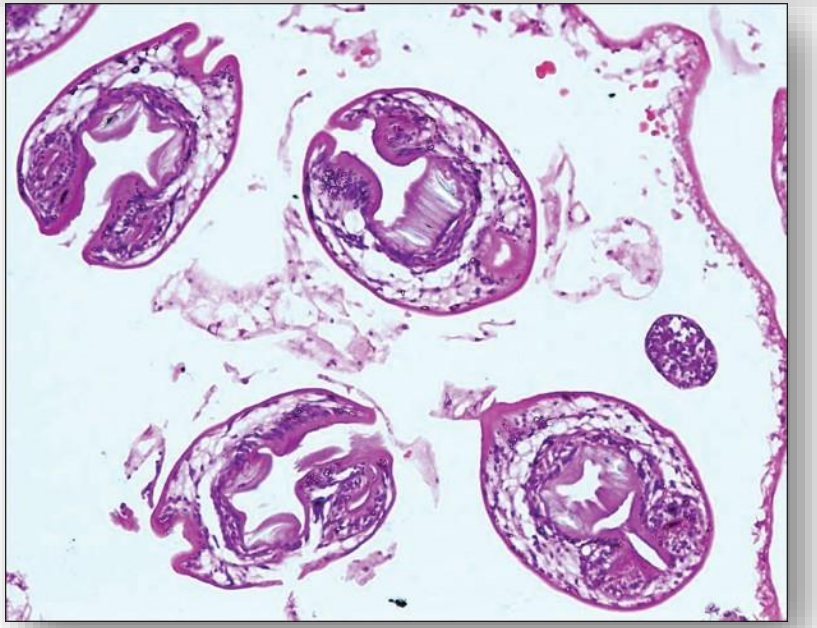




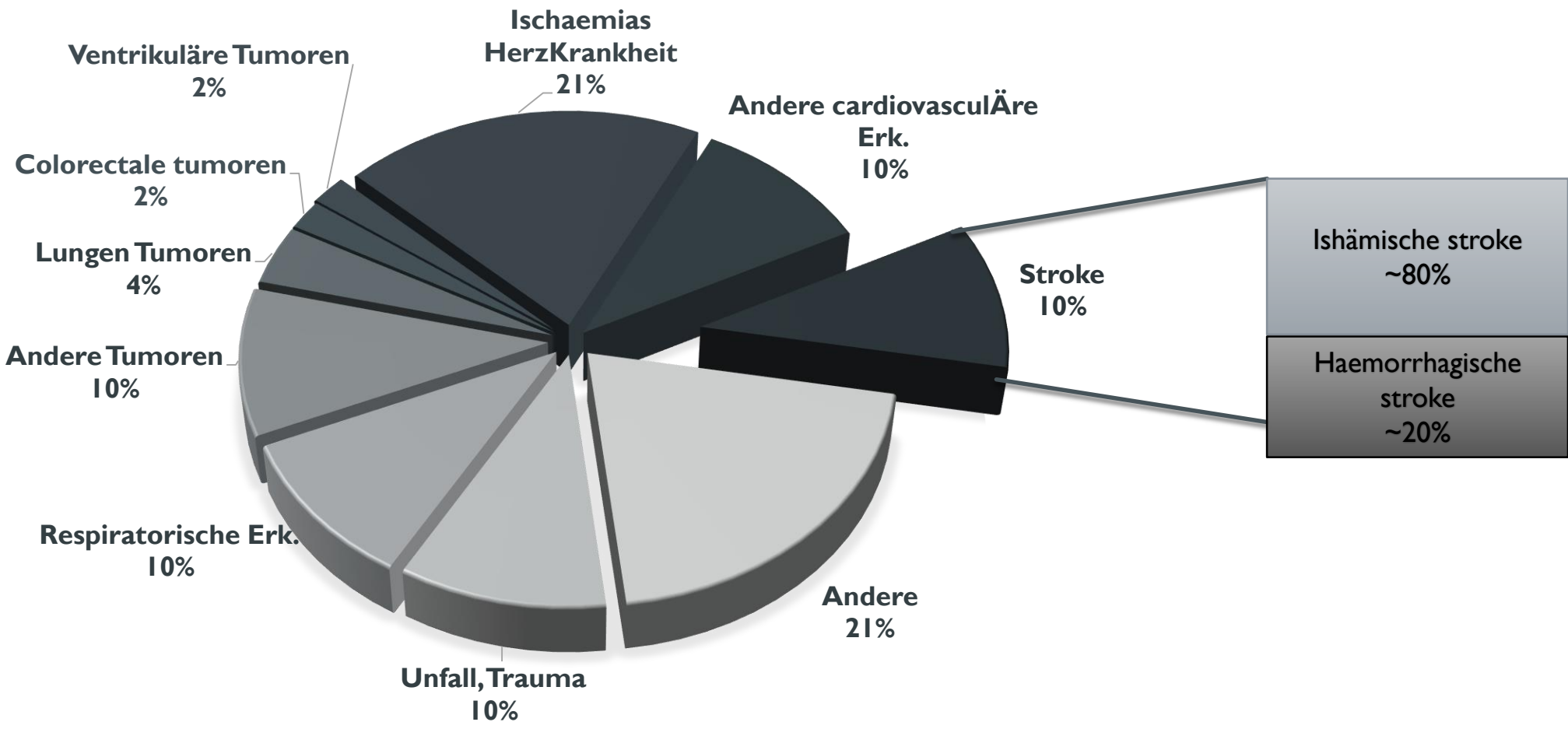
- Kindesalter
- Kontakt mitr Hunden
  - Einkalpseln sich– Leber, Lunge, Hirn

- Symptomen
  - Fokale
  - Epilepsy

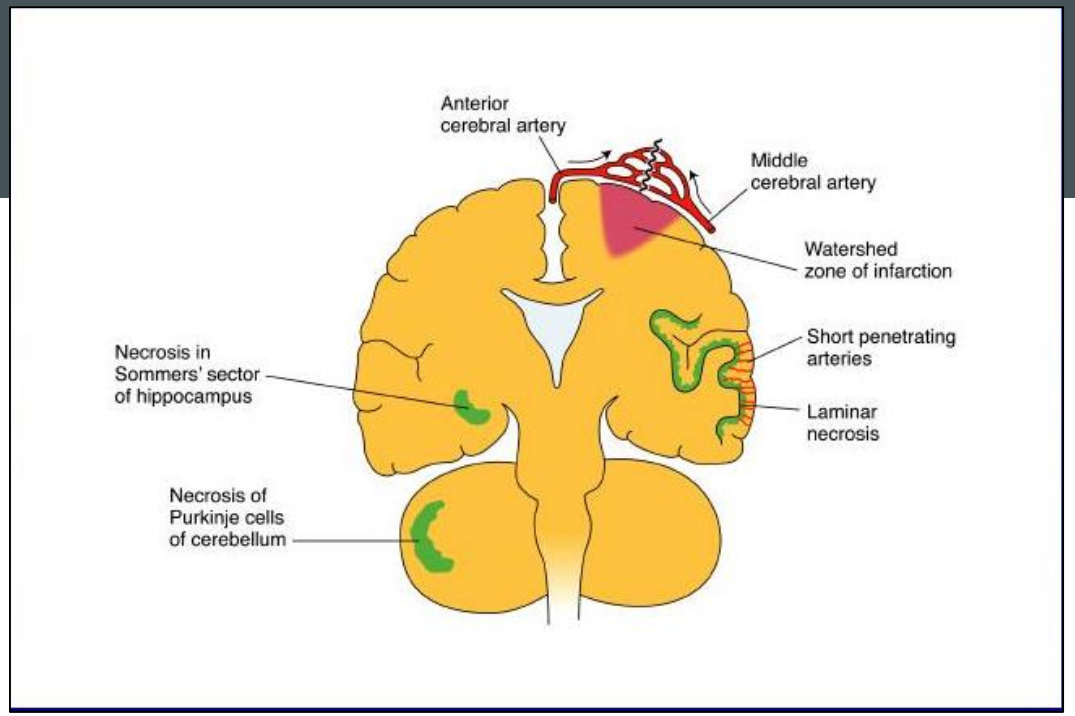




MORTALITÄT, EUROPÄISCHE KARDIOVASKULÄRE ERKRANKUNGEN, 2013



ZEREBRALE ISCHÄMIE

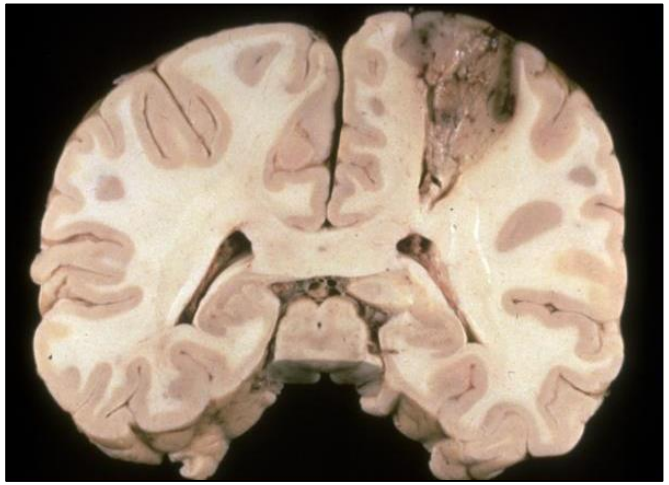


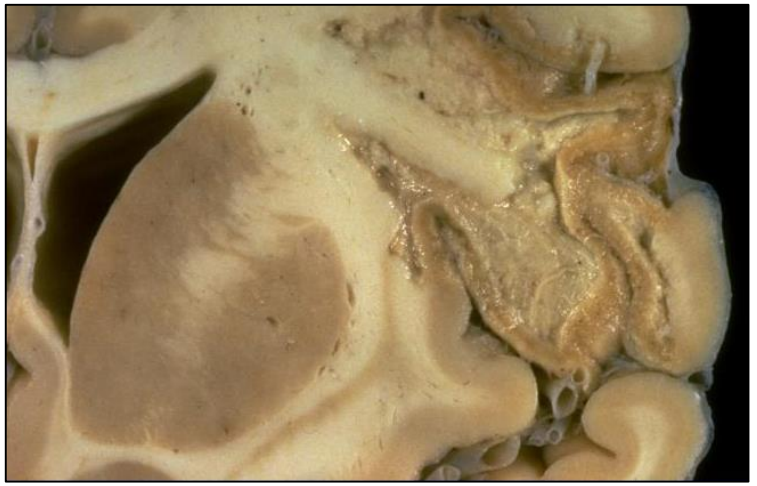
I. Globalen Ischämie

- Herzstillstand
- Schock

- diffusen ZNS-Schaden
- im Bereich der Grenzzonen der großen hirnversorgenden Arterien akzentuiert

Grenzzonen- oder Wasserscheideninfarkte



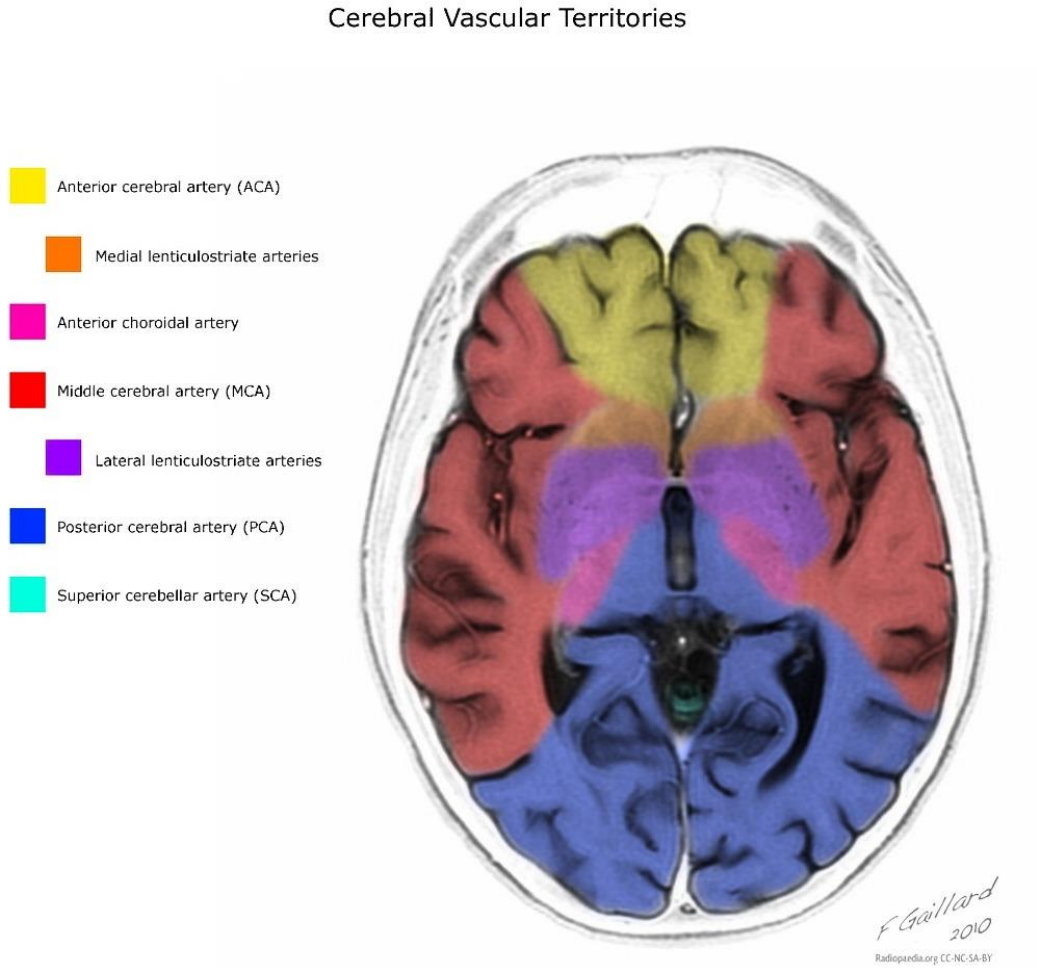


ZEREBRALE ISCHÄMIE

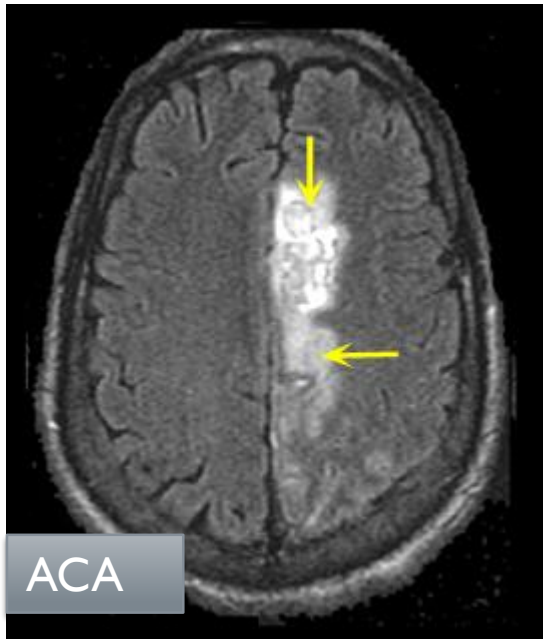
II. Lokalen Ischämie

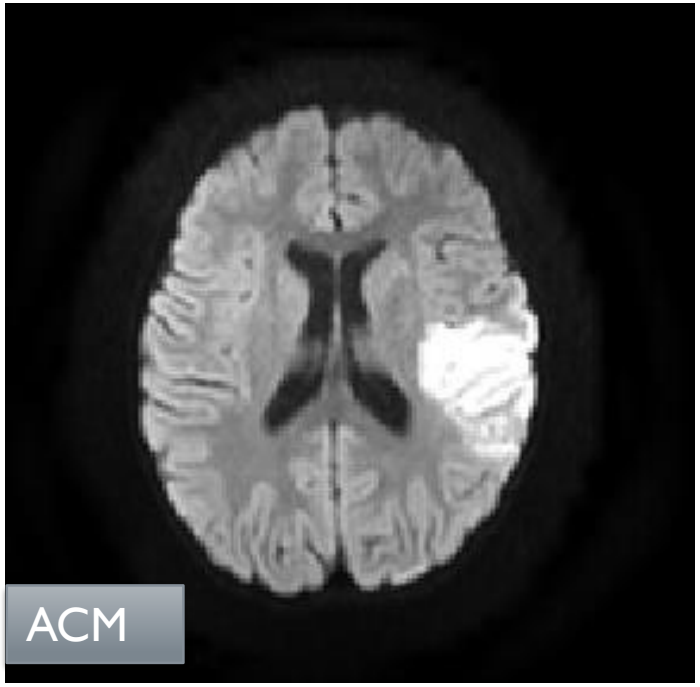
- Arteriosklerose
- Kardioembolie

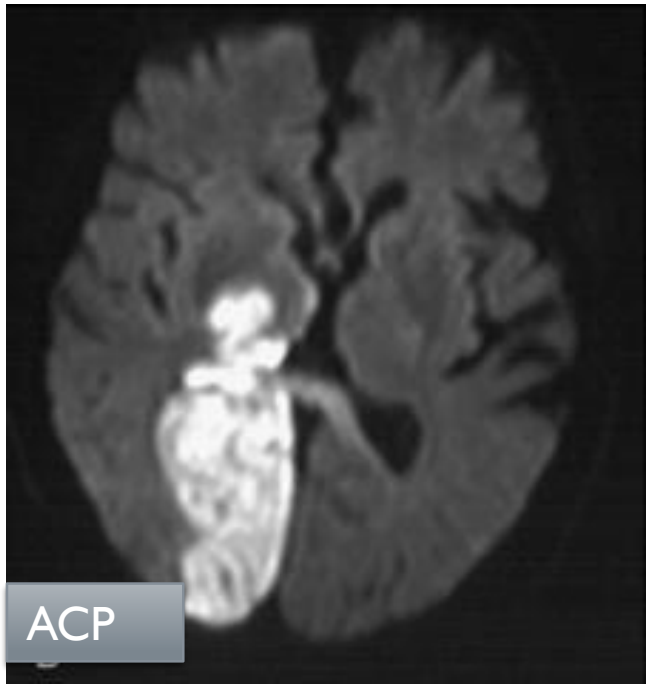
- arterielles Versorgungsgebiet komplett infarziert
  - **Territorialinfarkt**
- Verschluss kleinerer Arterienäste
  - **lakunäre Infarkte**



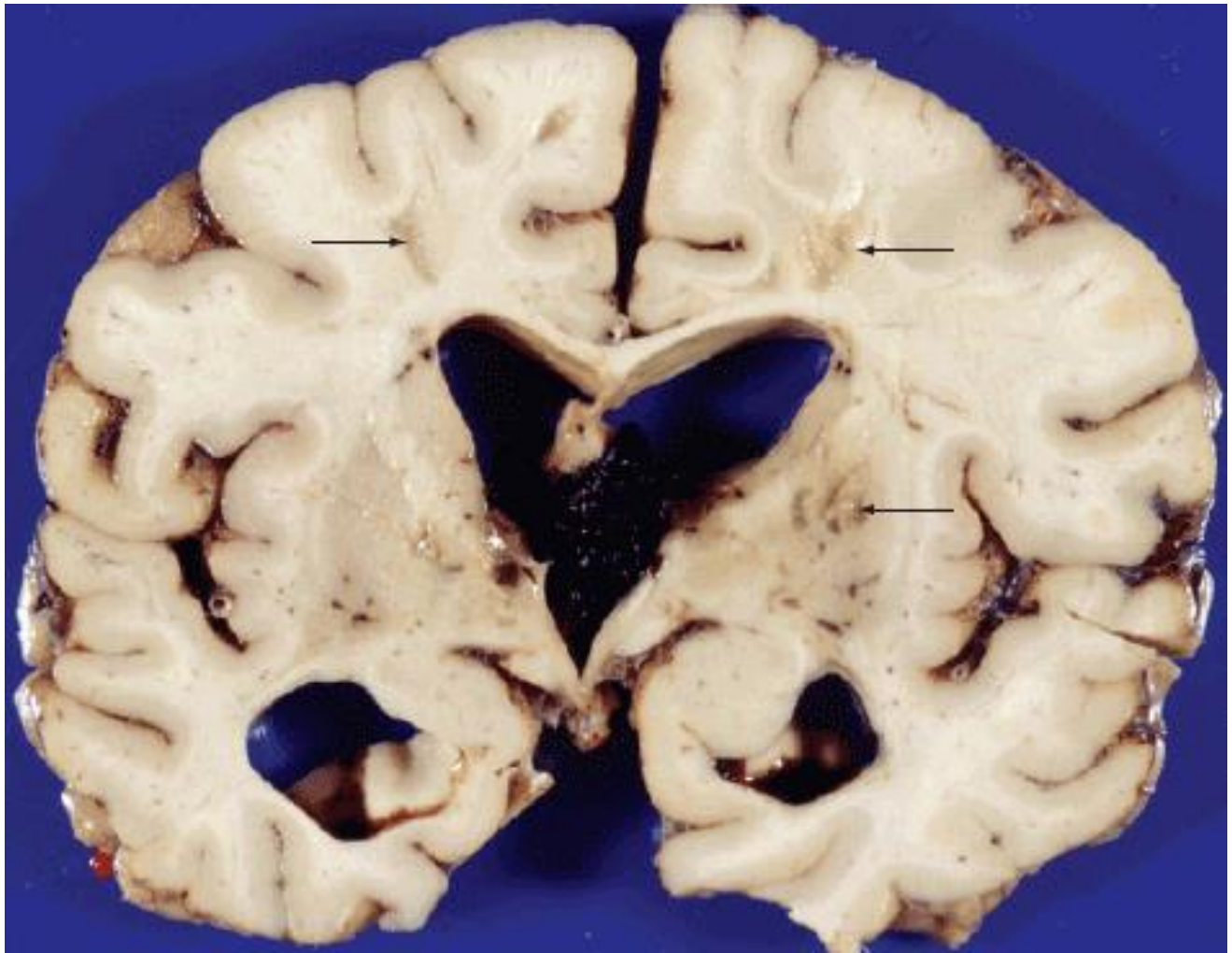
ZEREBRALE ISCHÄMIE - TERRITORIALINFARKT





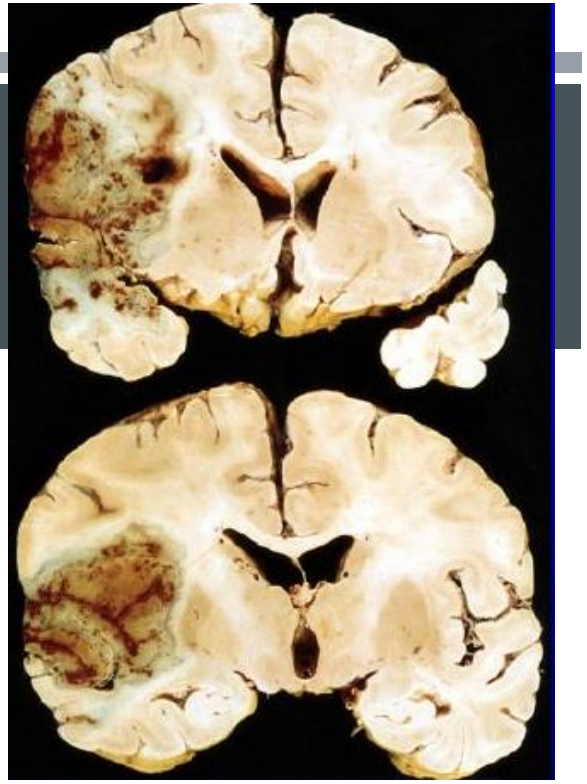


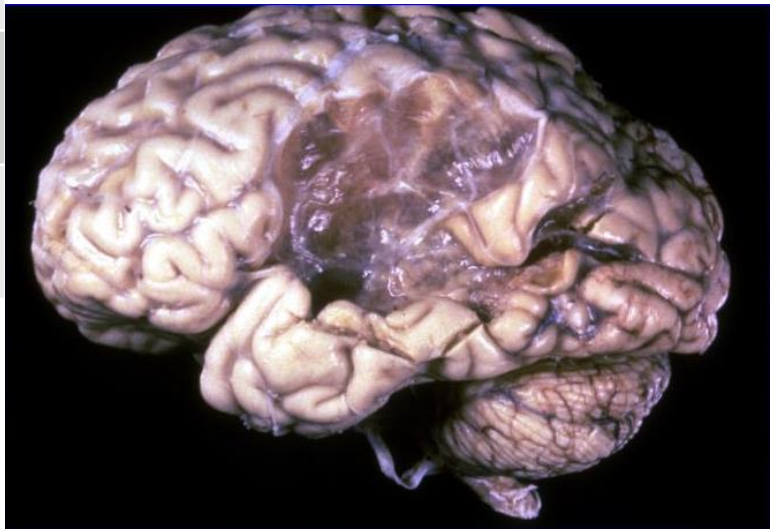
ZEREBRALE ISCHÄMIE - LAKUNÄRE INFARKTE



ZEREBRALE ISCHÄMIE - MORPHOLOGIE

Zeit	Makroskopisch	Mikroskopisch
Minuten	Keinen	Eosinophilie und Pyknose von Neuronen
Stunden -Tagen	Gewebsödem	Einwanderung von Neutrophilen, eine Mikrogliaaktivierung sowie ein vermehrtes Auftreten von Makrophagen („Schaumzellen“)
2 Wochen	Gewebsödem, Gewebeverflüssigung	Astrozytenaktivierung lipidbeladene Makrophagen
Monate-Jahren	Ausbildung einer oft zystischen Läsion	Fasergliose





INTRAKRANIELLE BLUTUNGEN

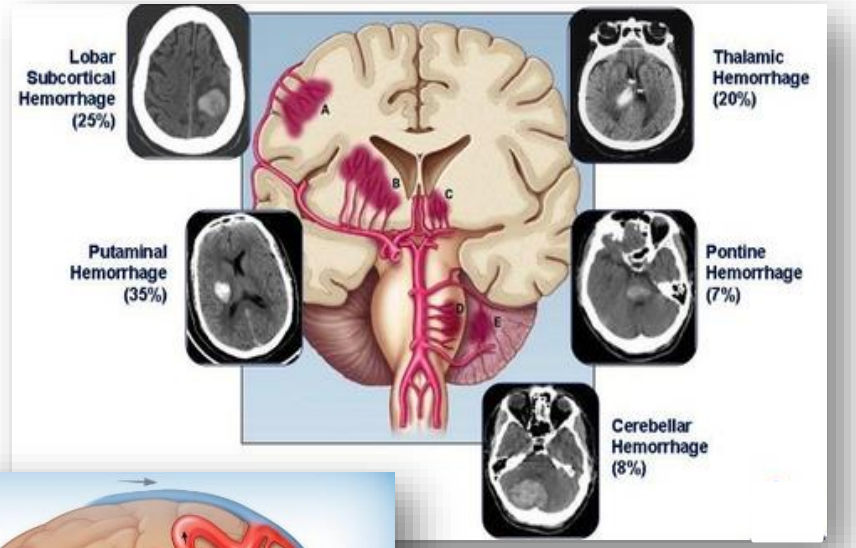
Etiologie:

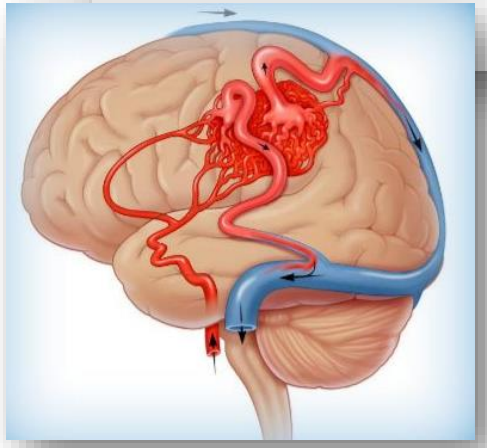
I. Schädigung von Hirngefäßen
   - Hypertonie
   - Abnormale Protein Ablagerung
   - Trauma

II. Gefäßmalformationen

III. Tumor

IV. Hämatologische Erkrankungen





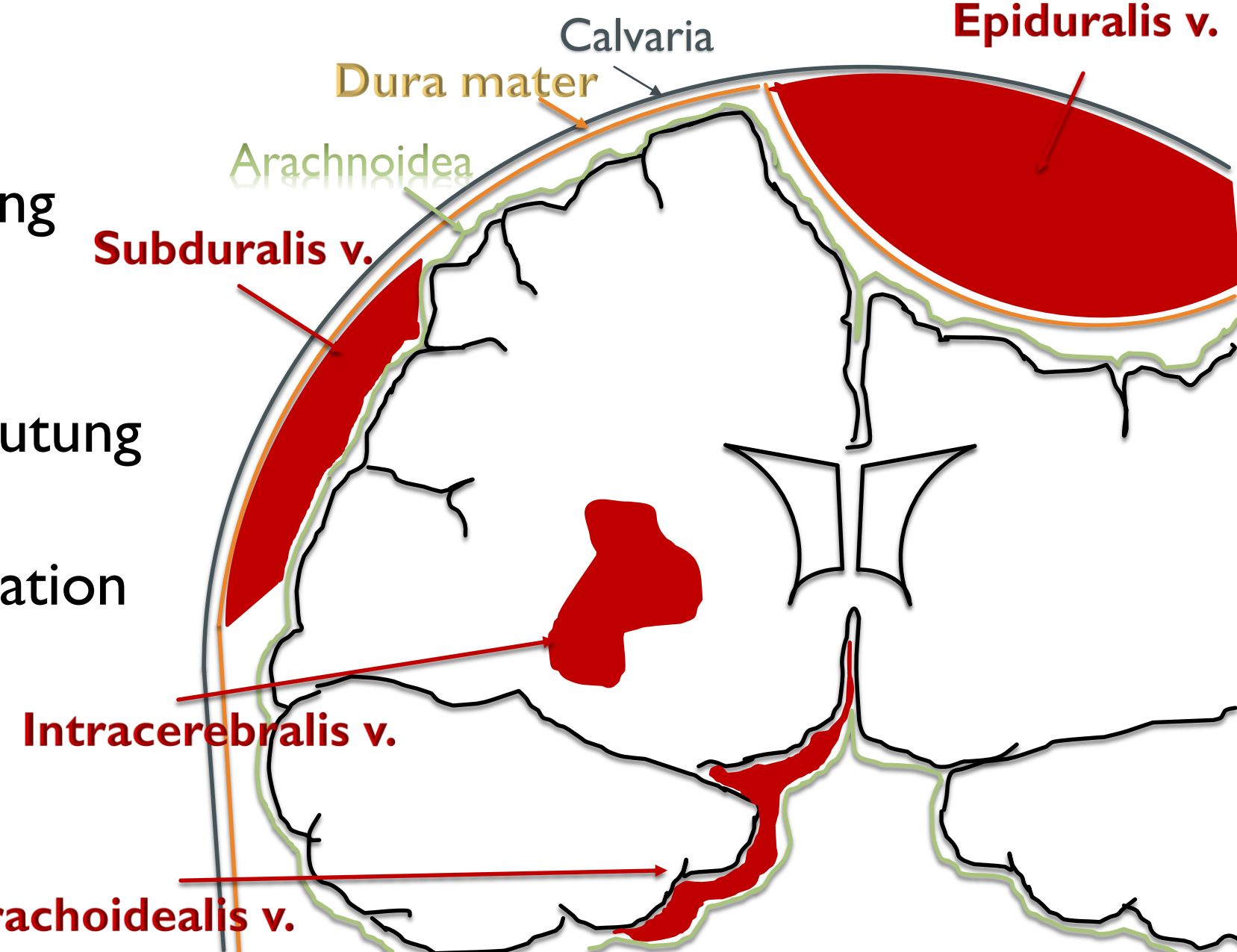
Lokalisierung:

I. Intrazerebrale Blutung
   - Hypertonus
   - Tumor

II. Subarachnoideale Blutung
   - Aneurysma
   - Vascular malformation

III. Subdurale Blutung
   - Trauma

IV. Epidurale Blutung
   - Trauma

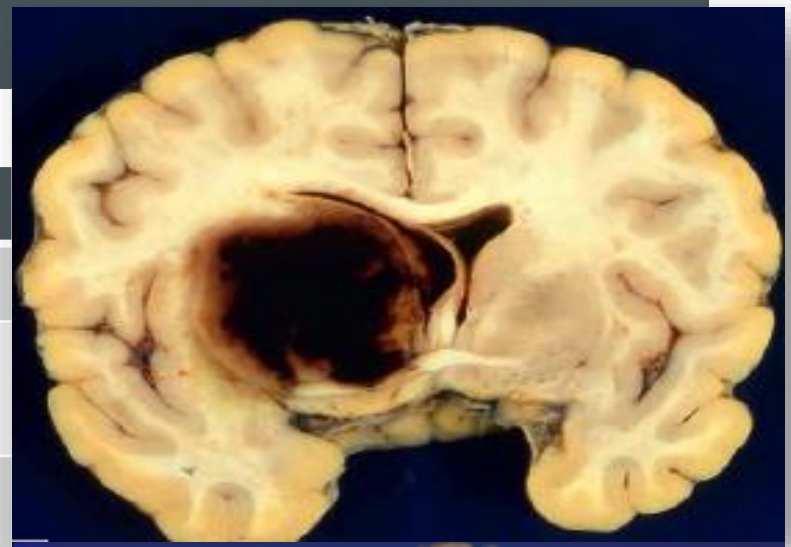


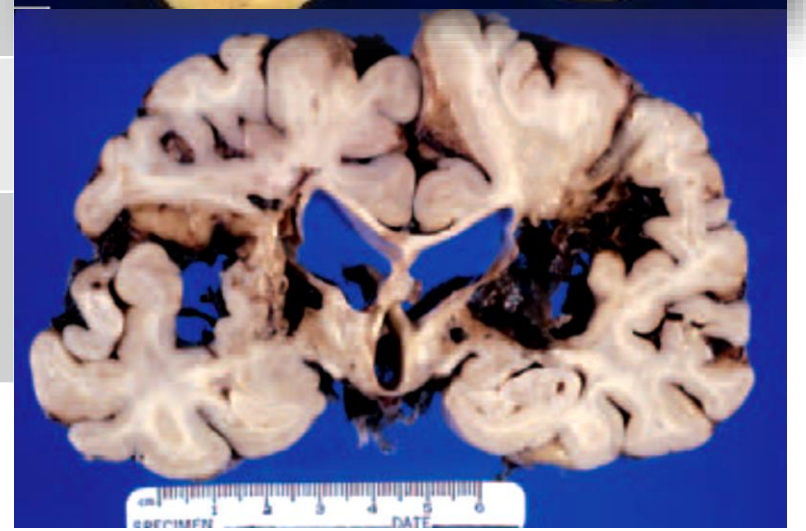
I. Intrazerebrale Blutung

1. Hypertonus ~ 50%
2. Cerebral amyloid angiopathie ~10-15%
3. Tumor ~8-10%
4. Gefäßmalformationen ~5%
5. Trauma
6. Gerinnungsstörungen

INTRAZEREBRALE BLUTUNG - MORPHOLOGIE

Zeit	Makroskopisch	Mikroskopisch
Minuten	Hematoma	Extravasation
Stunden	Perifokale oedema, Kompression	Erythrozyten lysis Oedema, ischaemia
2-3 Tagen-Wochen	Bräunliche Hematoma	hämosiderin beladene makrophagen Astrozytose
Wochen - Monaten	Gebrechliche Hematoma	Organisation Phagozytose
Monaten - 1 Jahr	Ausbildung einer zystischen Läsion, mit Bräunliche Liquor	Zystischen Läsion hämosiderin beladene makrophagen





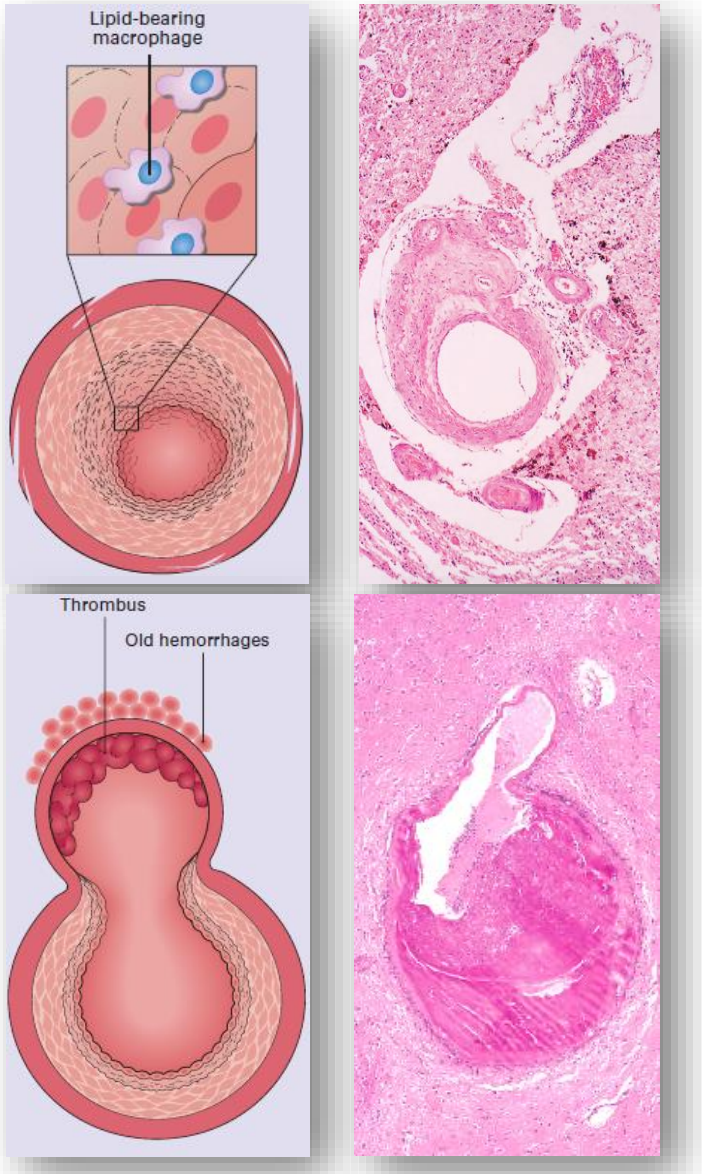
1. Hypertensiv bedingte intrazerebrale Blutungen

Strukturellen Veränderungen kleiner Blutgefäße

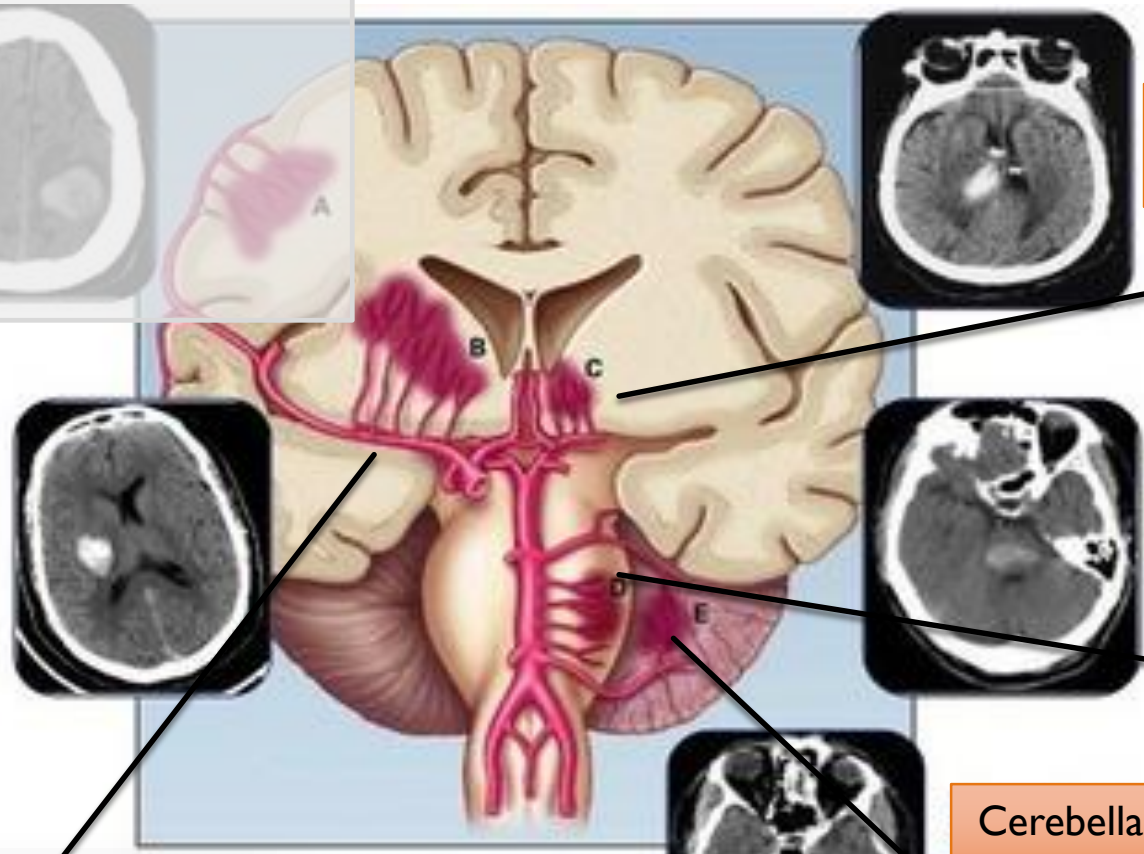
- Verenderungen von glatten Muskelzellen

- Fragmentation von Lamina elastica

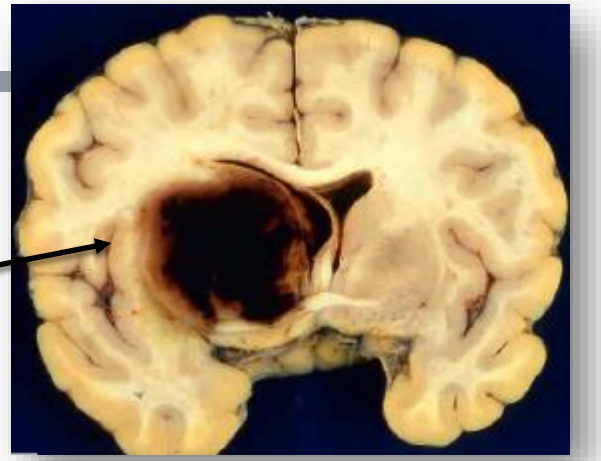
- Fokale Erveiterung von Gefäßvand
  - Charcot-Buchard microaneurysm



Lobaris
Subcorticalis
25%

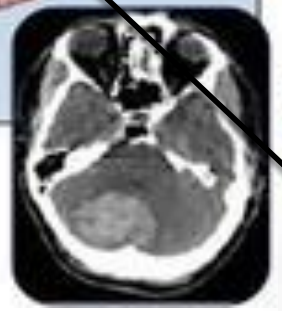


Thalamus
20%

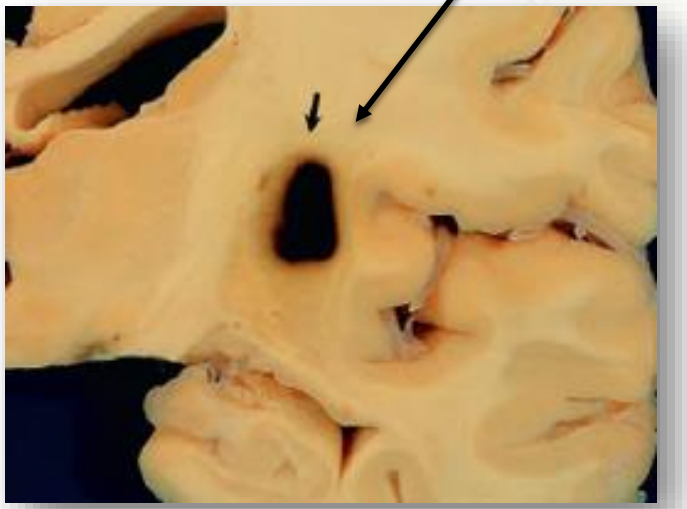


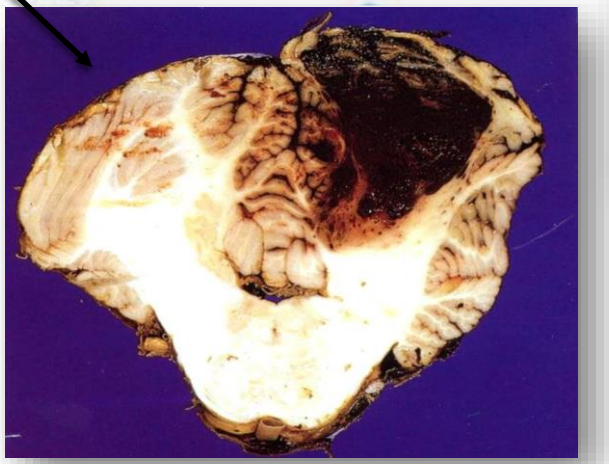
Putamen
35%

Pons
7%



Cerebellaris
8%

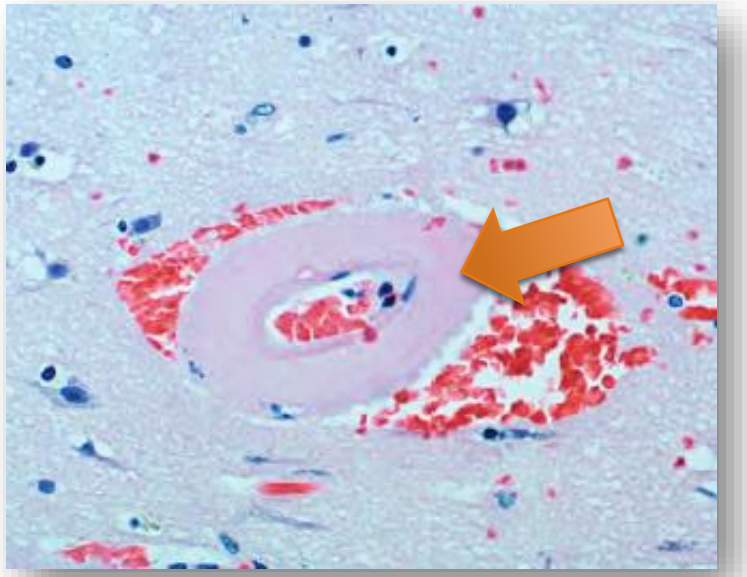


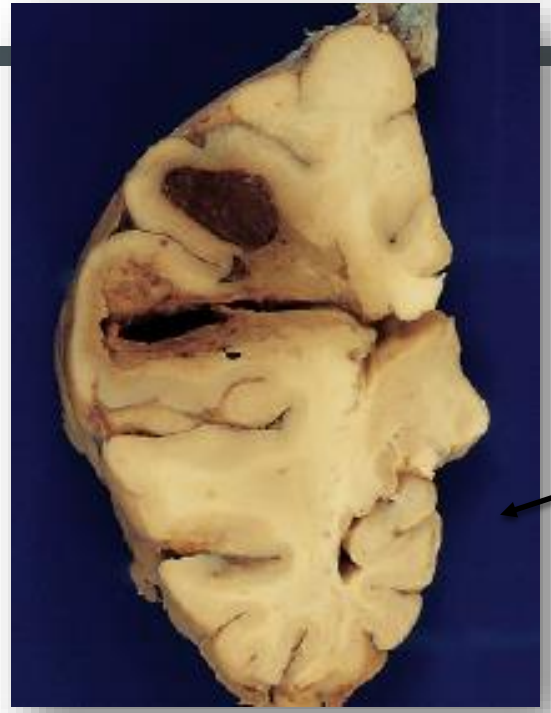


11. Zerebrale Amyloidangiopathie (CAA)

1. Sporadisch/Senil CAA
   - Mit Alzheimer-Demenz
   - Ablagerung von Beta-Amyloid(Aβ) in den Wänden der Blutgefäße

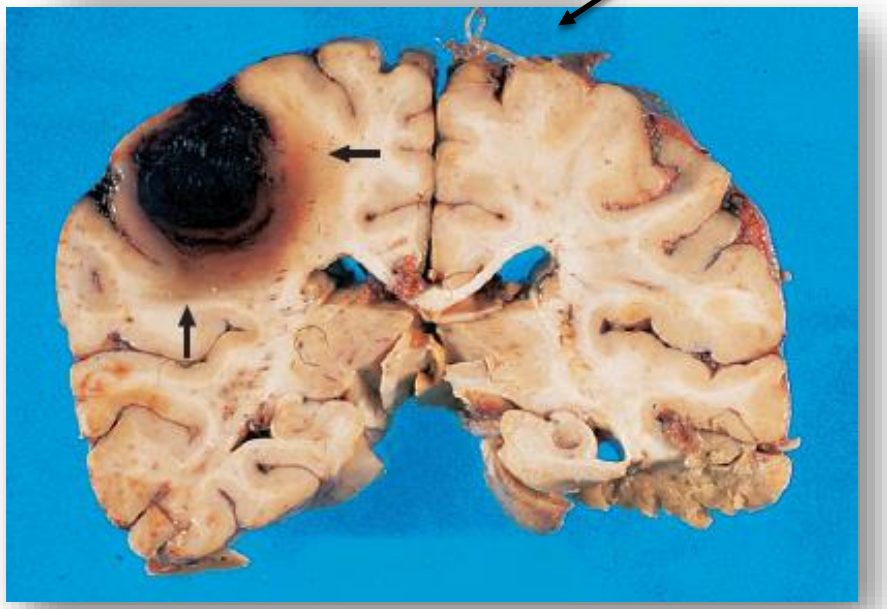
2. Hereditär CAA
   - im jüngeren Alter
   - AD
   - Amyloid precursor protein mutation



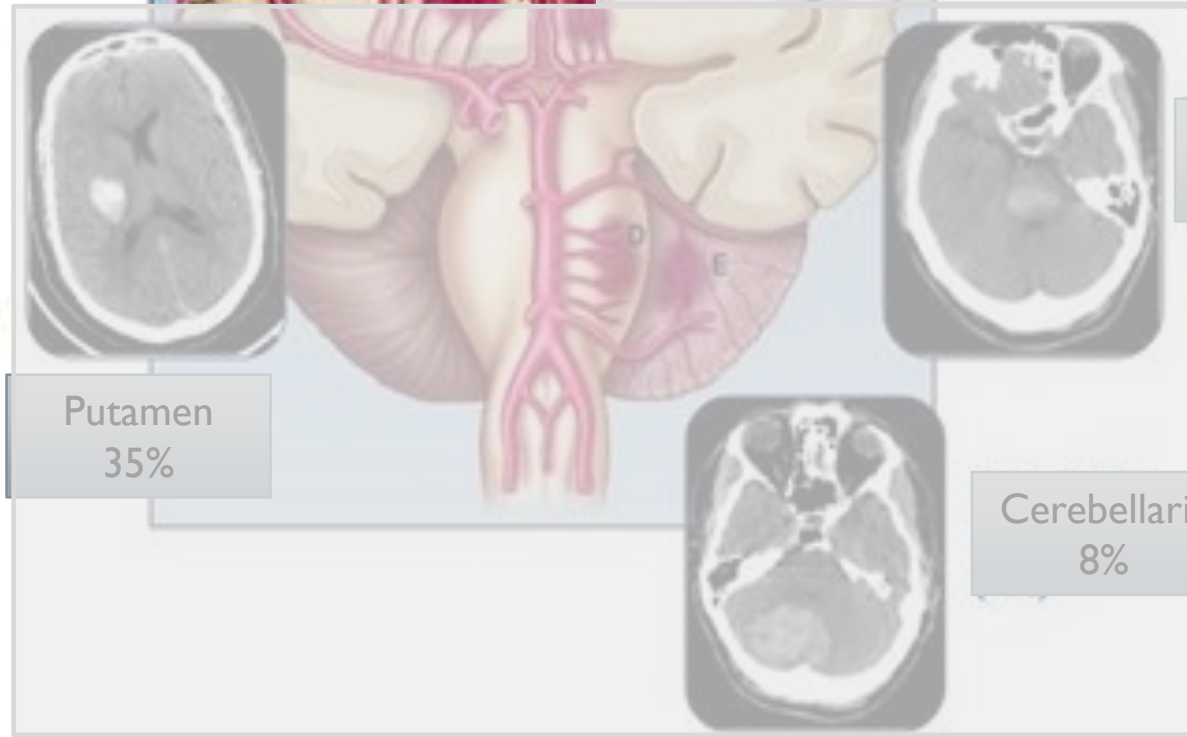


Lobaris
Subcorticalis
25%

Thalamus
20%



Putamen
35%



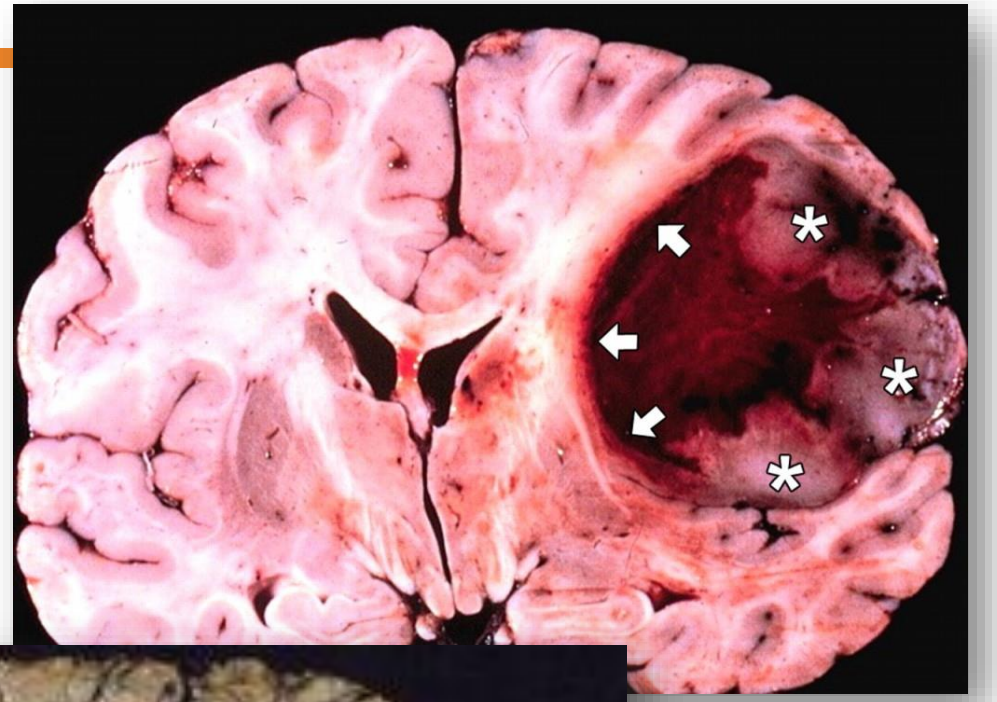
Pons
7%

Cerebellaris
8%

III. Tumor

1. Primär ZNS Tumor
   - Glioblastoma

2. ZNS Metastasis
   - Melanoma malignum
   - Urothelial cc.



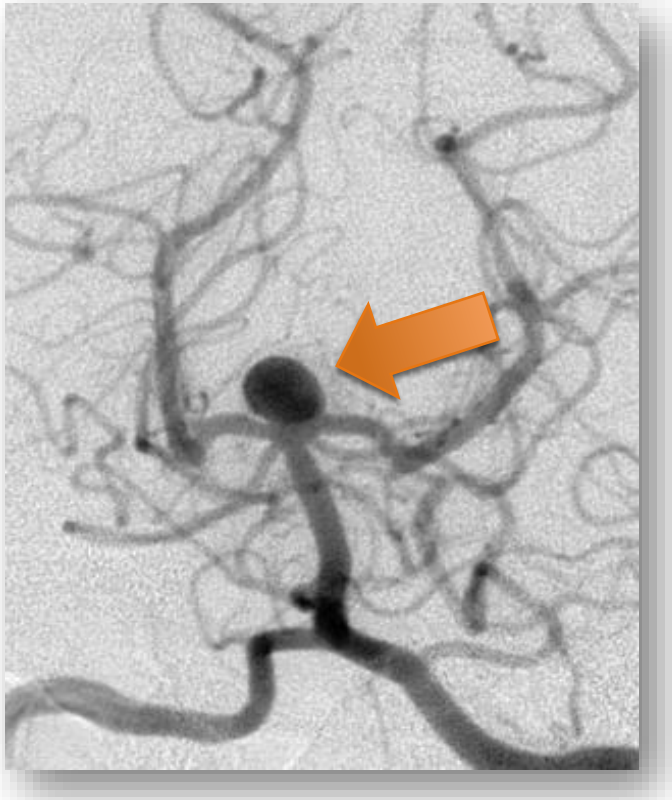
II. Subarachnoidale Blutung

1. Aneurysma
    - Sakkuläre /berry/ aneurysmen
    - Fusiforme /atherosclerosis/ aneurysmen
2. Gefäßmalformationen
3. Trauma

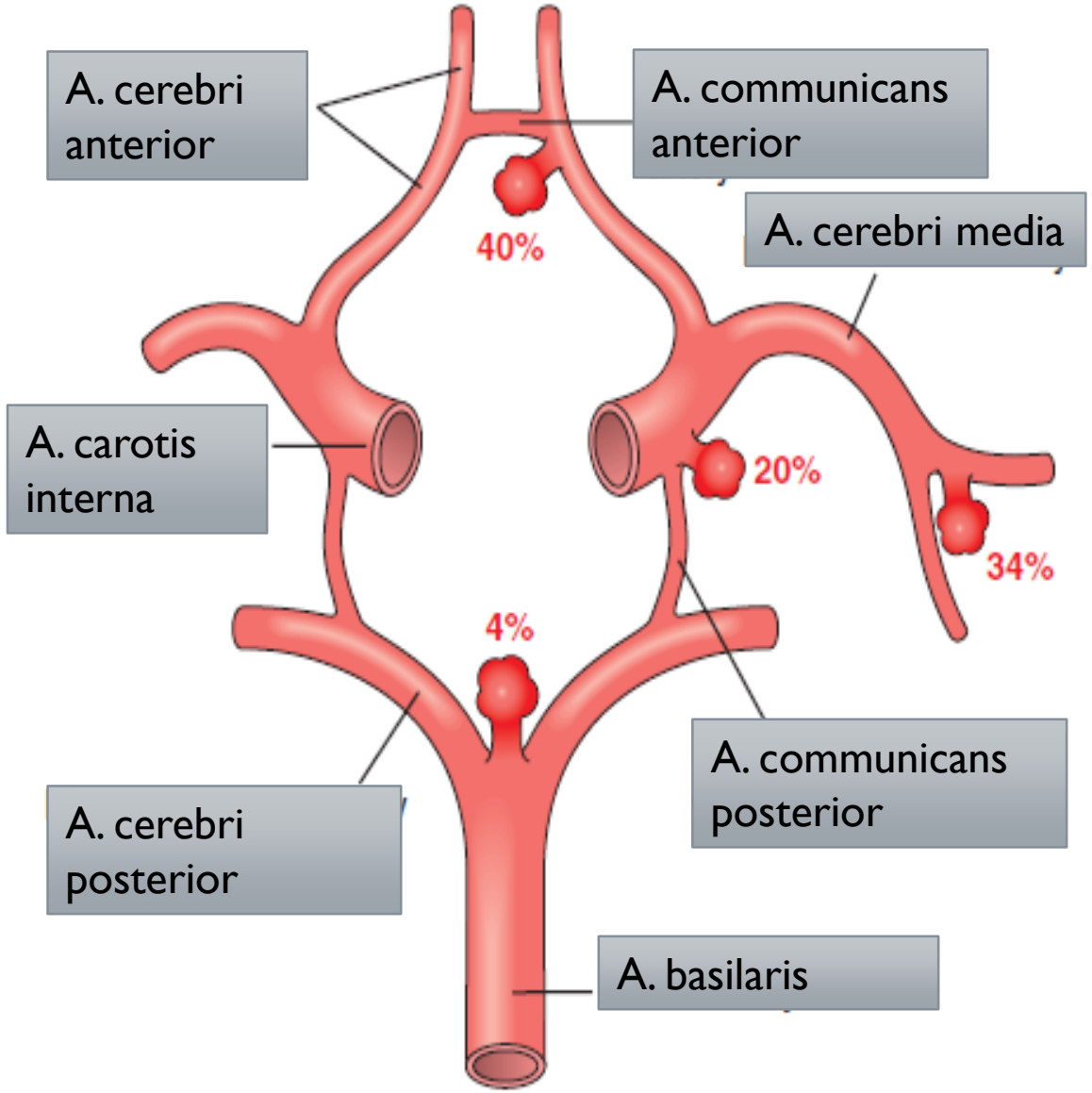
1. Gefäßwandaneurysmas

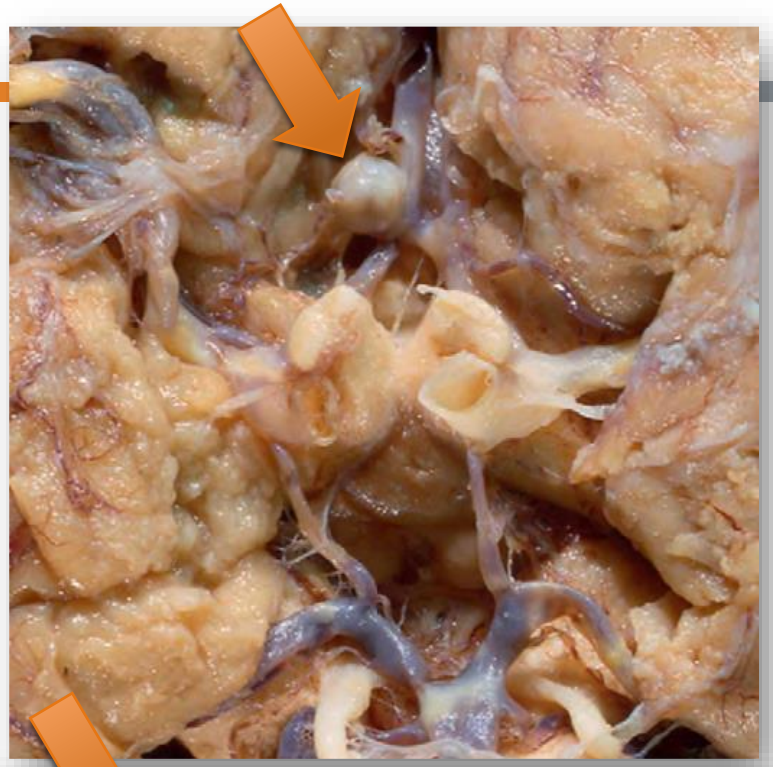
1.Sakkuläre /Berry/ aneurysmen

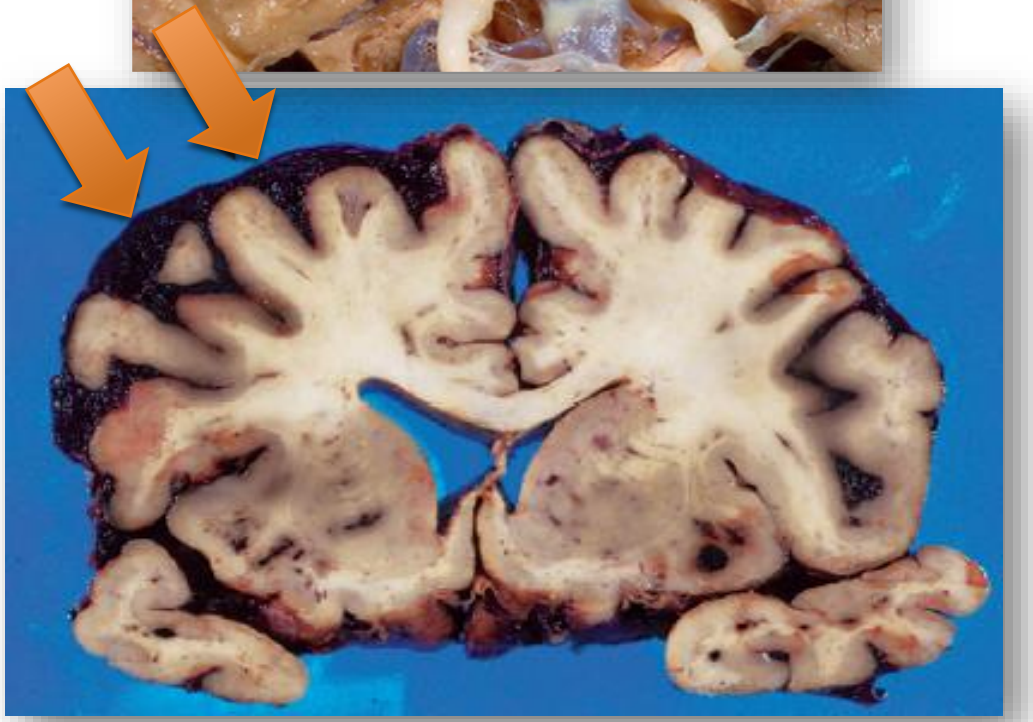
- Kongenitale – strukturellen Gefäßwandanomalien
- Verzweigungsstellen– Circulus Willisi



- Prädisponierende faktoren
  - Bluthochdruck
  - intrakranielle Druckerhöhungen
  - angeborene Störungen der Kollagensynthese
  - Polyzystische Nierenerkrankung


A. cerebri anterior
A. communicans anterior
A. cerebri media
A. carotis interna
A. cerebri posterior
A. communicans posterior
A. basilaris
40%
20%
34%
4%

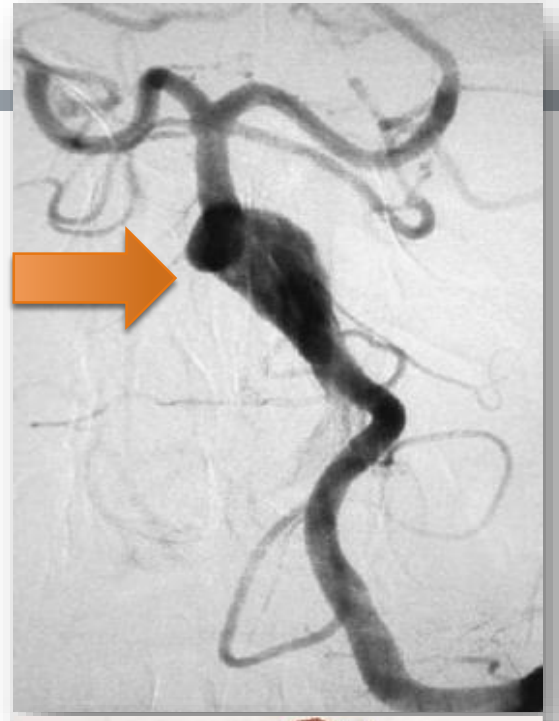




II. Fusiform aneurysmen

- Atherosclerose
  - A. basilaris und A. vertebralis

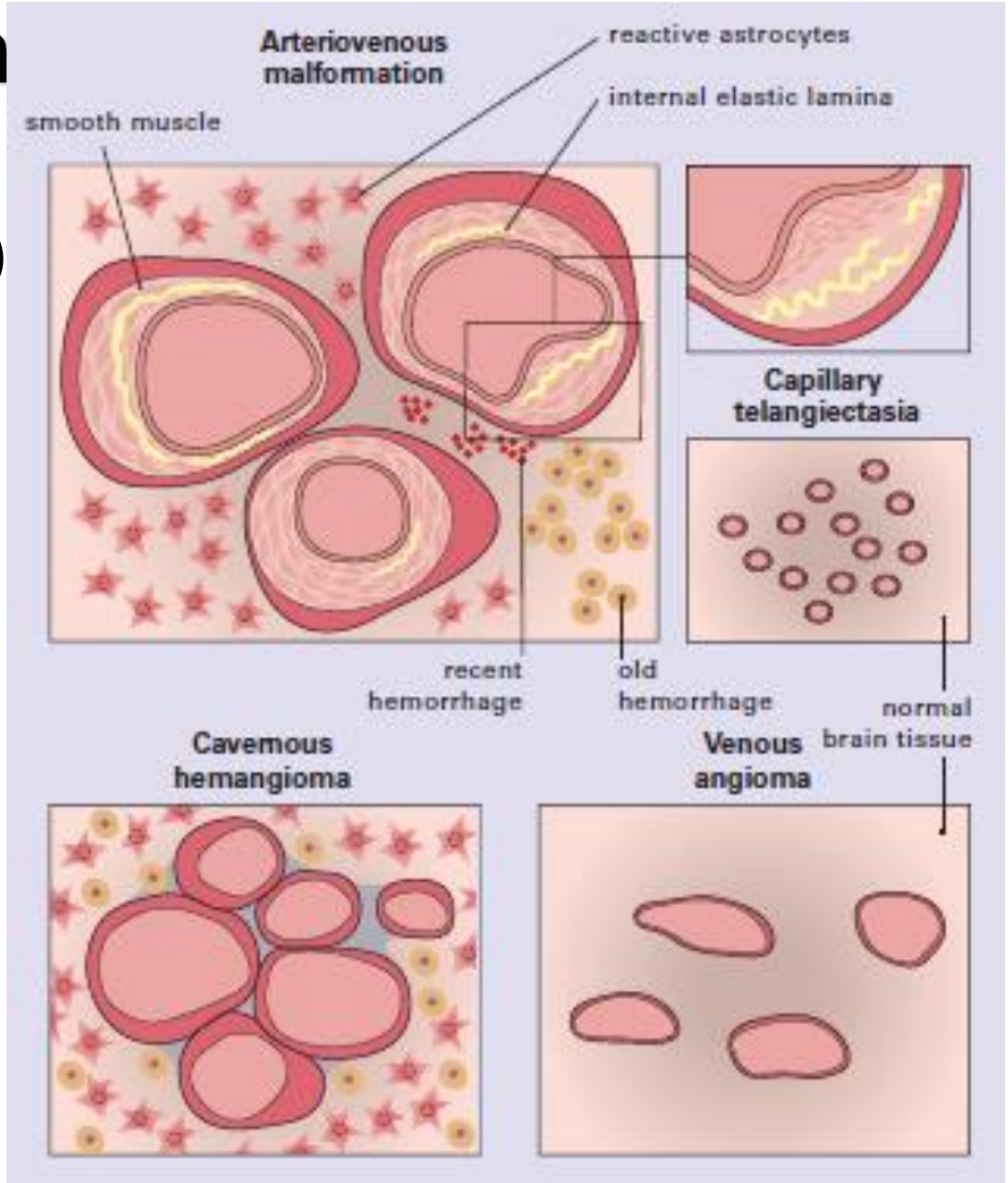
- Hirnnerven kompression
- Ischaemia
- Ruptur



11. Vaskuläre Malformationen

1. Arteriovenöse Malformation (AVM)
2. Kavernöses Malformation
3. Kapilläre Teleangiektasien
4. Venöses Angiom

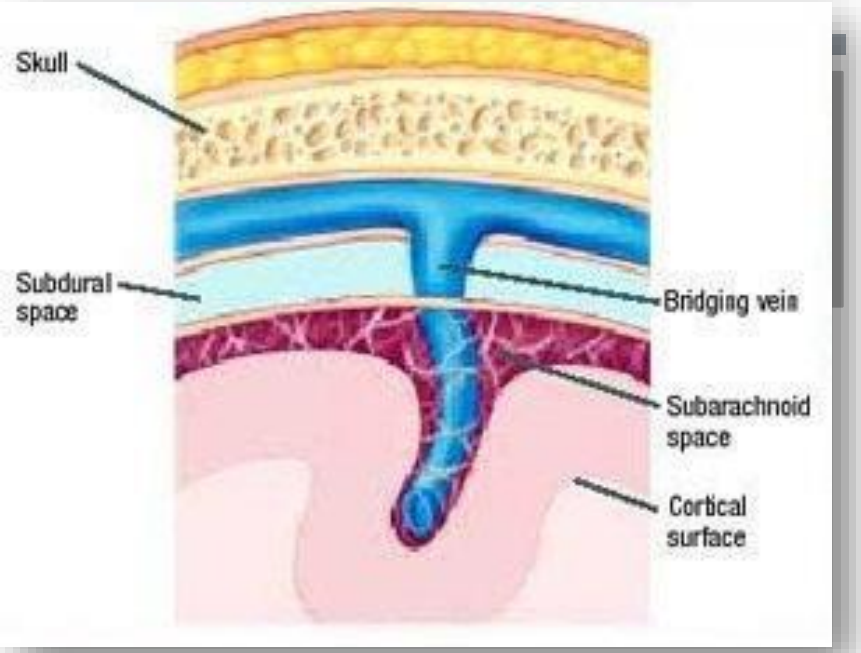
- Intrazerebrale Blutung auch!

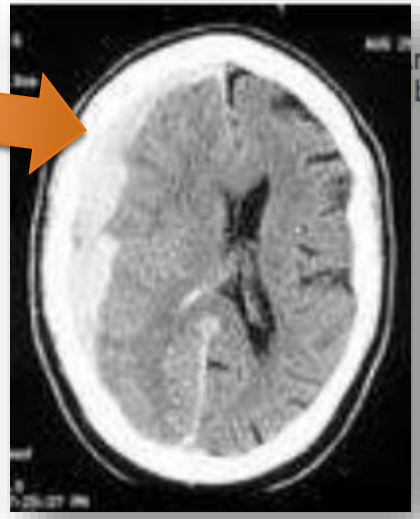


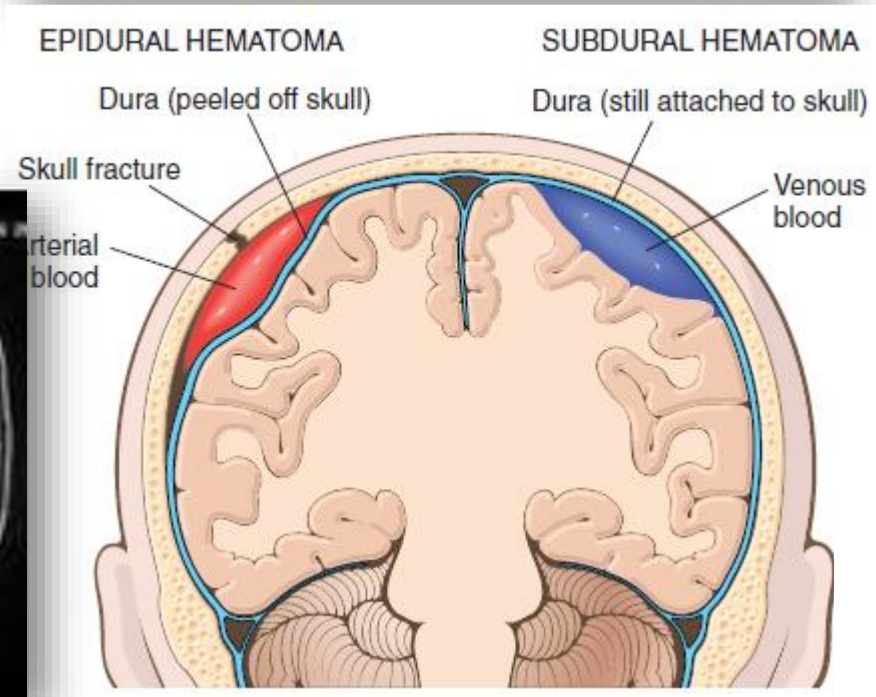
III. Subdurale Blutung

- Trauma
- Höheren Erwachsenenalter - Hirnatrophie
- Neugeborenen
  - Geburtstraumata oder forensisch relevante Schütteltraumata

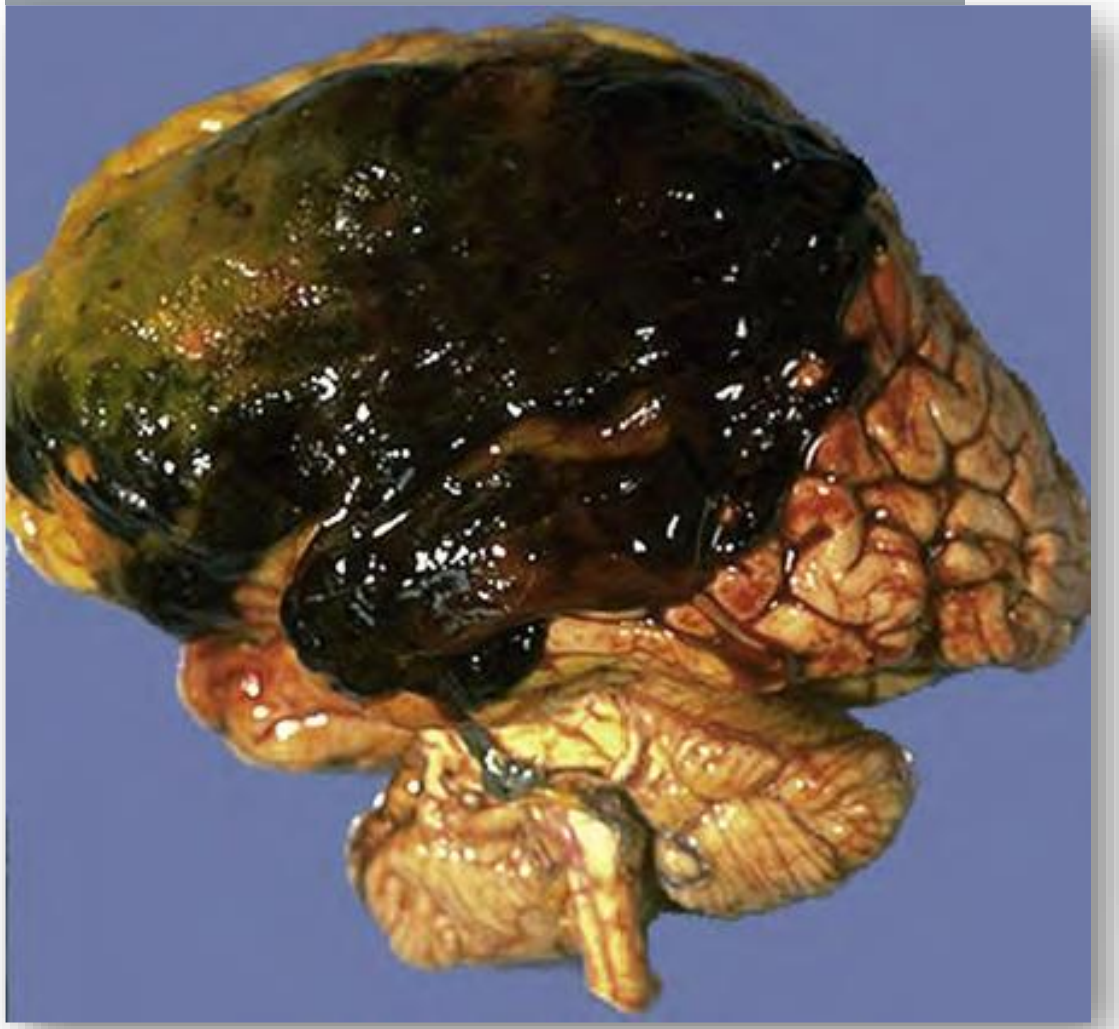
- Traumatisch bedingte Zerreißung von Brückenvenen

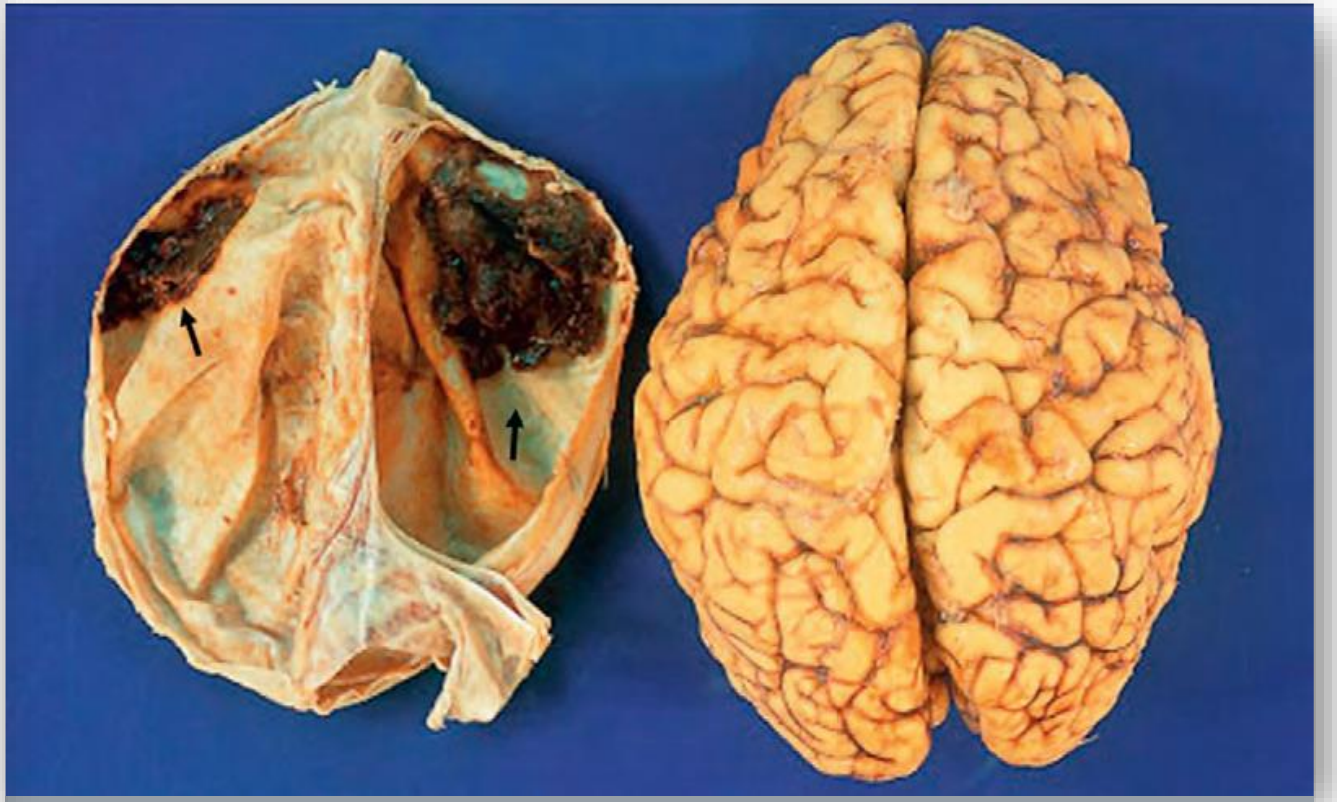






Akut subdurale Blutung



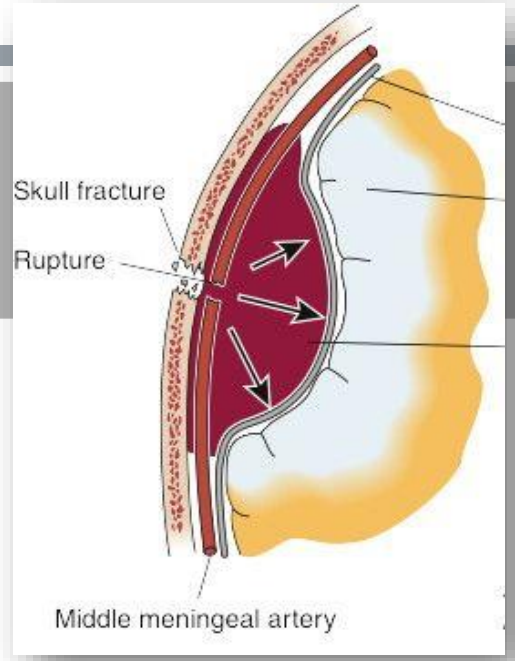


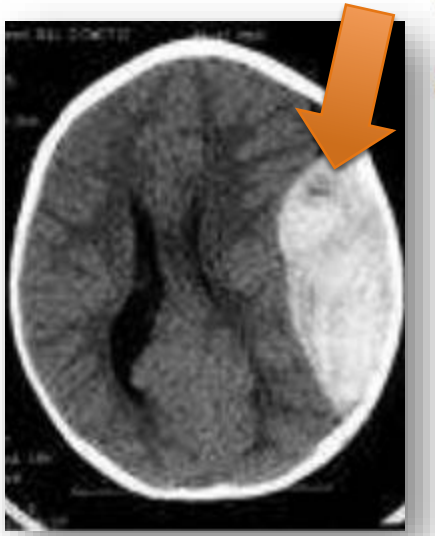
Chronisch subdurale Hämatome

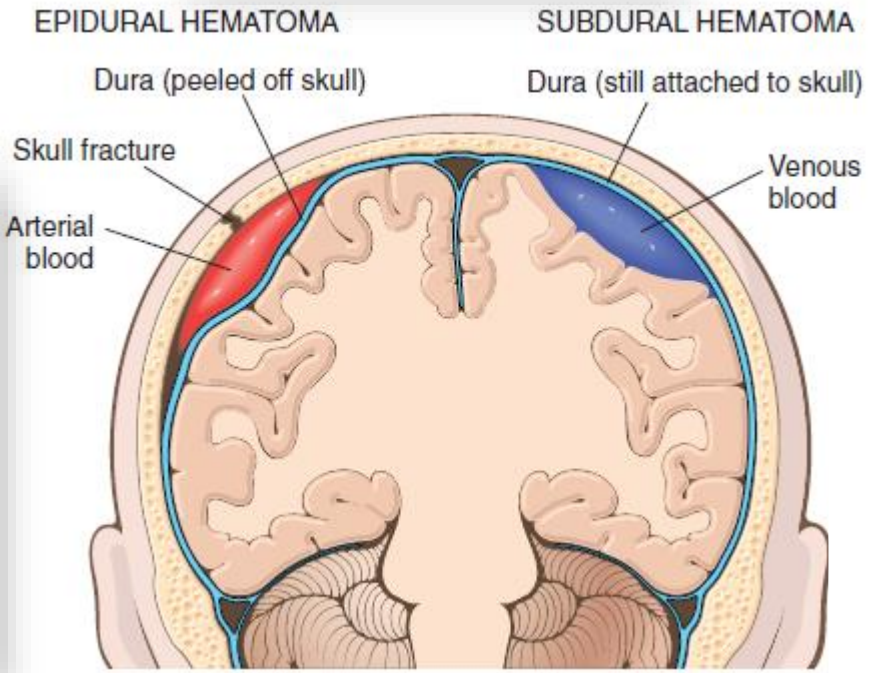
IV. Epidurale Blutung

- Trauma – Schädelfraktur
  - Arteria meningea media
- Erwachsenen – fossa temporalis
- Kinder – fossa posterior

- Intervalum lucidum



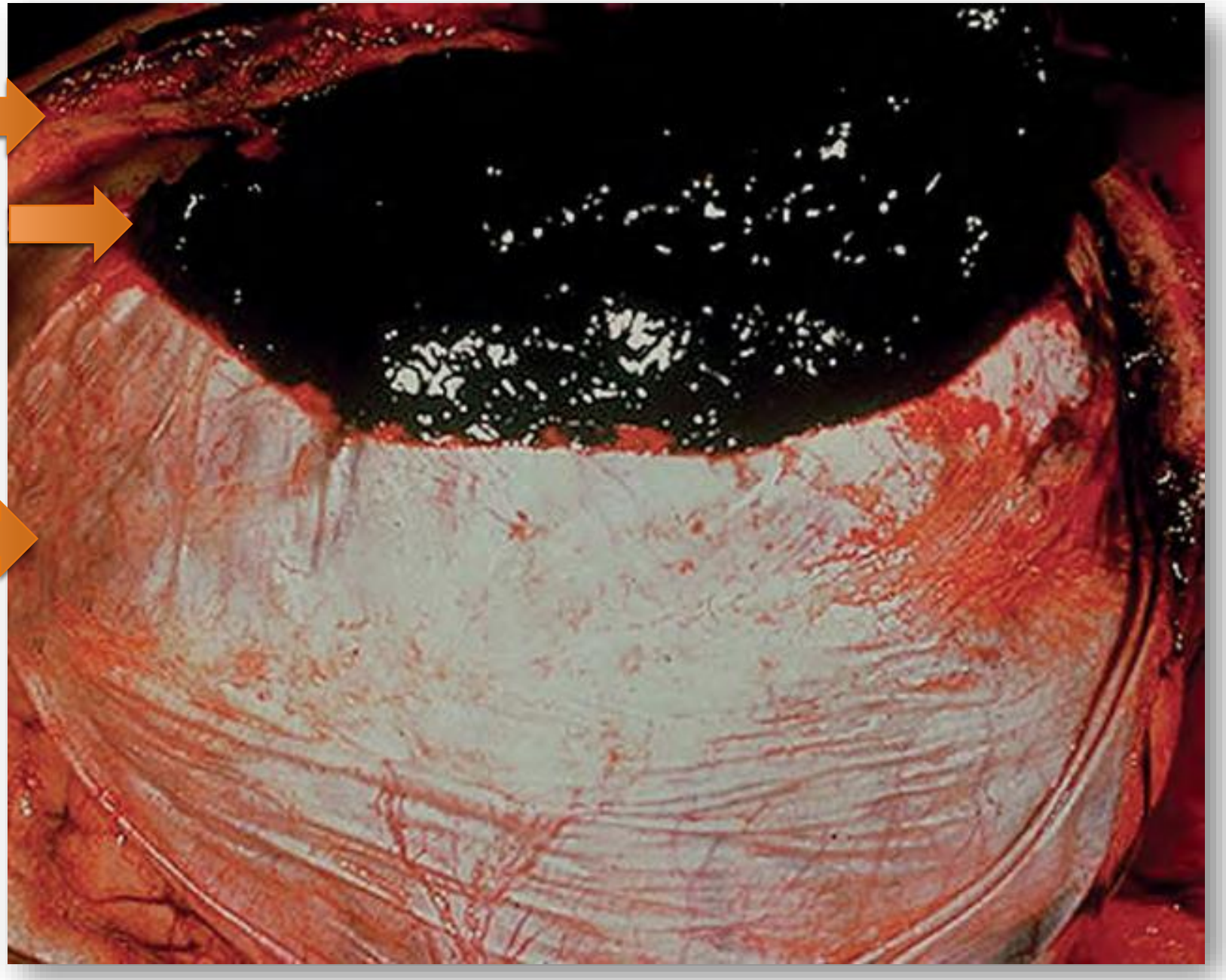




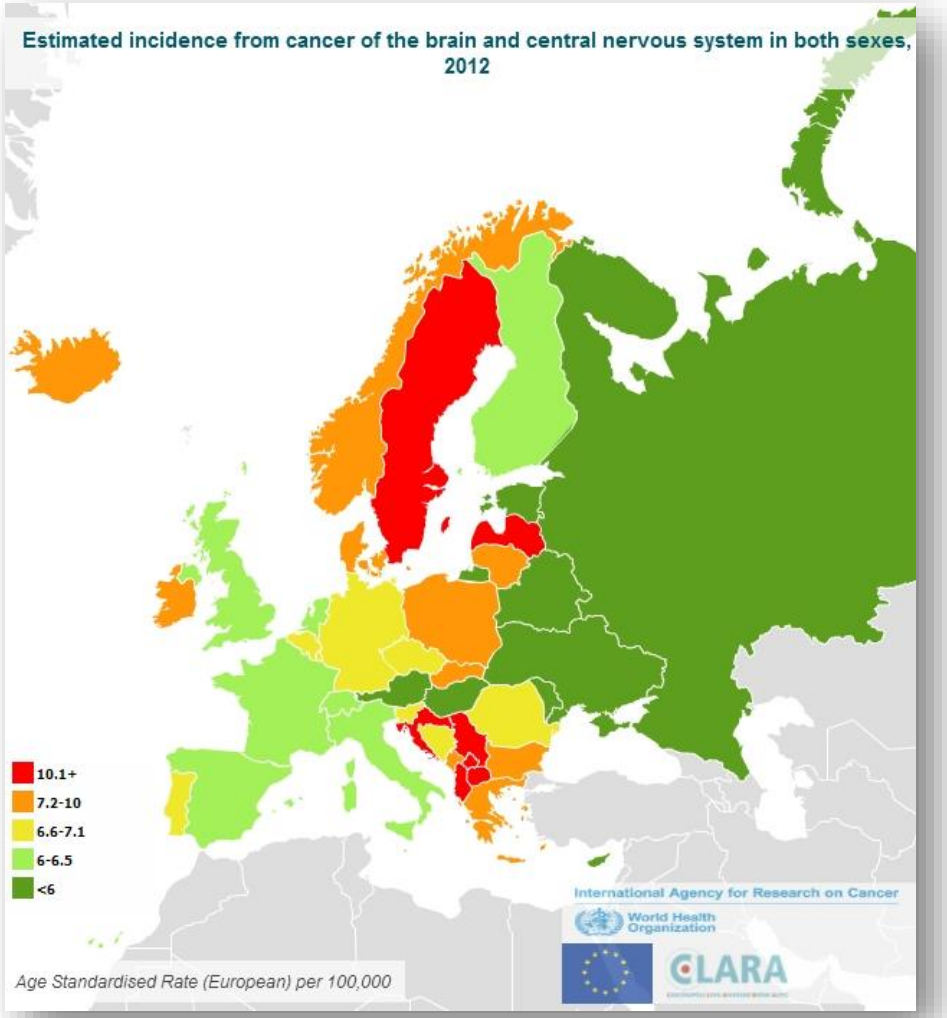
Schädelknochen

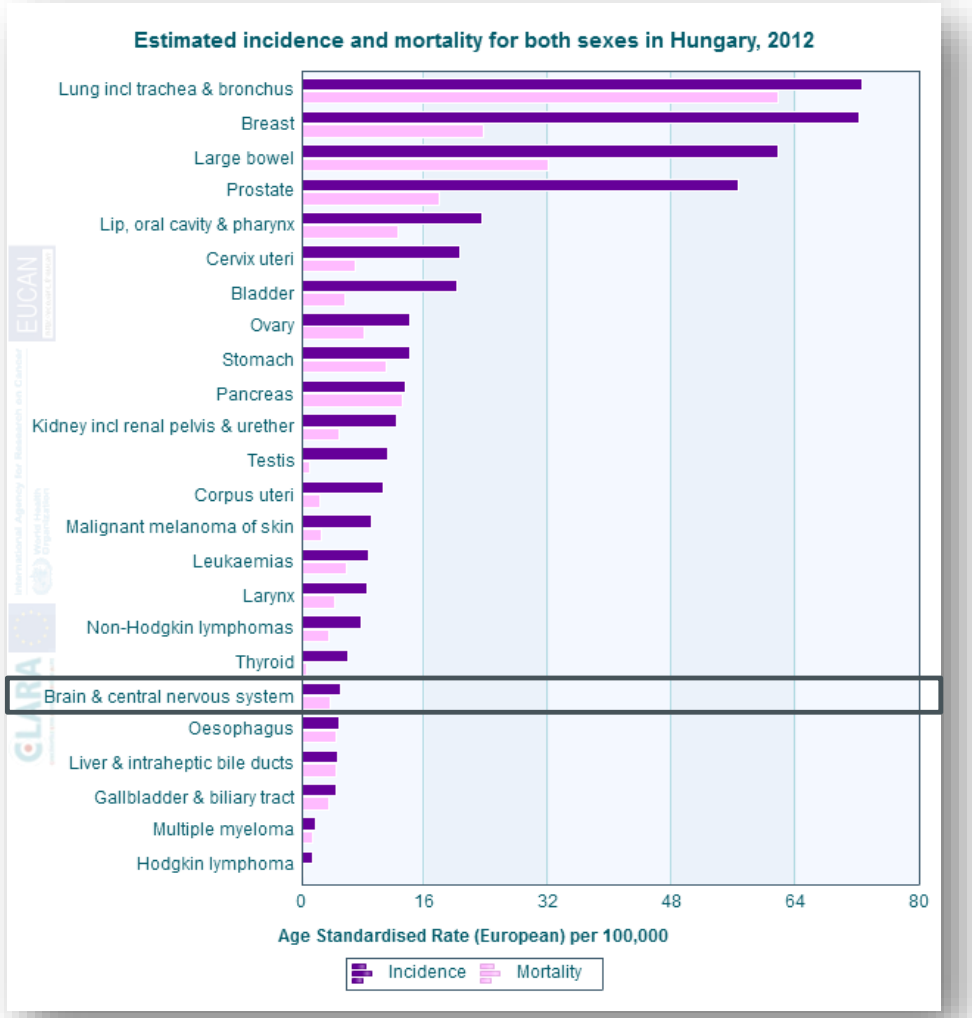
Akute epidurale Blutung

Dura mater

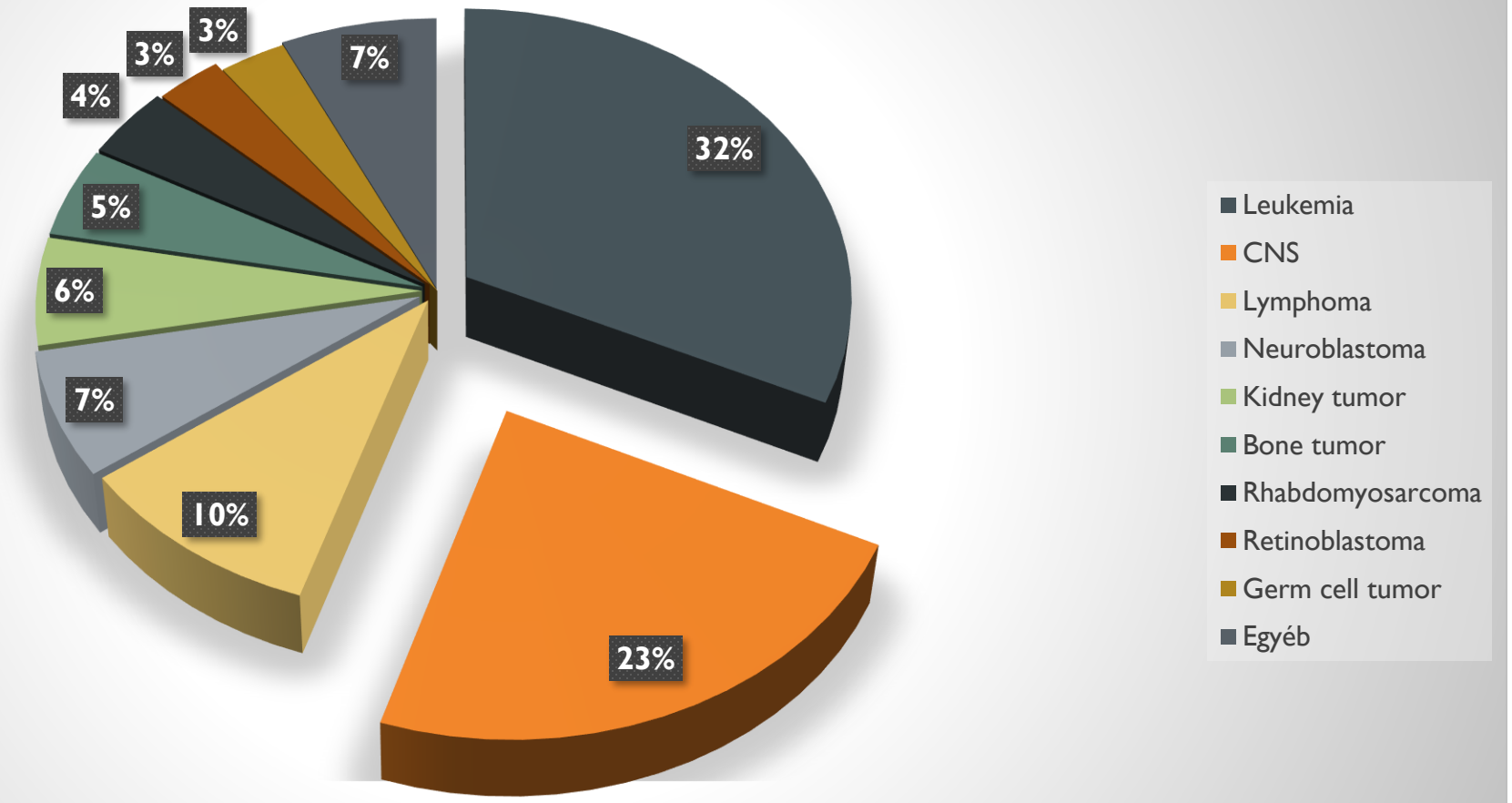


TUMOREN DES NERVENSYSTEMS



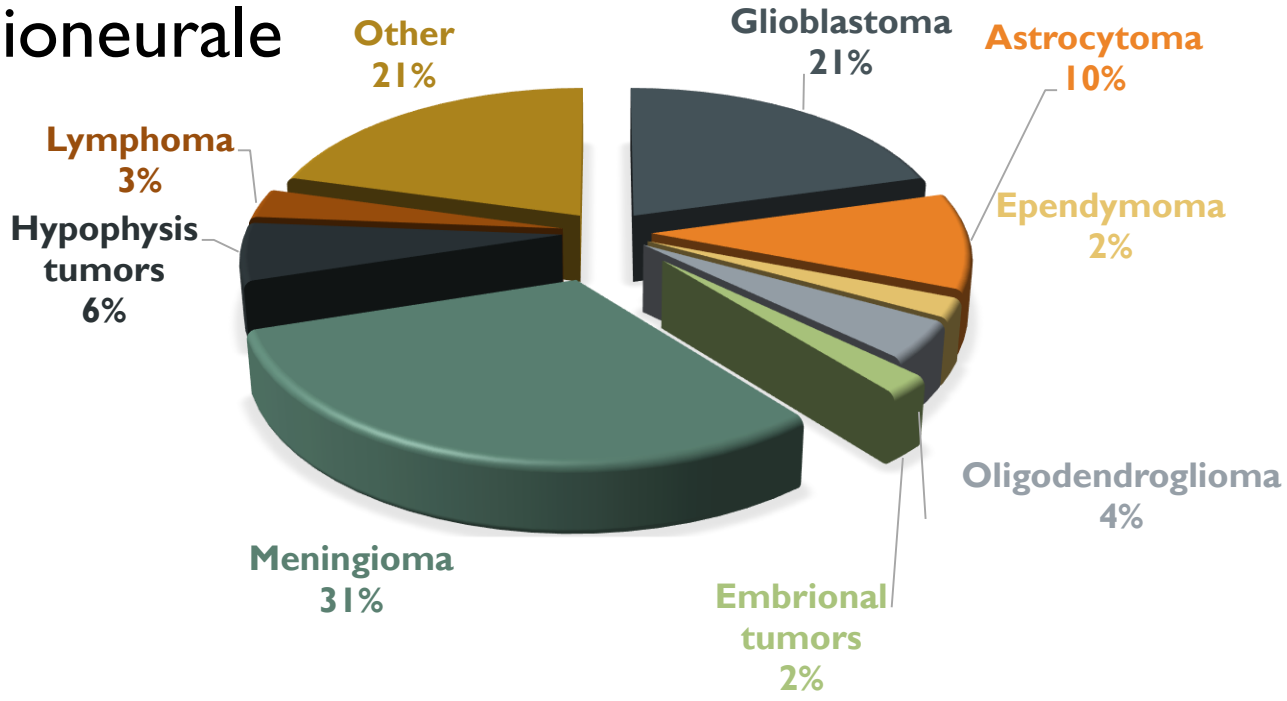


INCIDANCE OF CHILDHOOD NEOPLASMS



PRIMÄRE TUMOREN DES ZNS

1. Neuroepitheliale Tumoren
   1. Gliome
   2. Neuronale oder gemischte glioneurale Tumoren
2. Plexus Choroideus Tumoren
3. Embryonale Tumoren
4. Tumoren der Hirnhäute
5. Andere parenchymale Tumoren
   - Haematologische Neoplasien
   - Keimzelltumoren



Allgemeine Aspekte

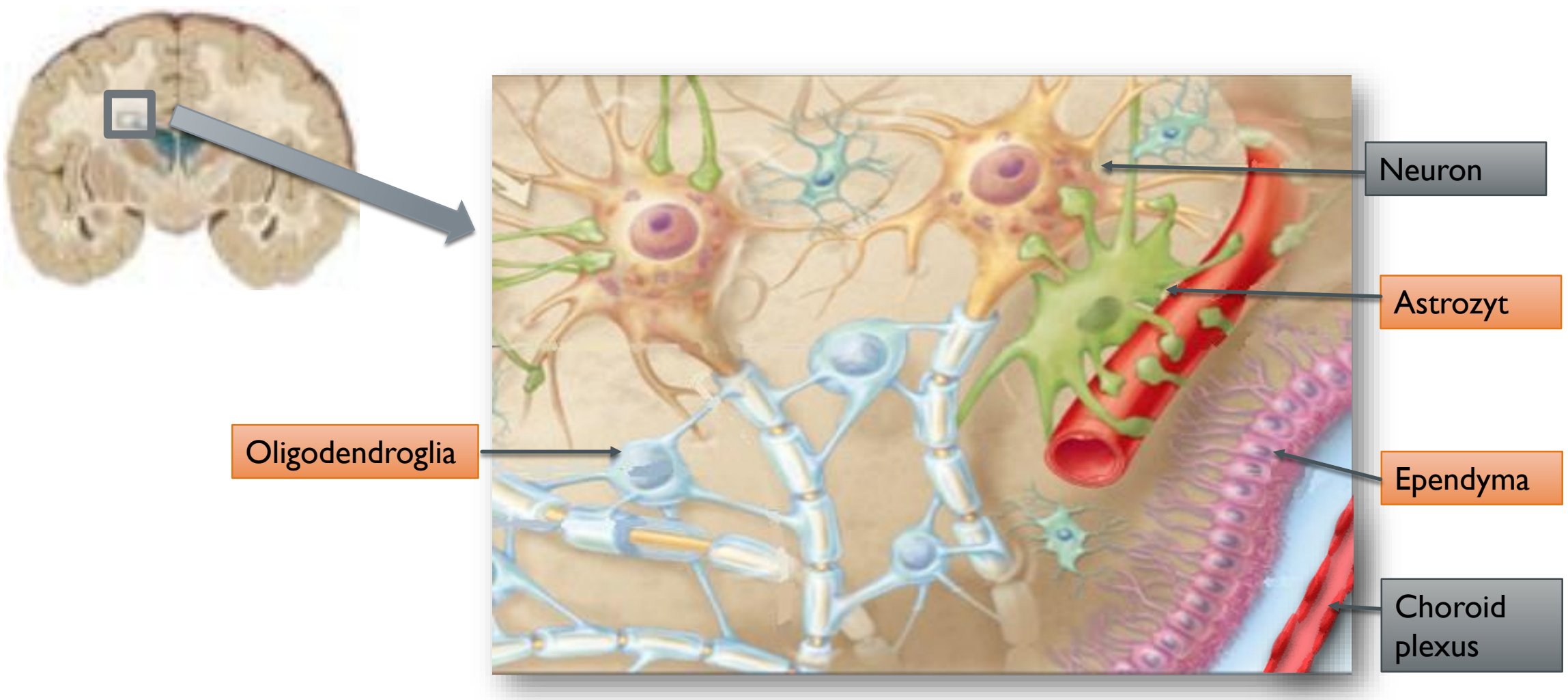
- Symptomen
  - Krampfanfälle
  - Zeichen eines erhöhten Hirndrucks
  - Fokale neurologische Ausfälle
  - Hydrocephalus
- Diagnose
  - Alter
  - Geschlecht
  - Lokalisation
  - Anamnäse

Grad

- Prognoseabschätzung

- Grad I
  - niedrige Proliferationsaktivität
  - gutartiger Krankheitsverlauf
- Grad II
  - Infiltrative
  - höheres Rezidivrisiko
  - Progression
- Grad III
  - Kernpleomorphie
  - Hohe Mitoserate
- Grad IV
  - hohes Rezidivrisiko
  - maligner Krankheitsverlauf

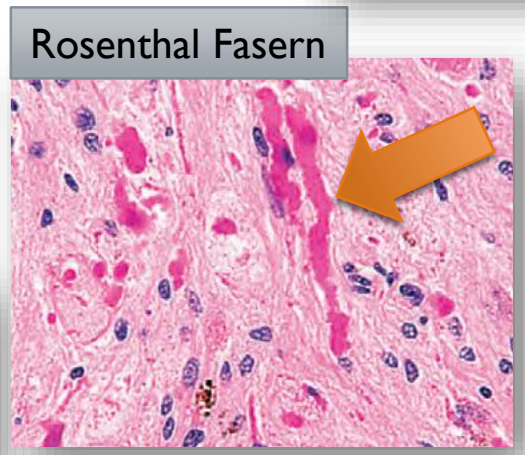
I. GLIOMAS

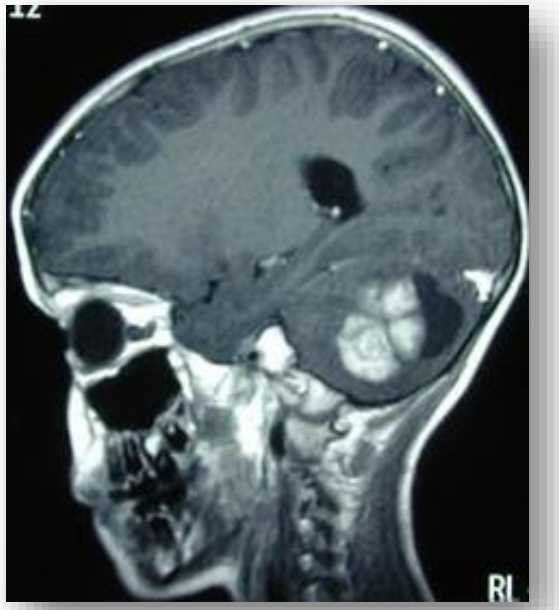

Neuron
Astrozyt
Oligodendroglia
Ependyma
Choroid plexus

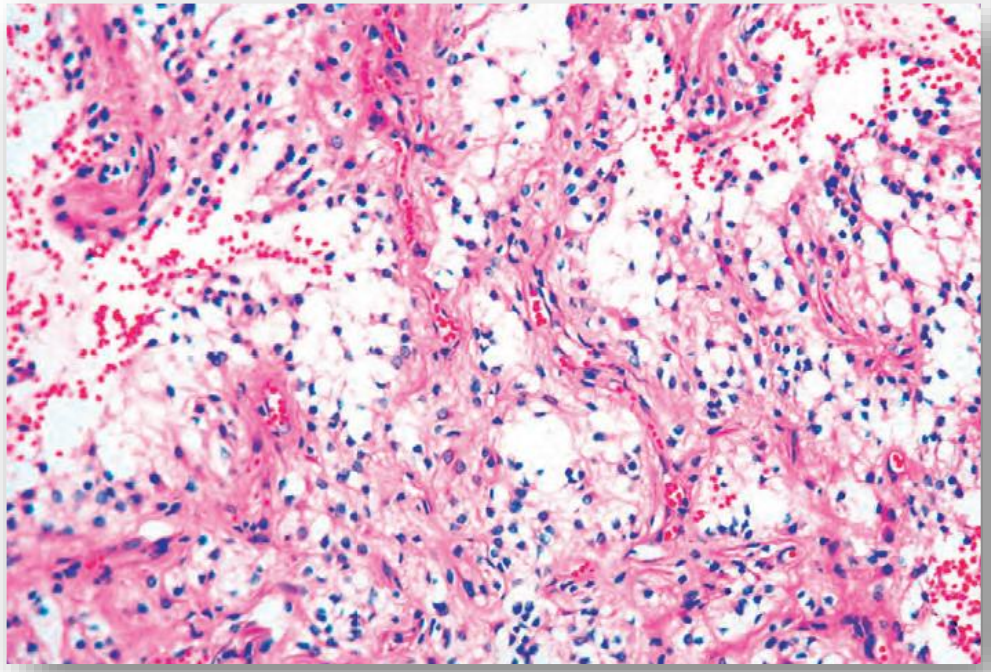
I. I. Astrozytome

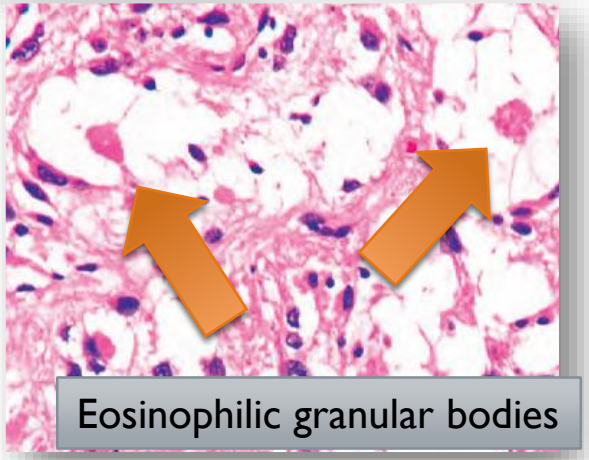
1. Pilozytische astrocytom (Grade I)

- Tumor des Kindes- und Jugendalters
- sehr gute Prognose
- Cerebellum, Hirnstamm









II. Diffuse astrozytomen (Grad II-IV)

- Mittleren Lebensalter
- weißen Substanz der Großhirnhemisphären

Astrozytome Grad II

- Kernpolymorphie+ Hohe Zellularität

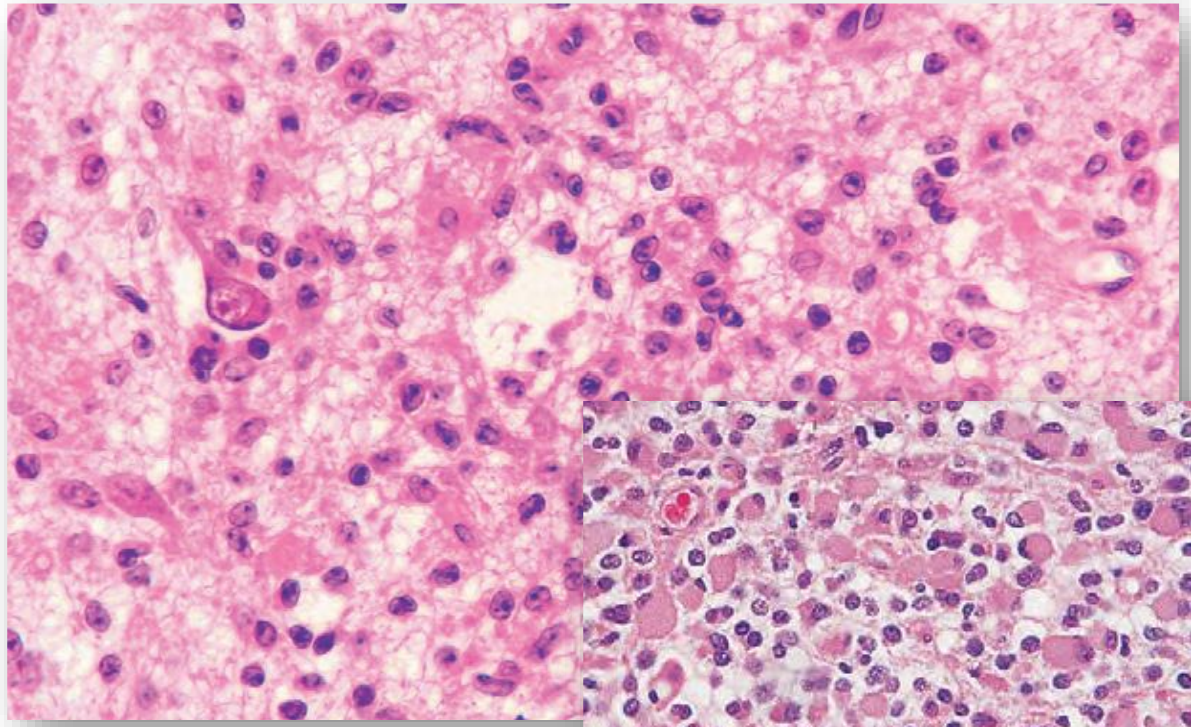
Anaplastische Astrozytome Grad III

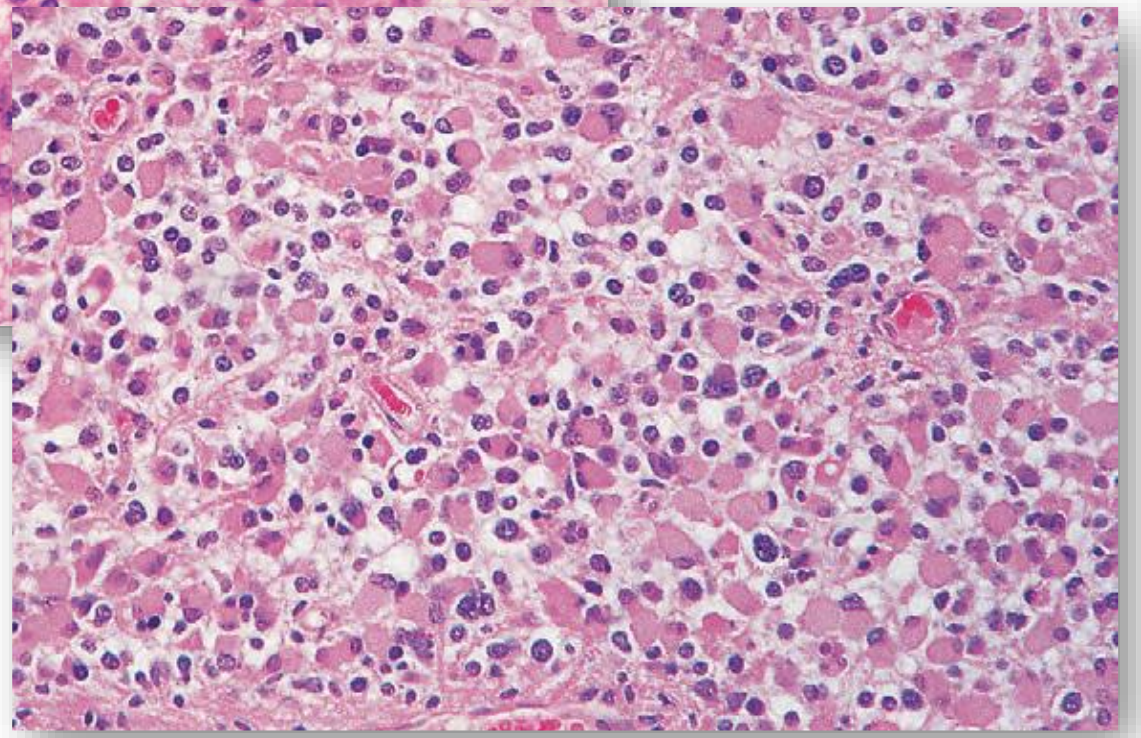
- Kernpolymorphie+Hohe Zellularität +erhöhte Mitoseaktivität

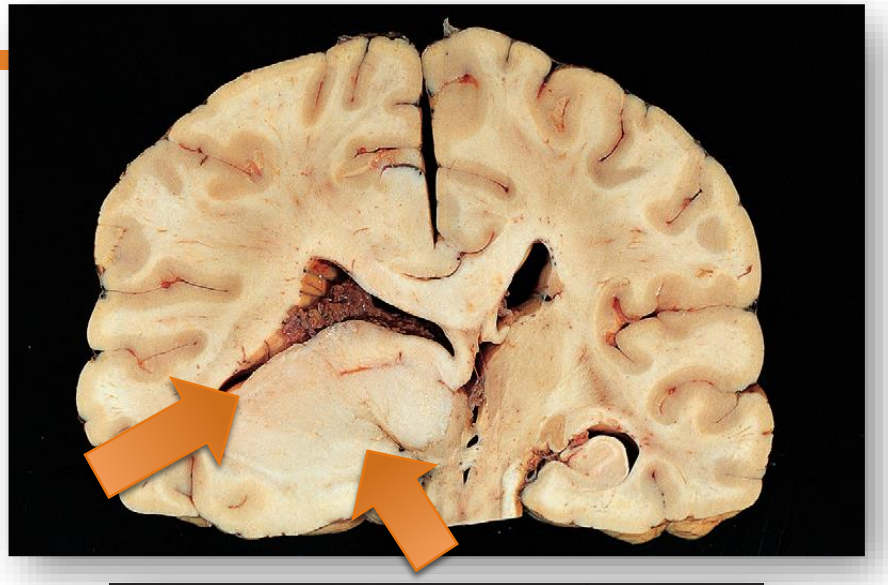
Glioblastom Grad IV

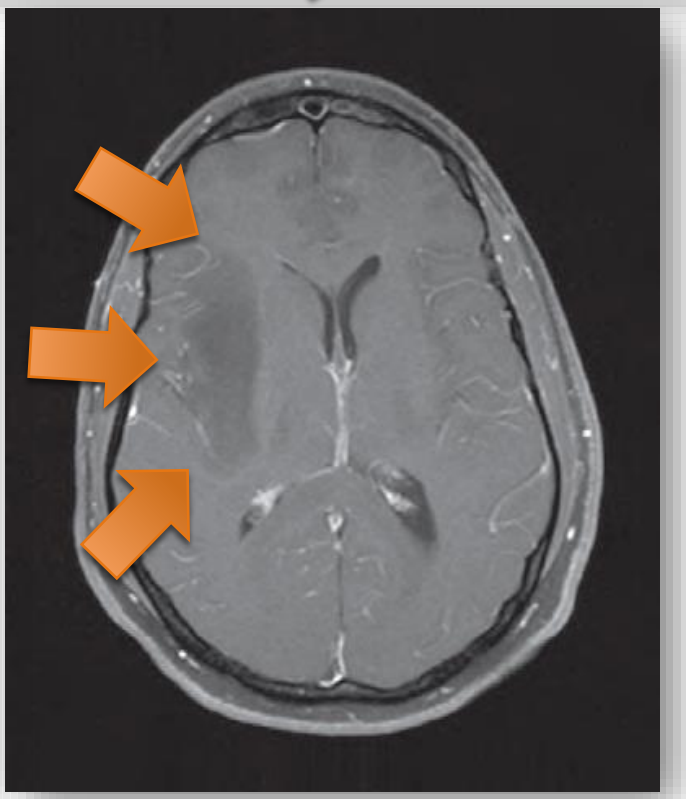
- Kernpolymorphie+Hohe Zellularität +erhöhte Mitoseaktivität + Nekroze/Endothelzellen proliferation(Gefäßneubildung)

Astrozytom Grad II

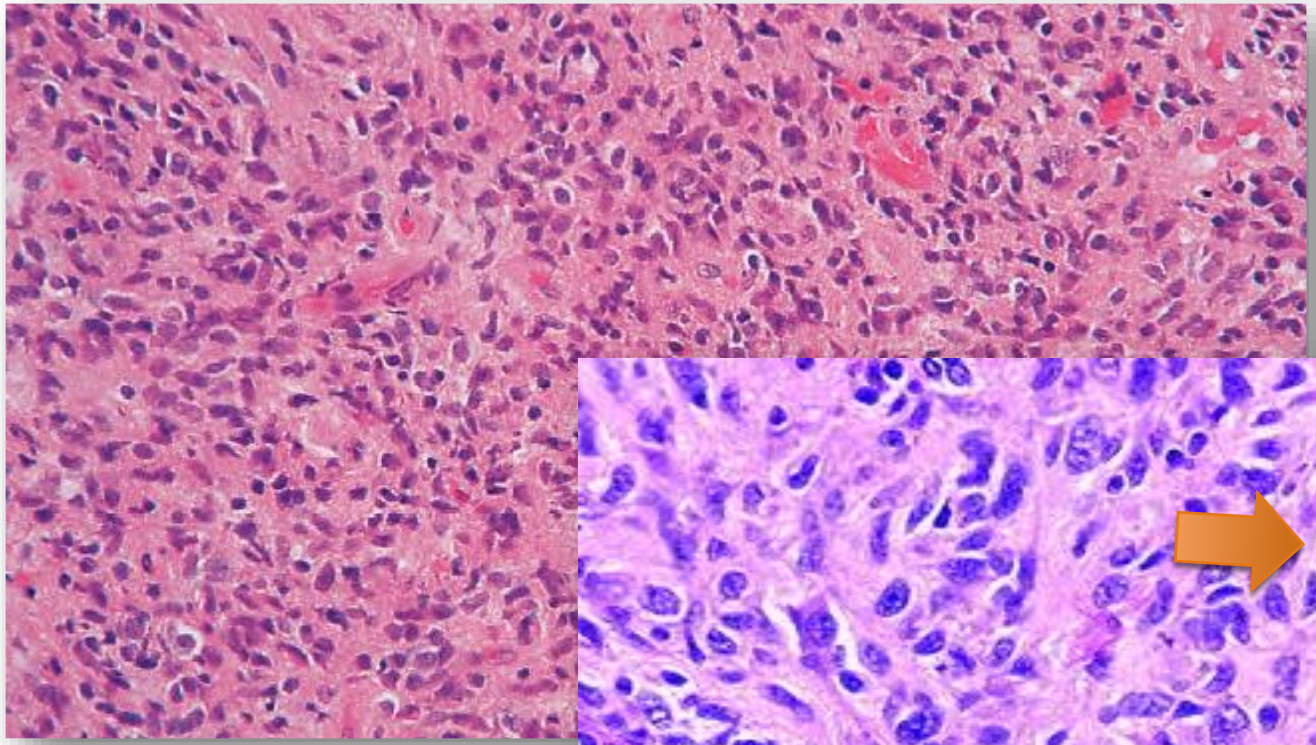


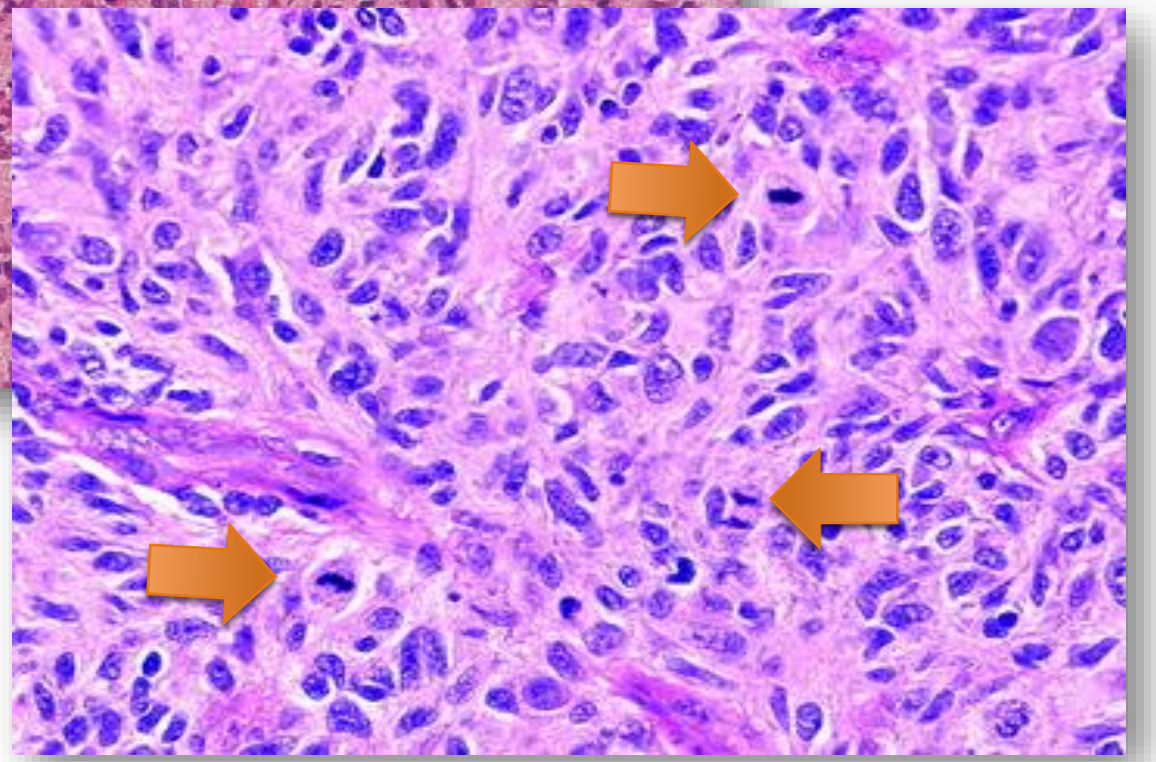


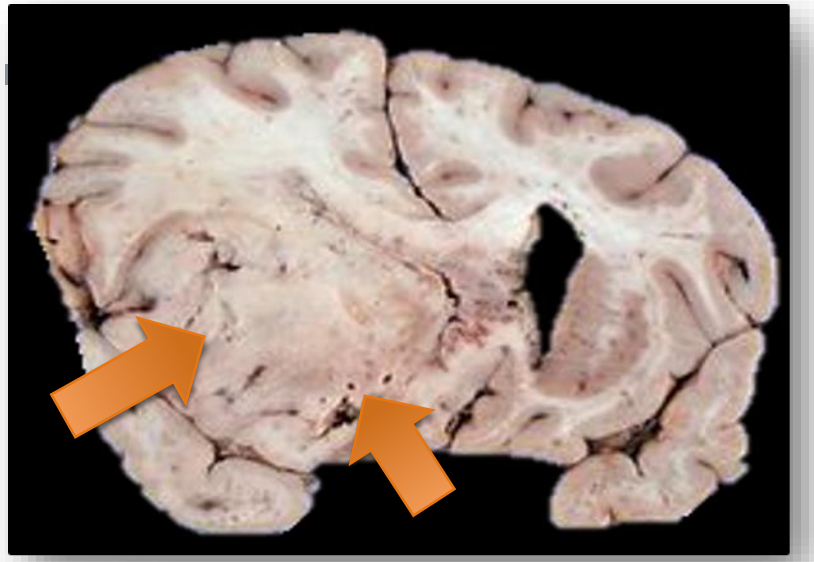


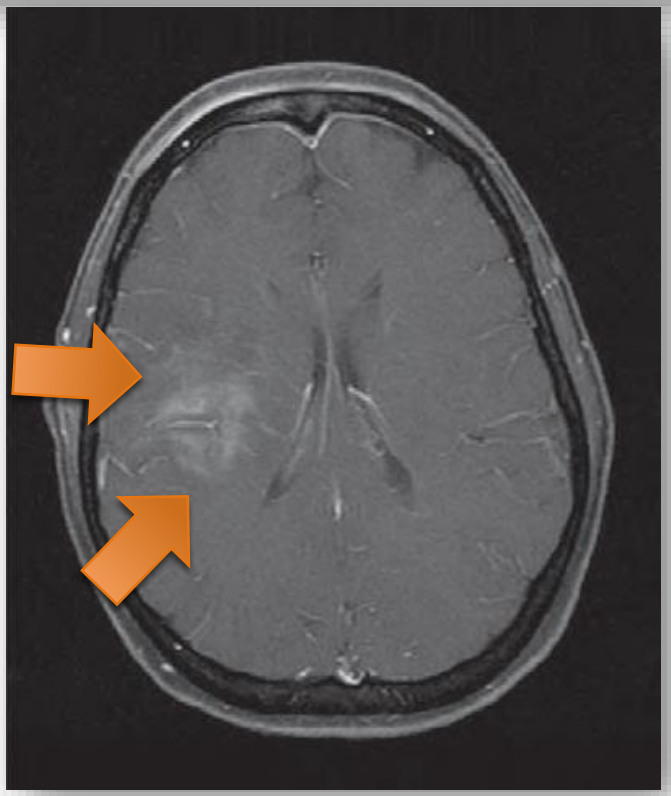


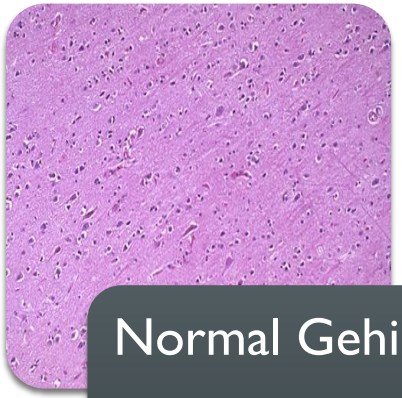
Anaplastische Astrozytom Grad III











Normal Gehirn

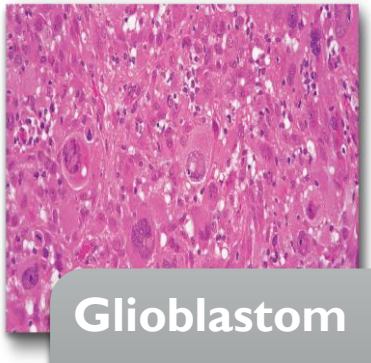
- IDH1 mutation

Astrozytom

- Loss of 19 ch
- ATRX loss

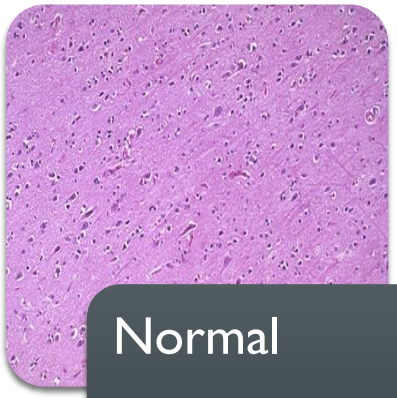
Anaplastische astrocytom

- 10 q loss

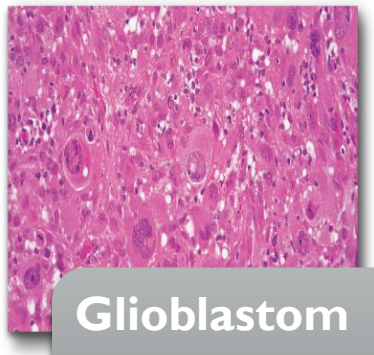


Glioblastom

- Secunder



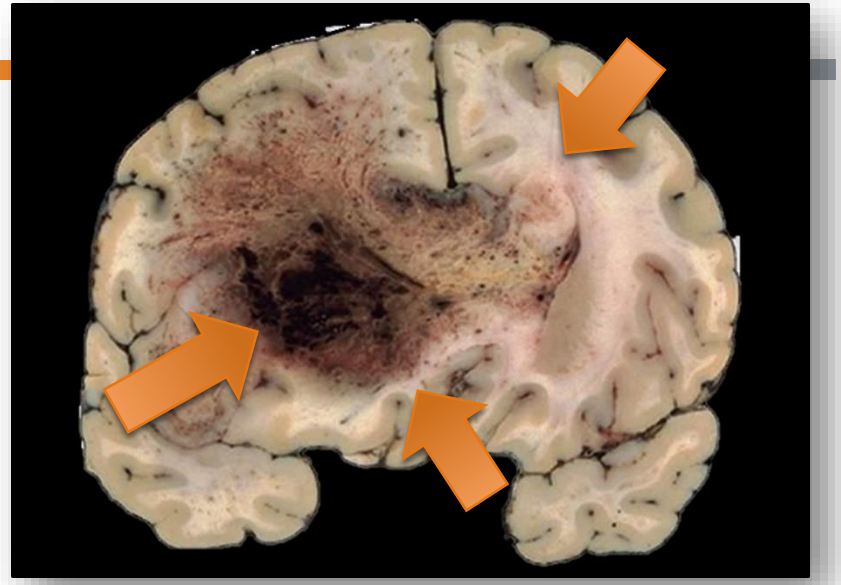
Normal

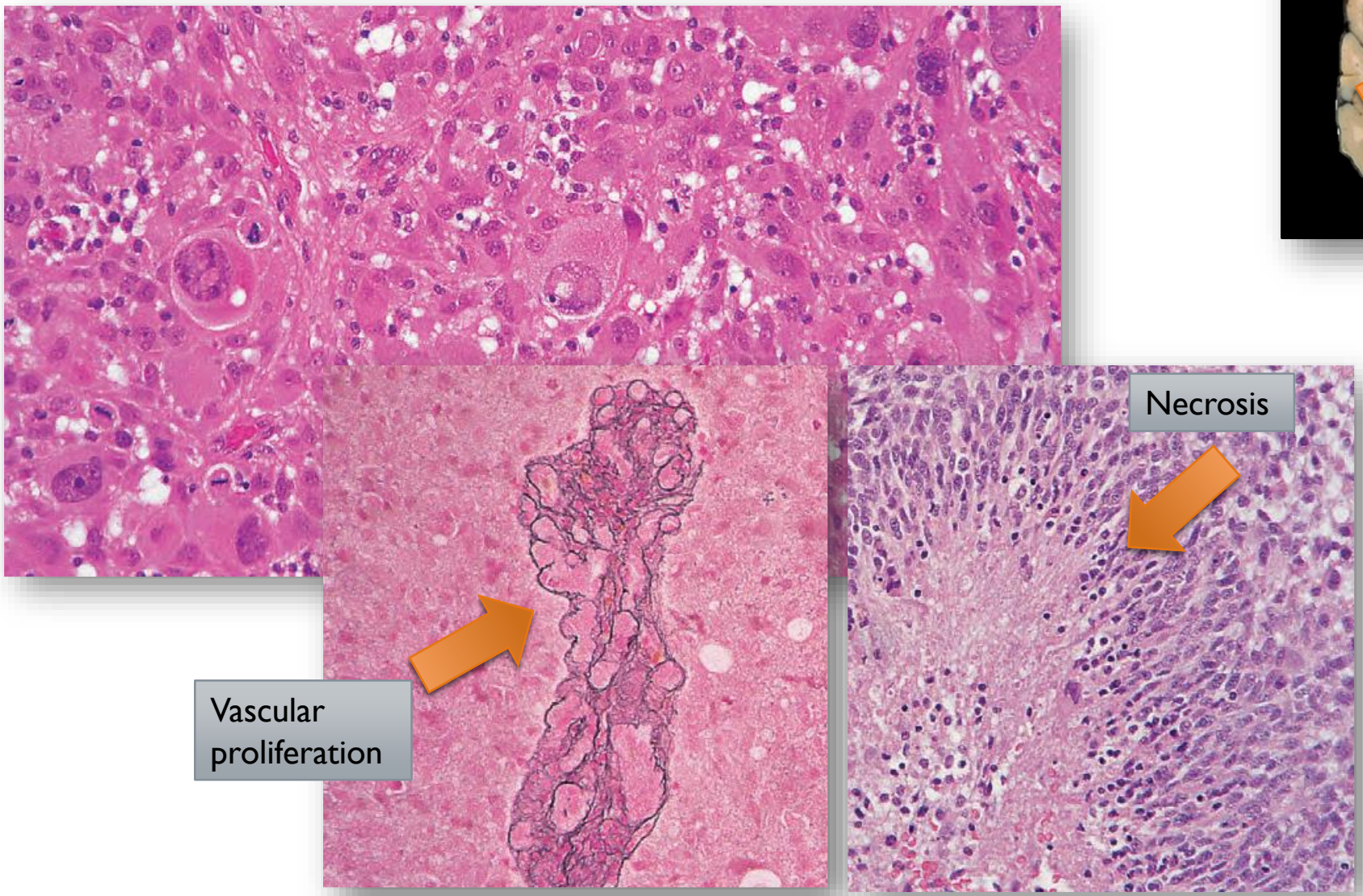


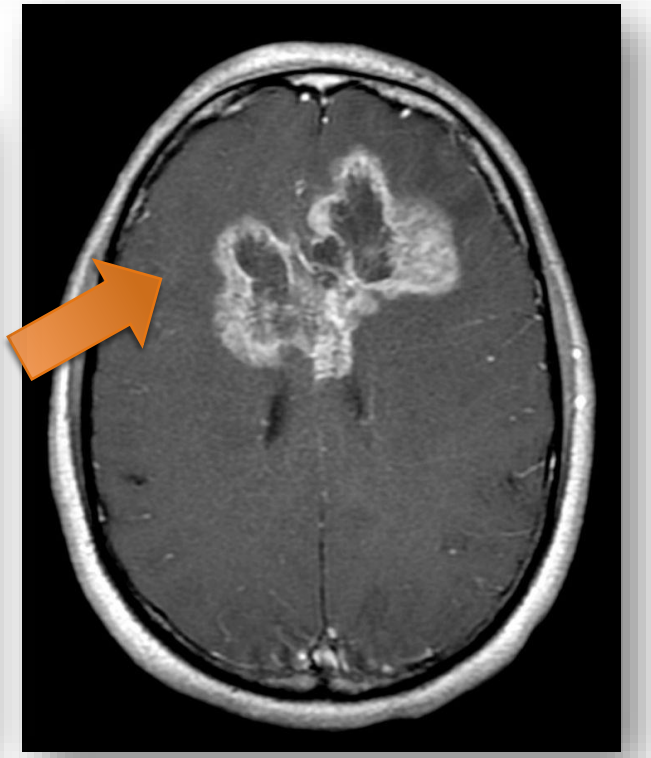
Glioblastom

- Primer
- EGFR overexpr.
- 10q loss
- PTEN mutation

Glioblastom Grade IV



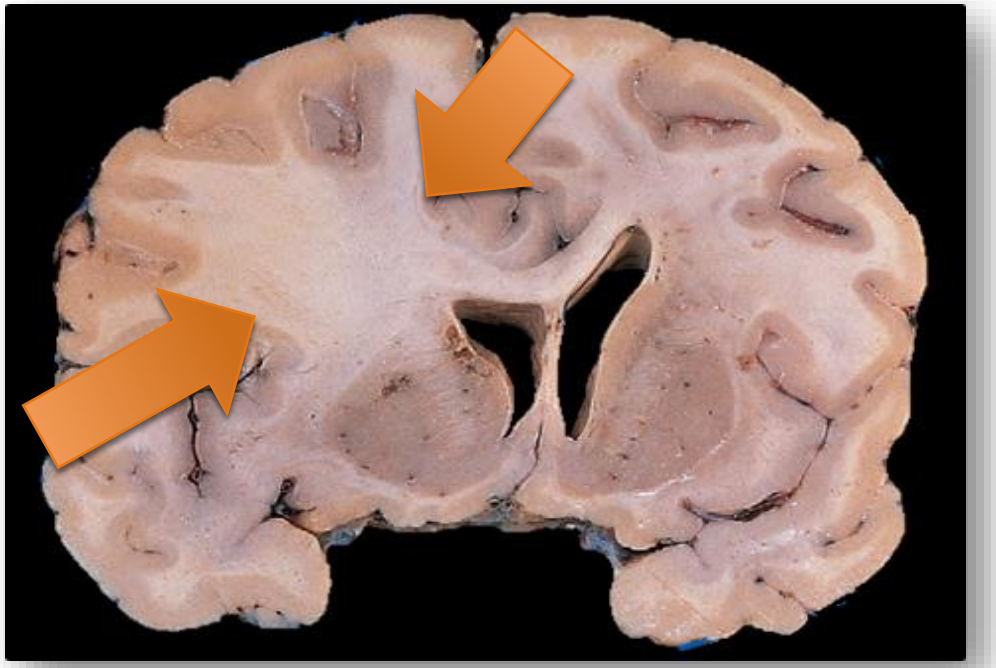


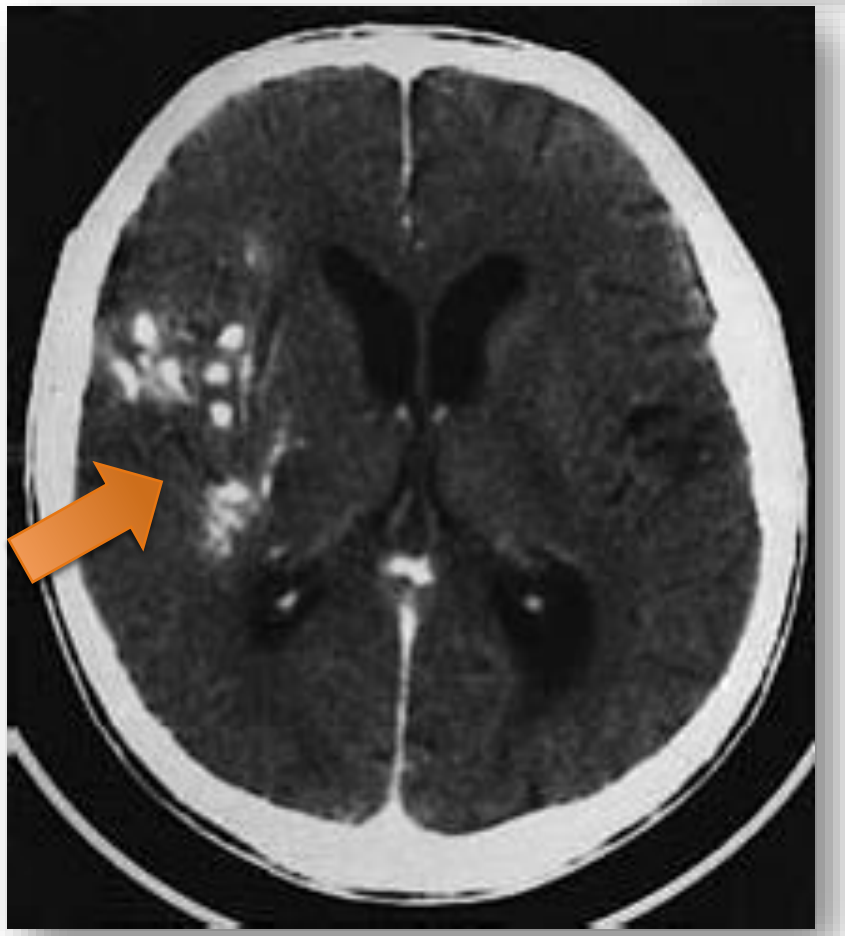


I. II. Oligodendrogliome

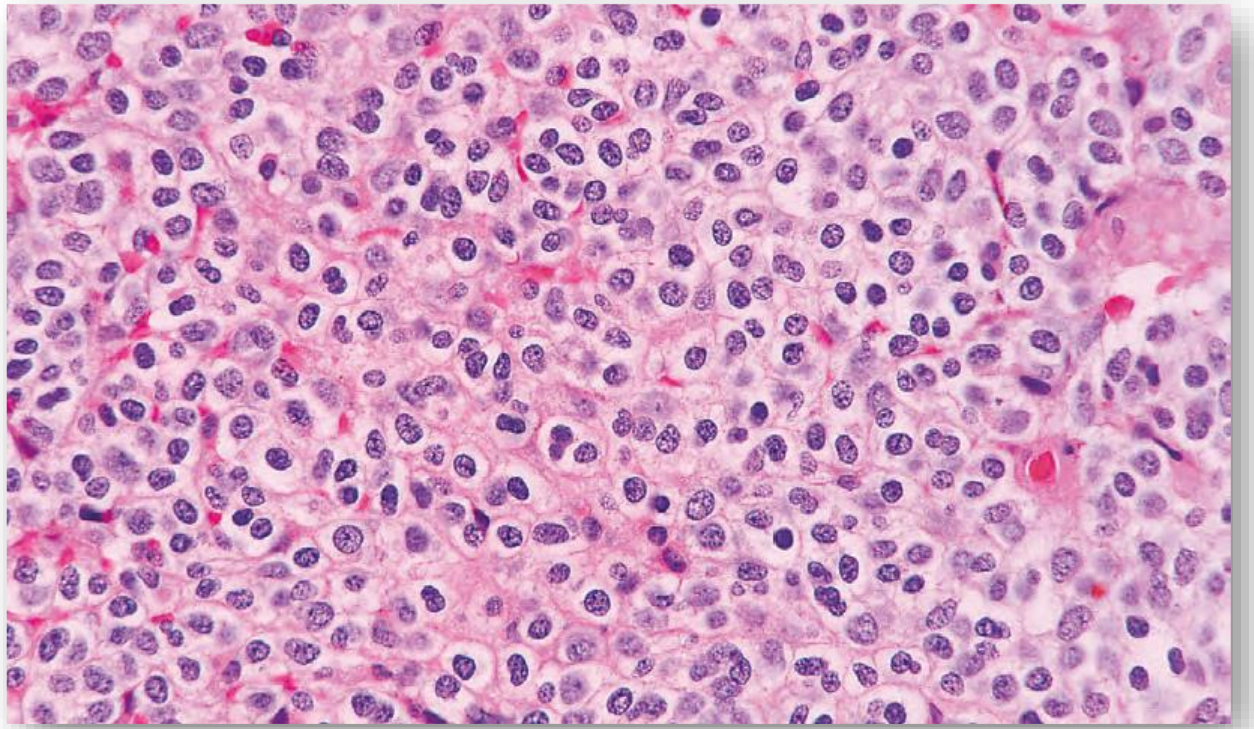

Oligodendroglia

- Im mittleren Lebensalter
- weißen Substanz der Großhirnhemisphären
  - Frontale, temporale Lappen
- 1p 19q codeletion

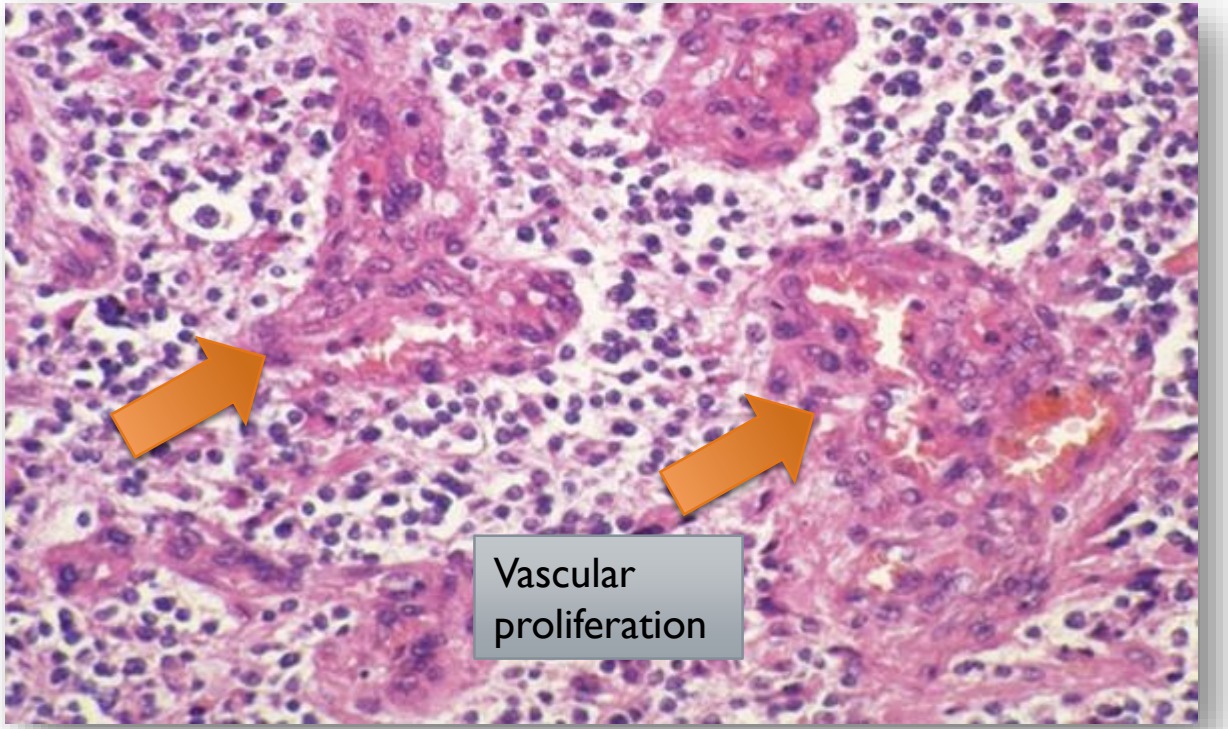




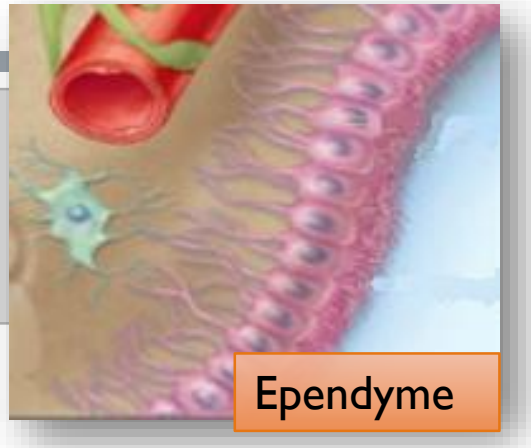
Oligodendrogliome Grad II



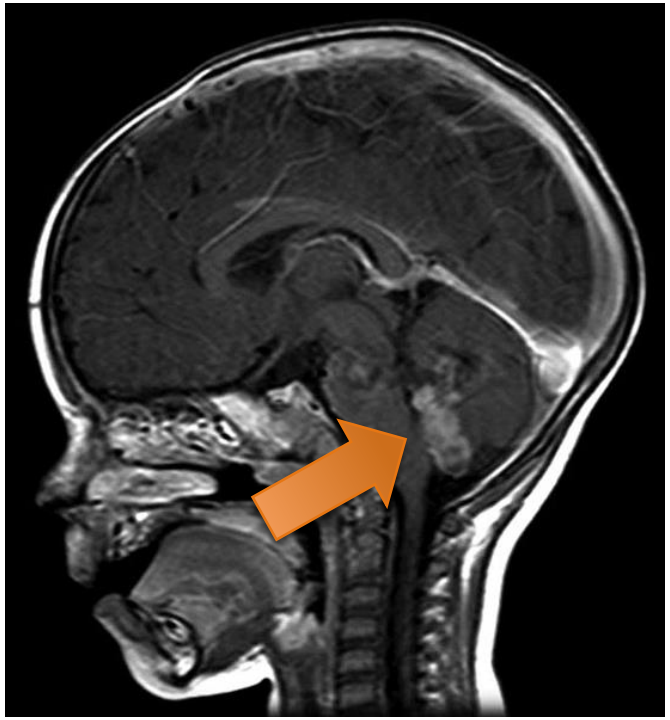
Anaplastische oligodendrogliome Grad III

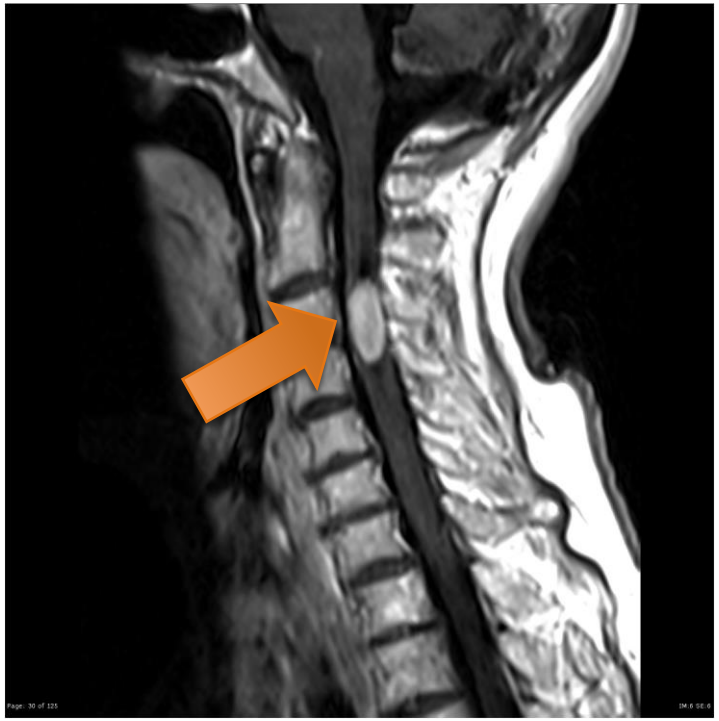


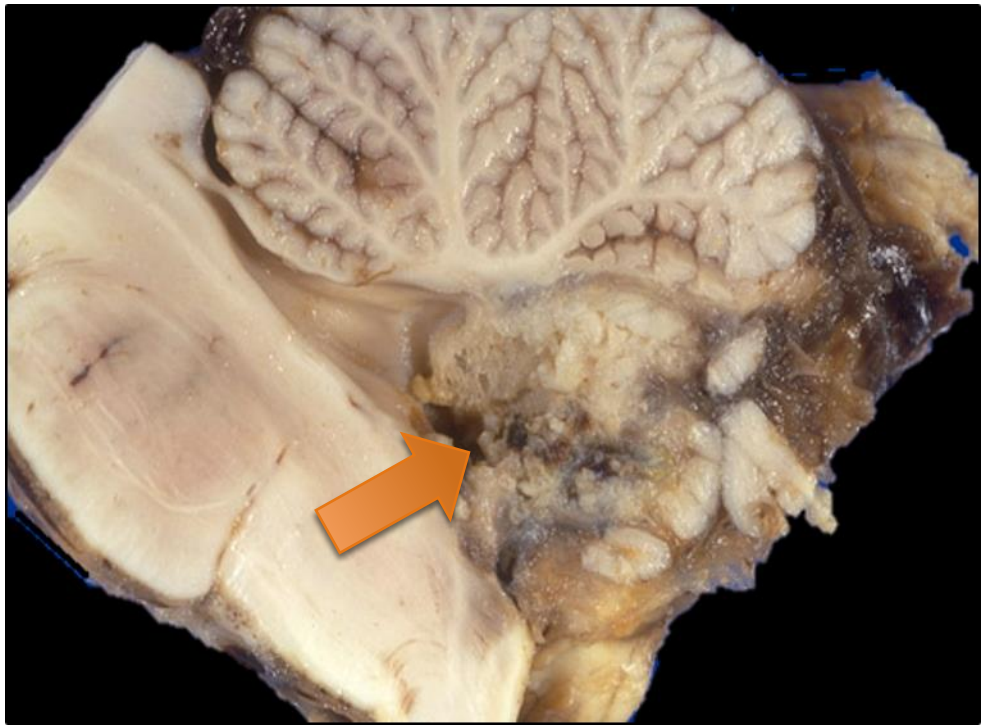
I. III. Ependymome



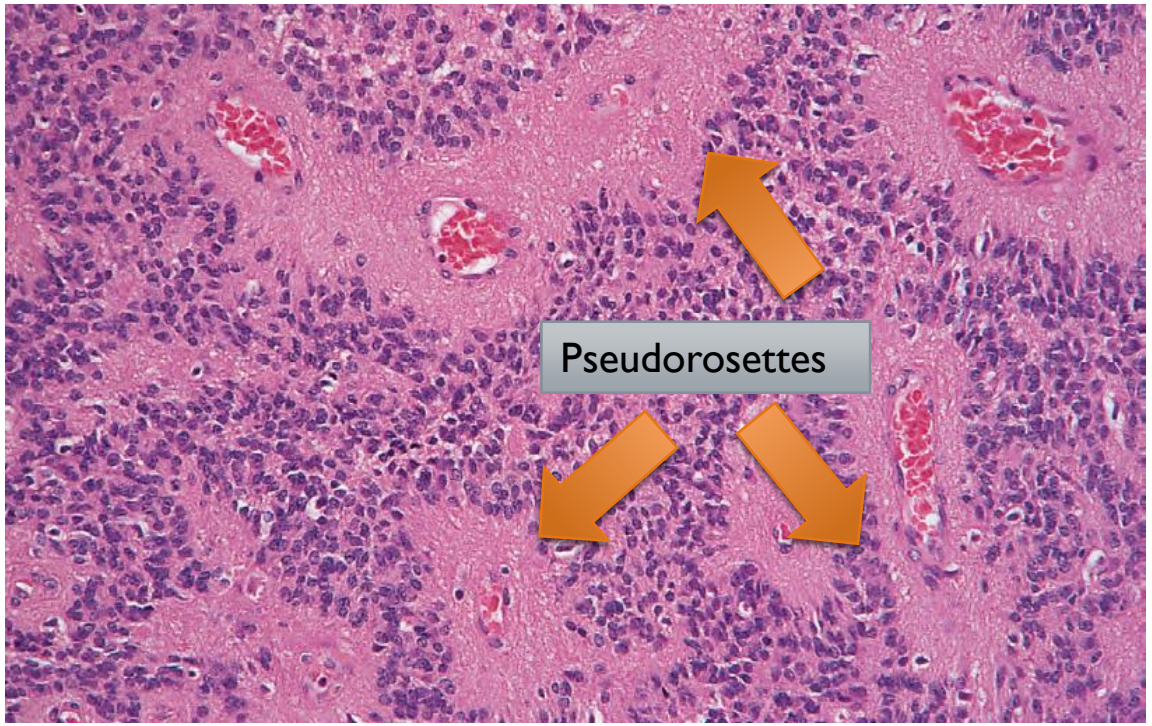
- Intrakraniell – Kindesalter
  - IV. Ventrikel, III. Ventrickel
- Spinale ependymome – 20-40 Jahre



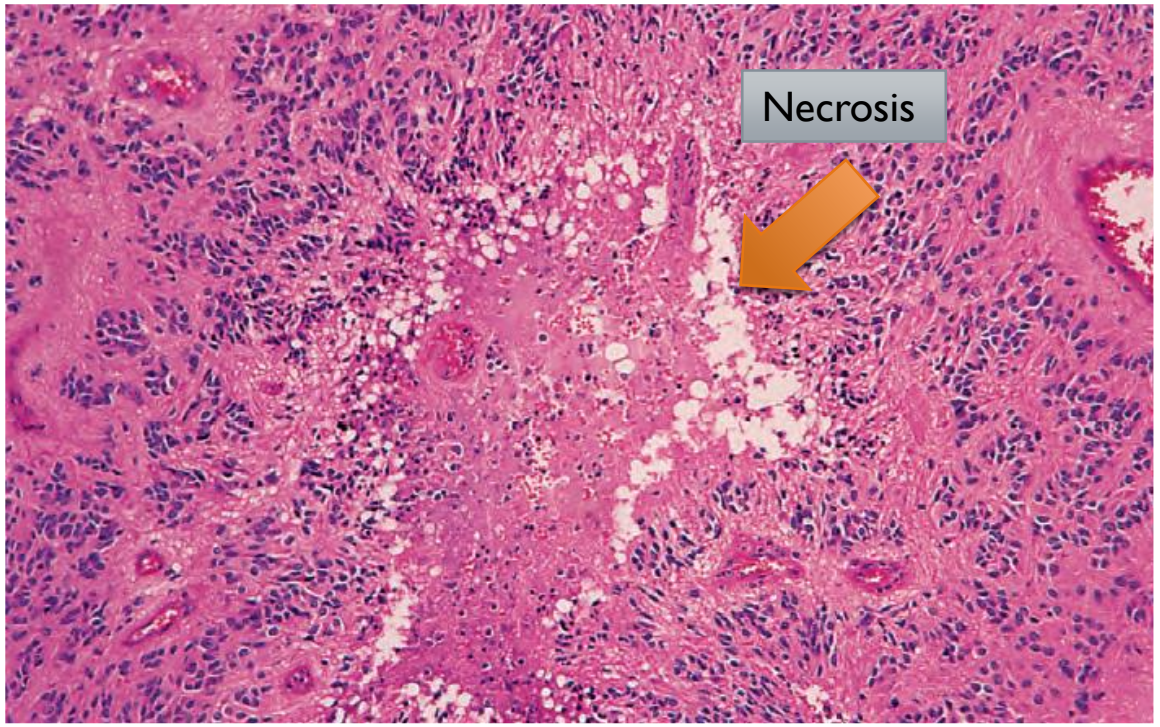




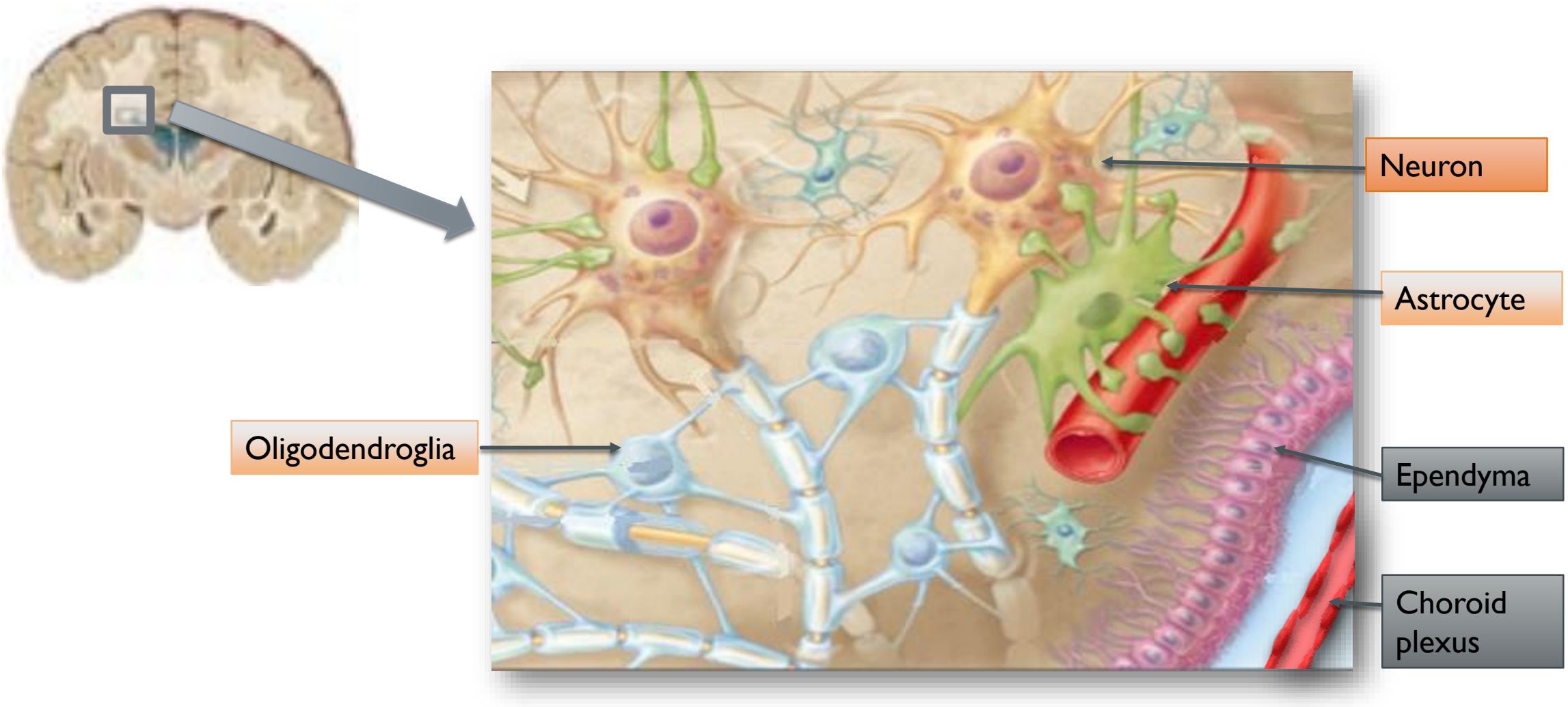
Ependymome Grad II



Anaplastische ependymom Grad III

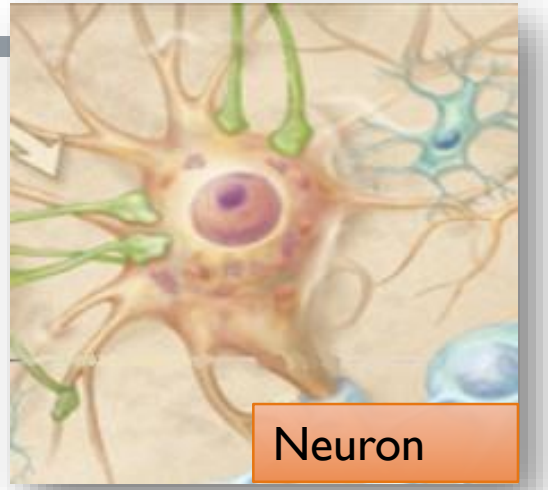


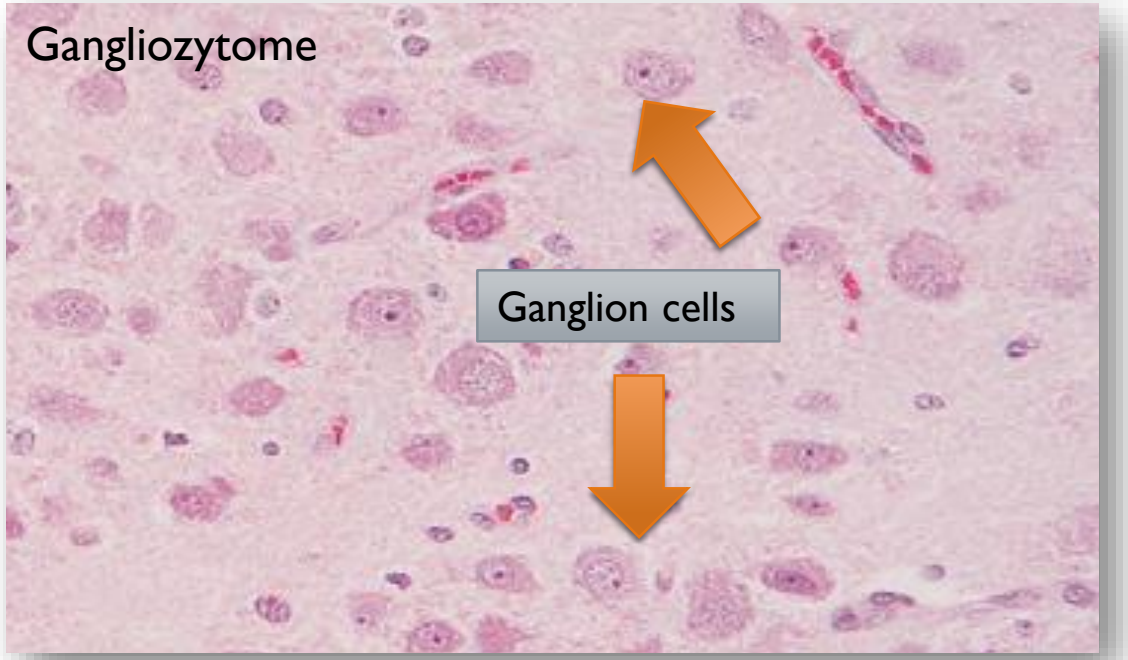
II. NEURONALE/GLIONEURONALE TUMOREN

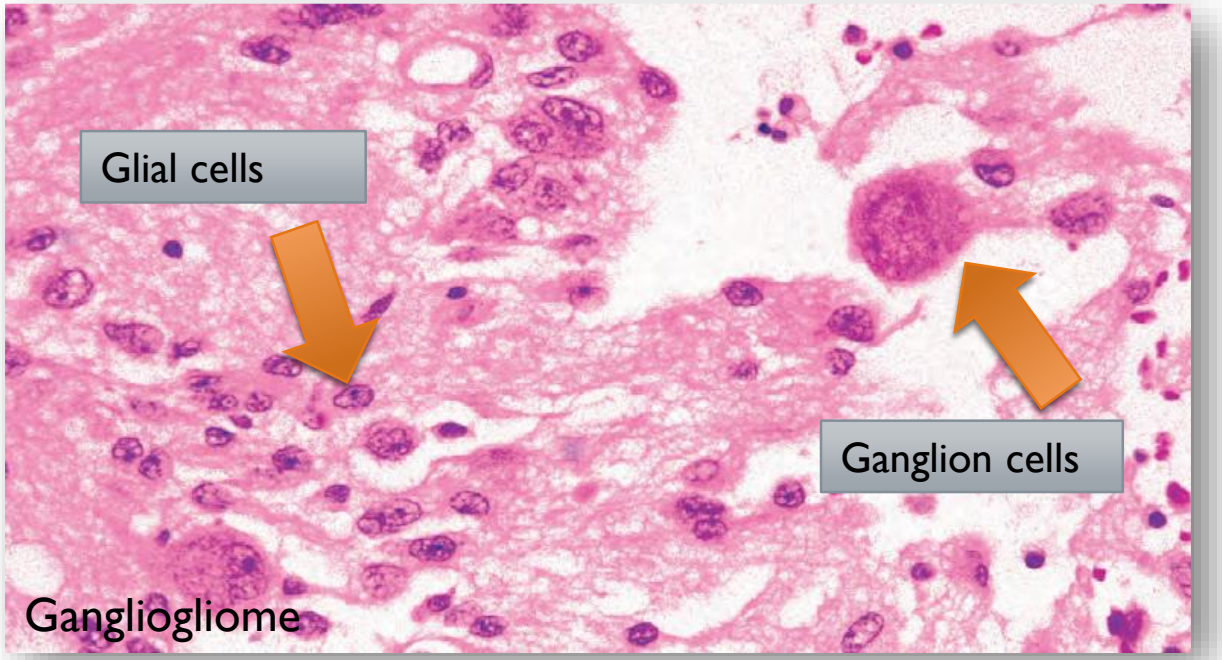


Gangliozytome, Gangliogliome Grad I

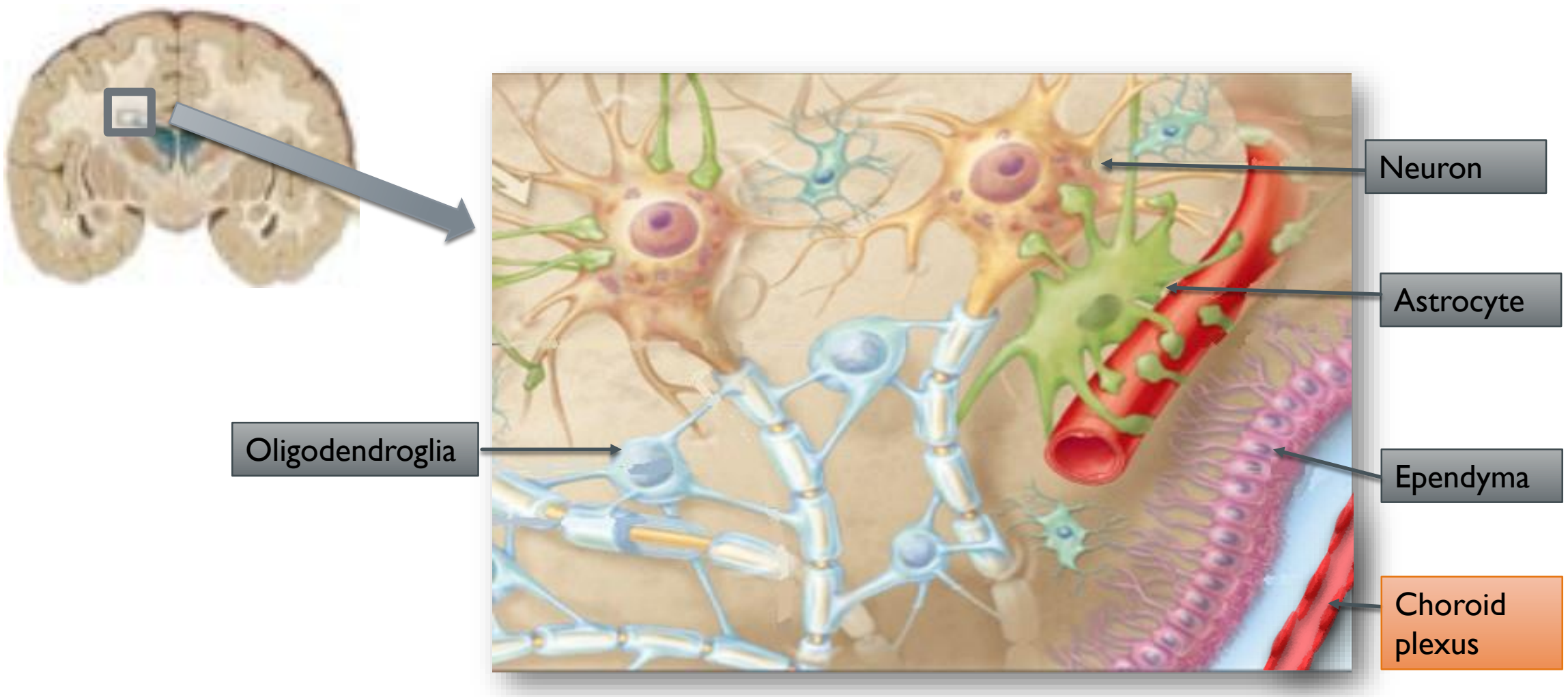
- Temporallappen
- Kindes- und jungen Erwachsenenalter
- neuronaler Differenzierung – *Ganglienzelltumoren*



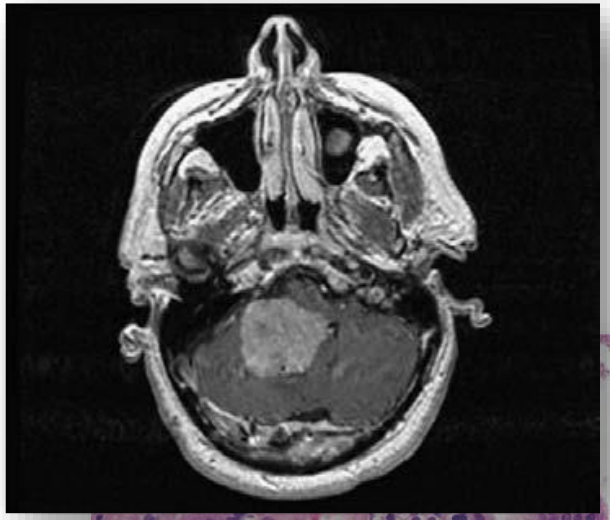


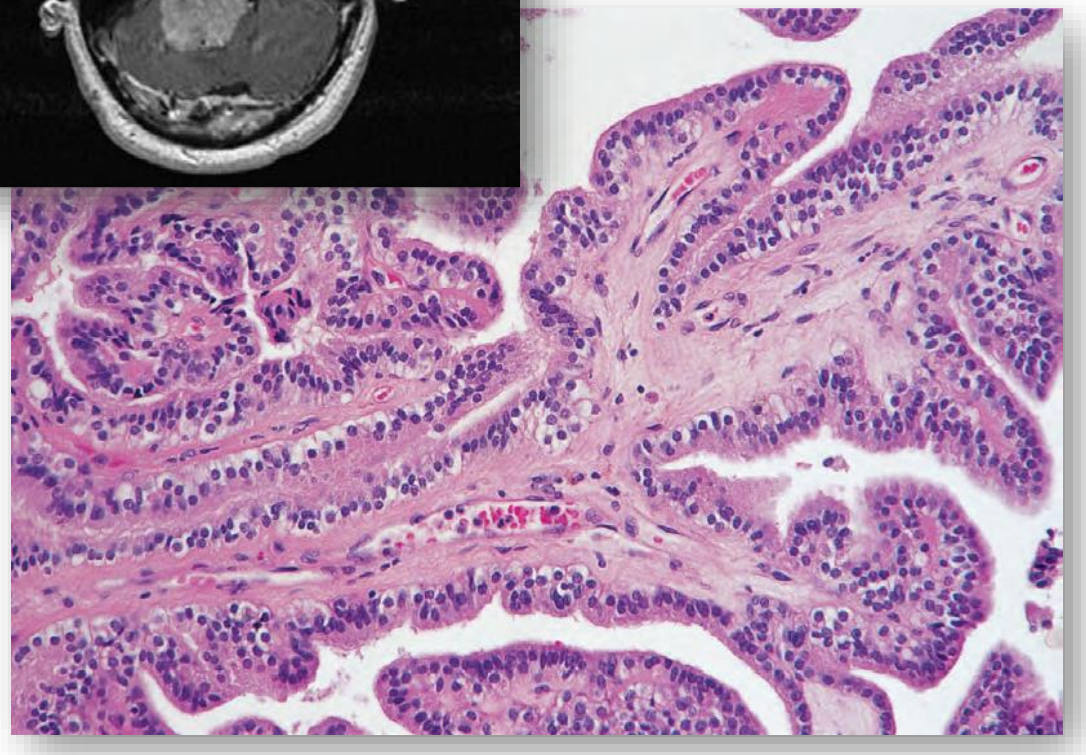


III. TUMOREN DES PLEXUS CHOROIDEUS

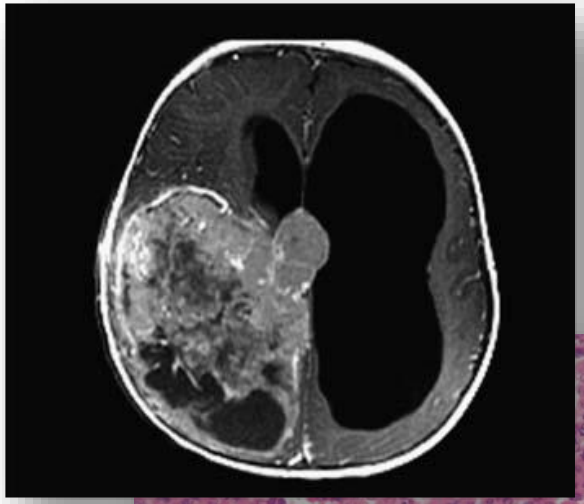


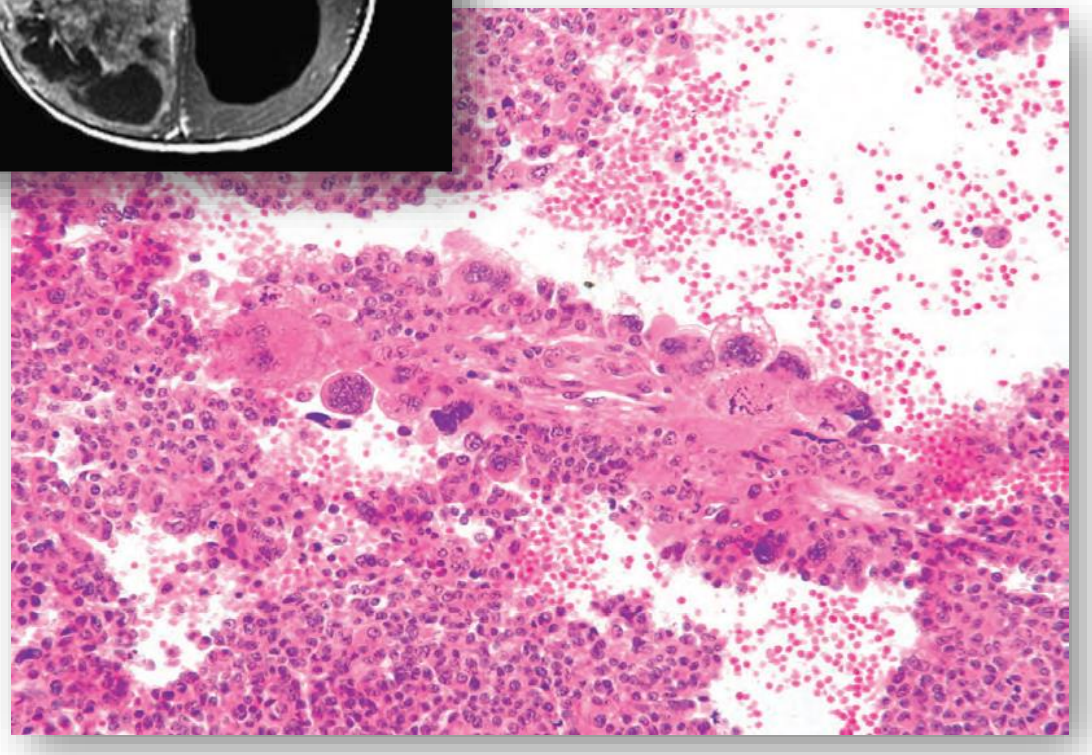
Plexus Choroideus papillom





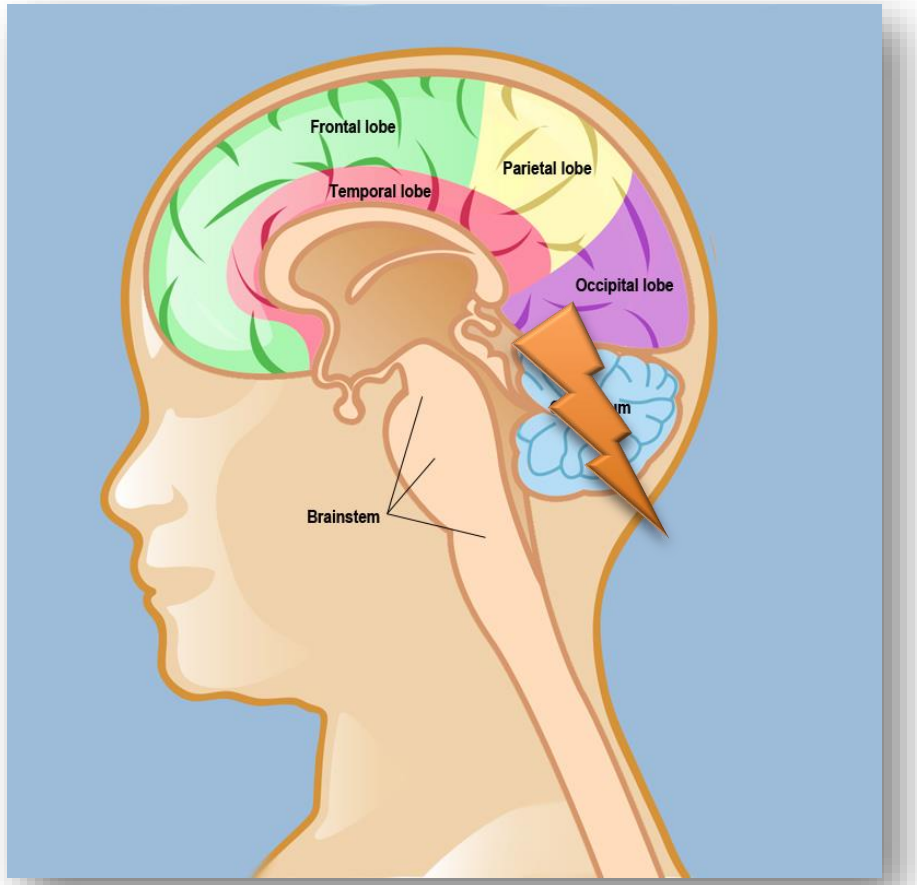
Plexus choroideus karzinome





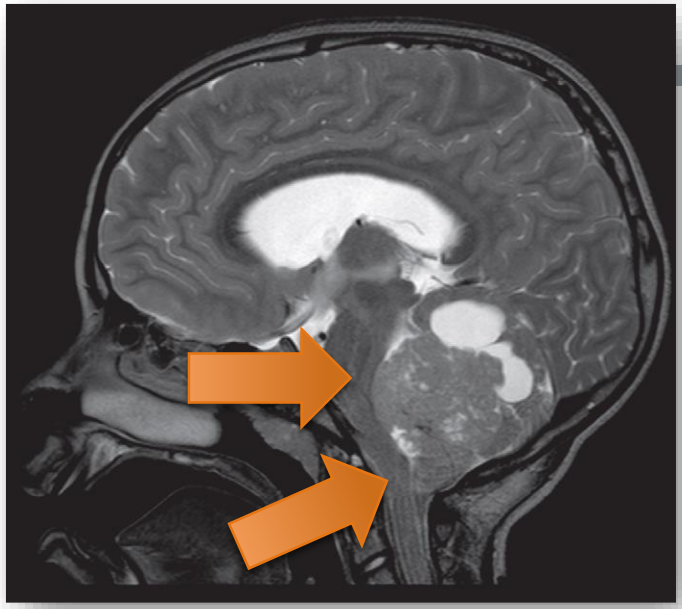
IV. EMBRYONALE NEUROEPITHELIALE TUMOREN

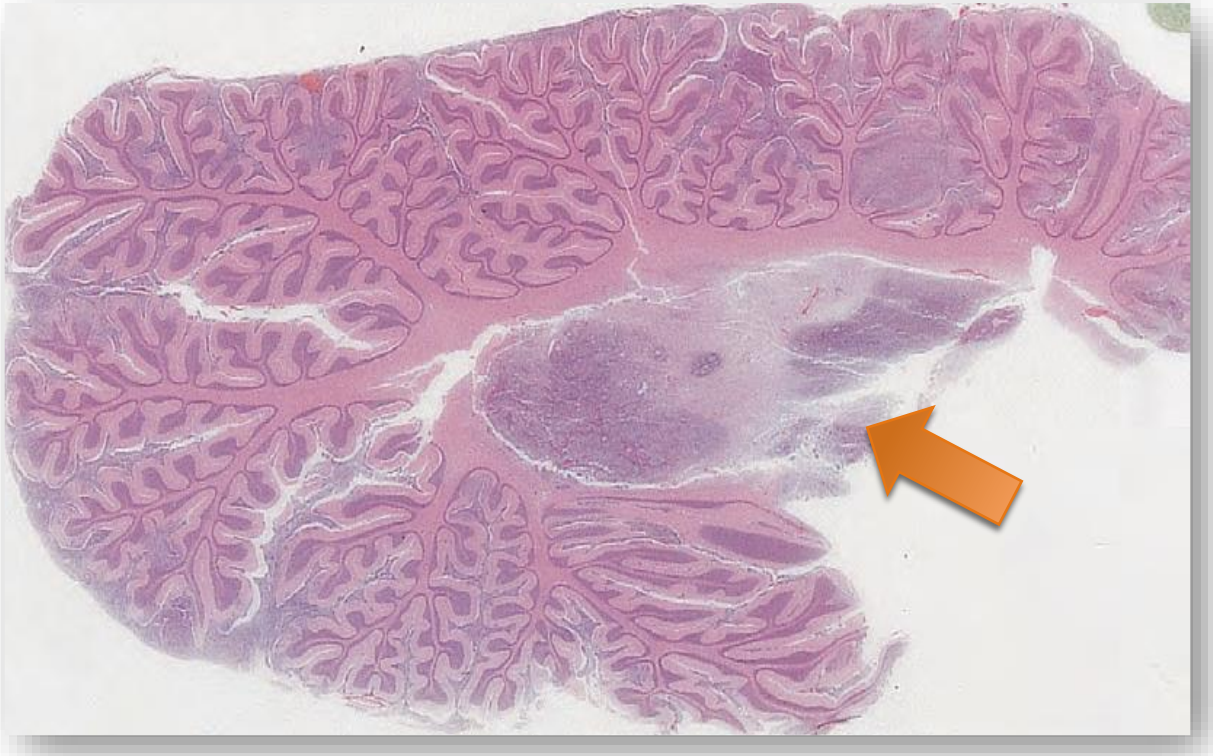
- Kindesalter
- Spinalen Metastasierung
- Undifferenzierte Tumorzellen
- Hohe Mitoserate
- Divergente neuroepitheliale differentiation

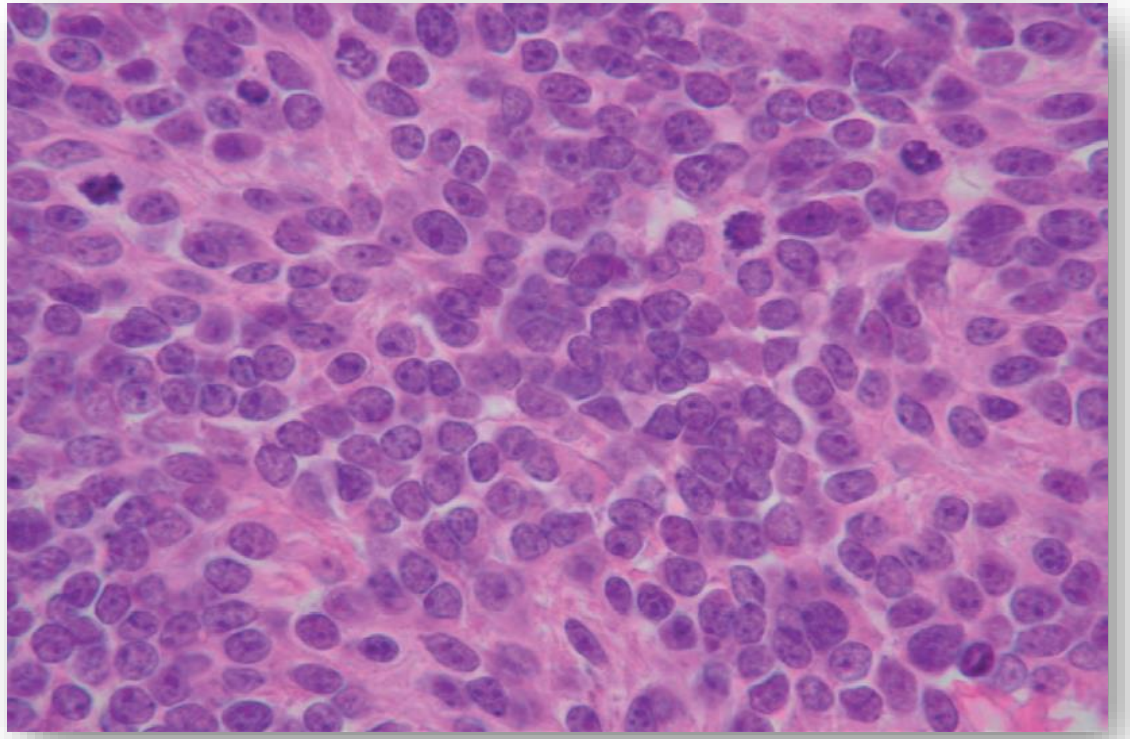


I. Medulloblastom Grad IV

- 20% kindlicher ZNS Tumoren
- Infratentoriell, IV. Ventrikel

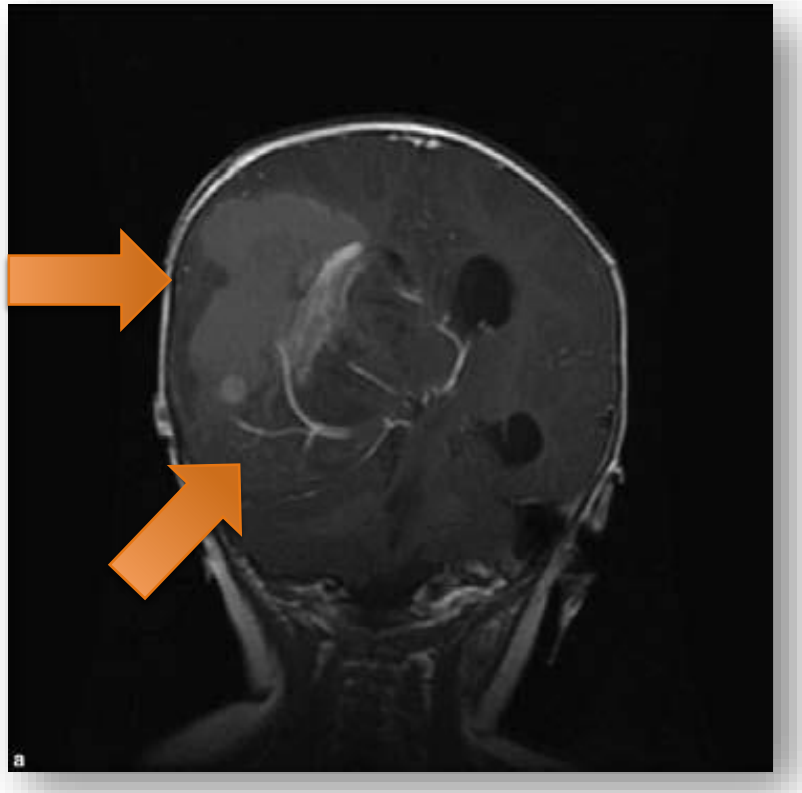


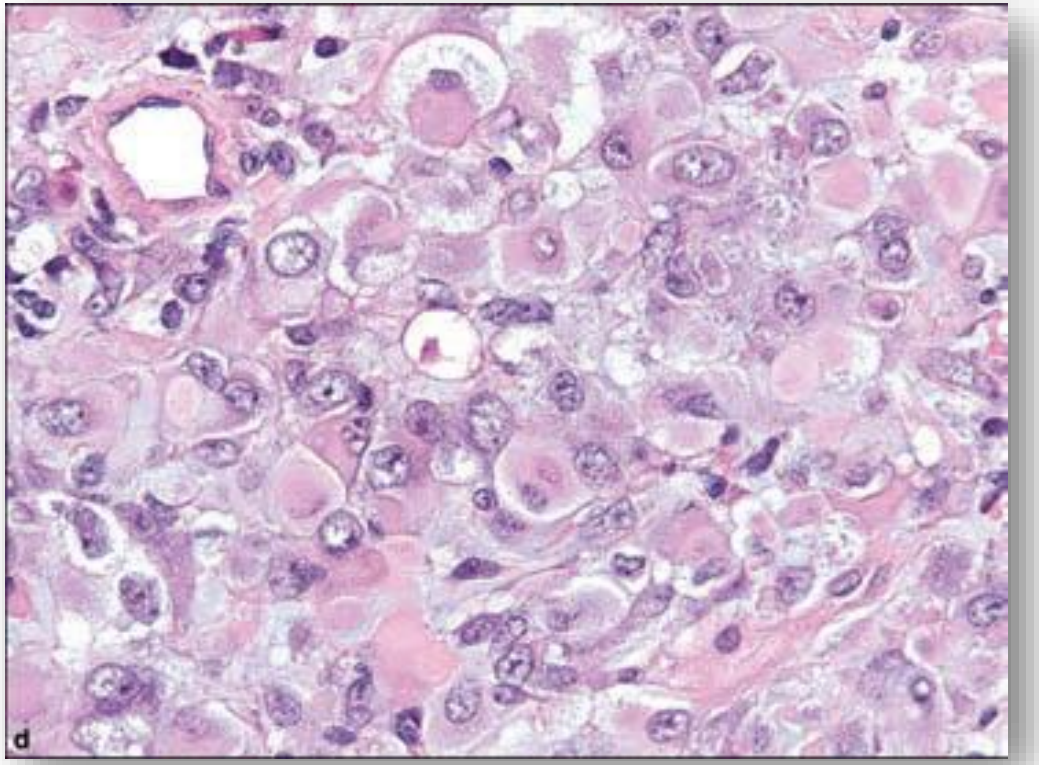




II. Atypischer teratoider/rhabdoider Tumor (ATRT) Grad IV

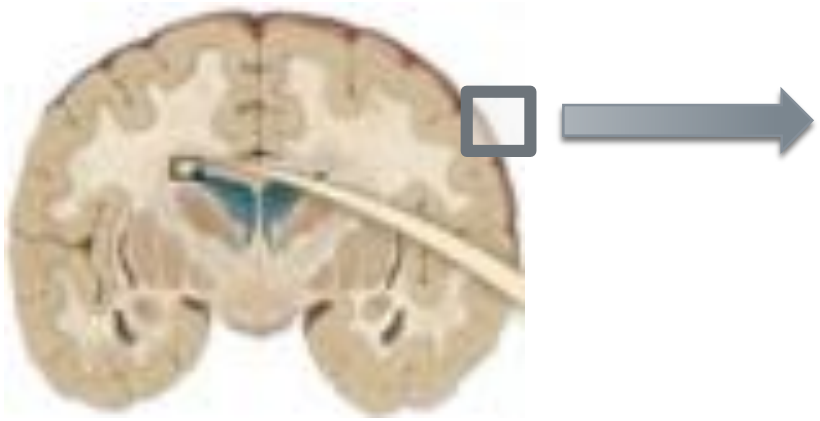
- Kleinkindern <5 Jahre
- Schlechte Prognose
- Nicht nur im hinteren Schädelgrube

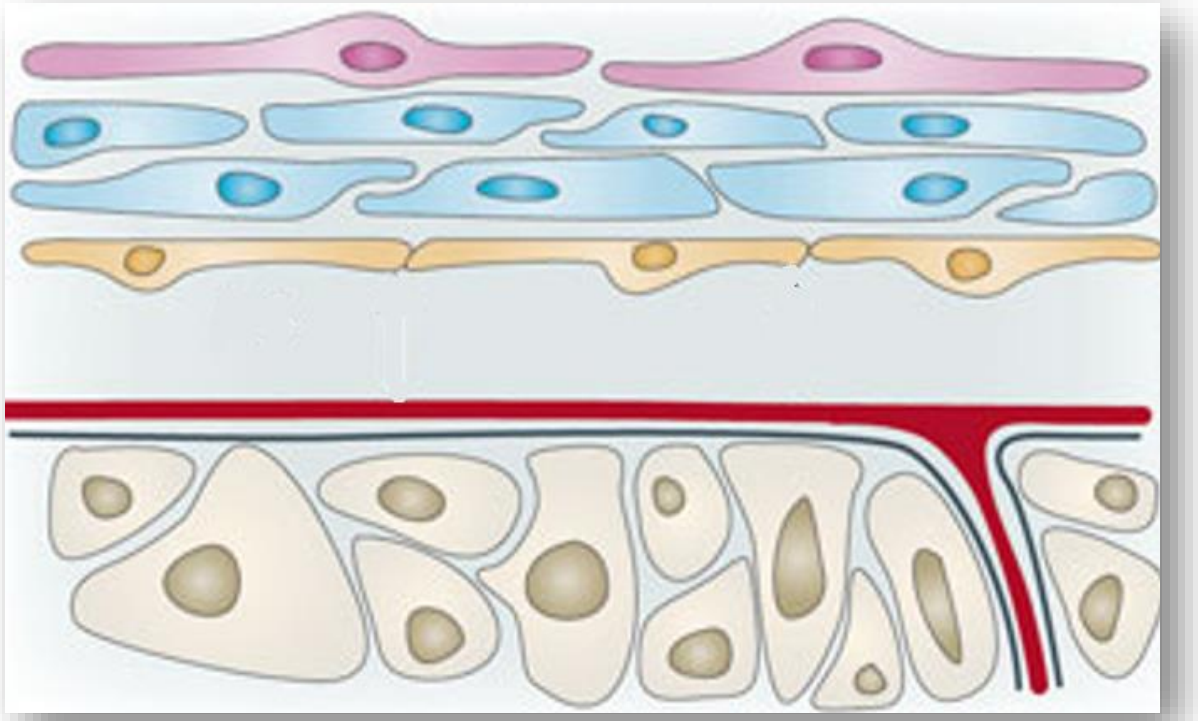




V. TUMOREN DER HIRNHÄUTE

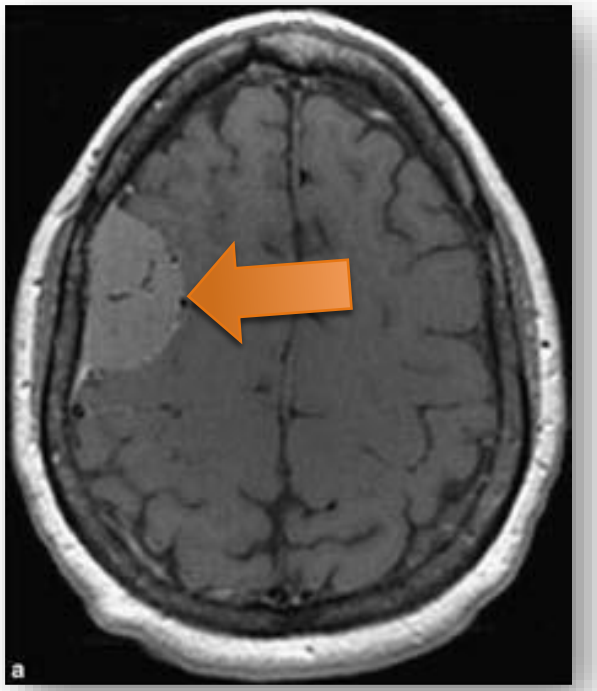
- Arachnoidalen Deckzellen
- die Falx, das Tentorium sowie Keilbein und Kleinhirnbrückenwinkel
- Fokale neurologische Ausfälle

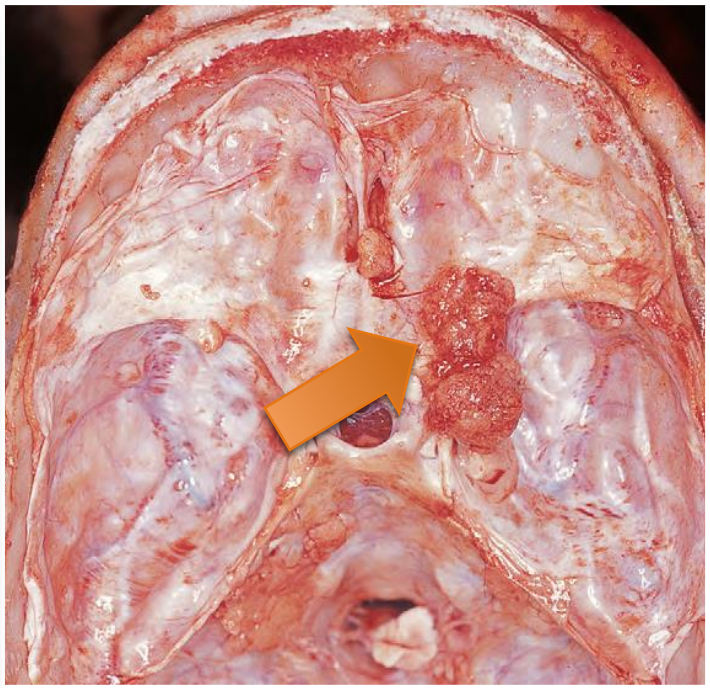


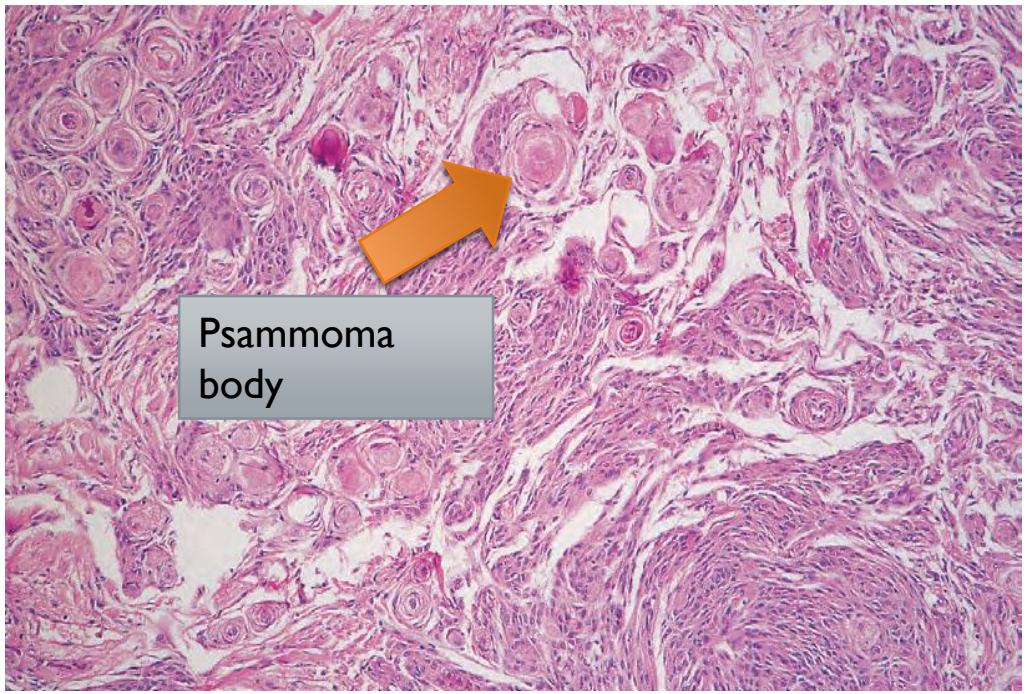


I. Meningiome Grad I.

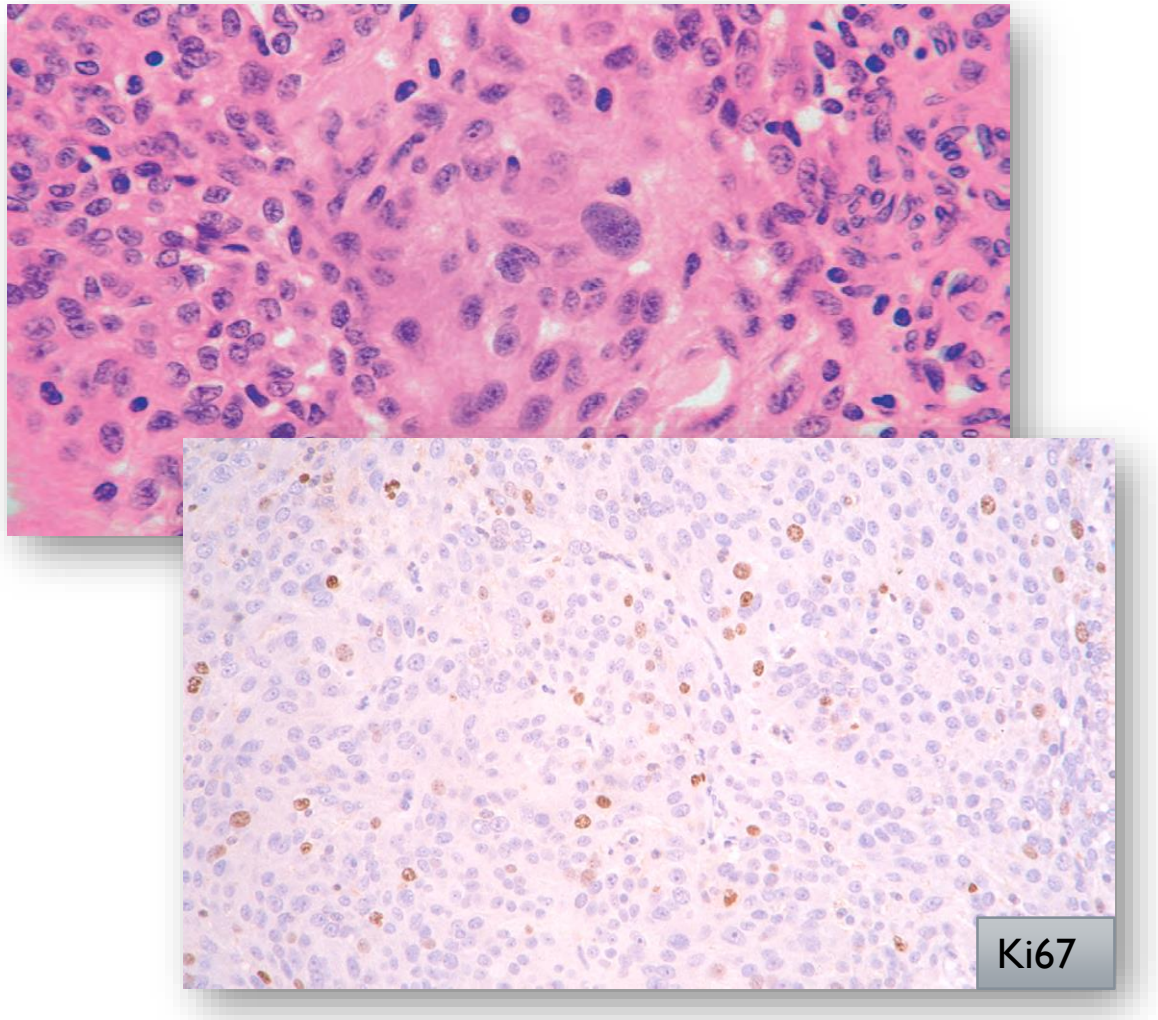
- Tumoren des höheren Erwachsenenalters
- 20-30 % aller intrakraniellen Tumoren
- Viele histologischen Varianten



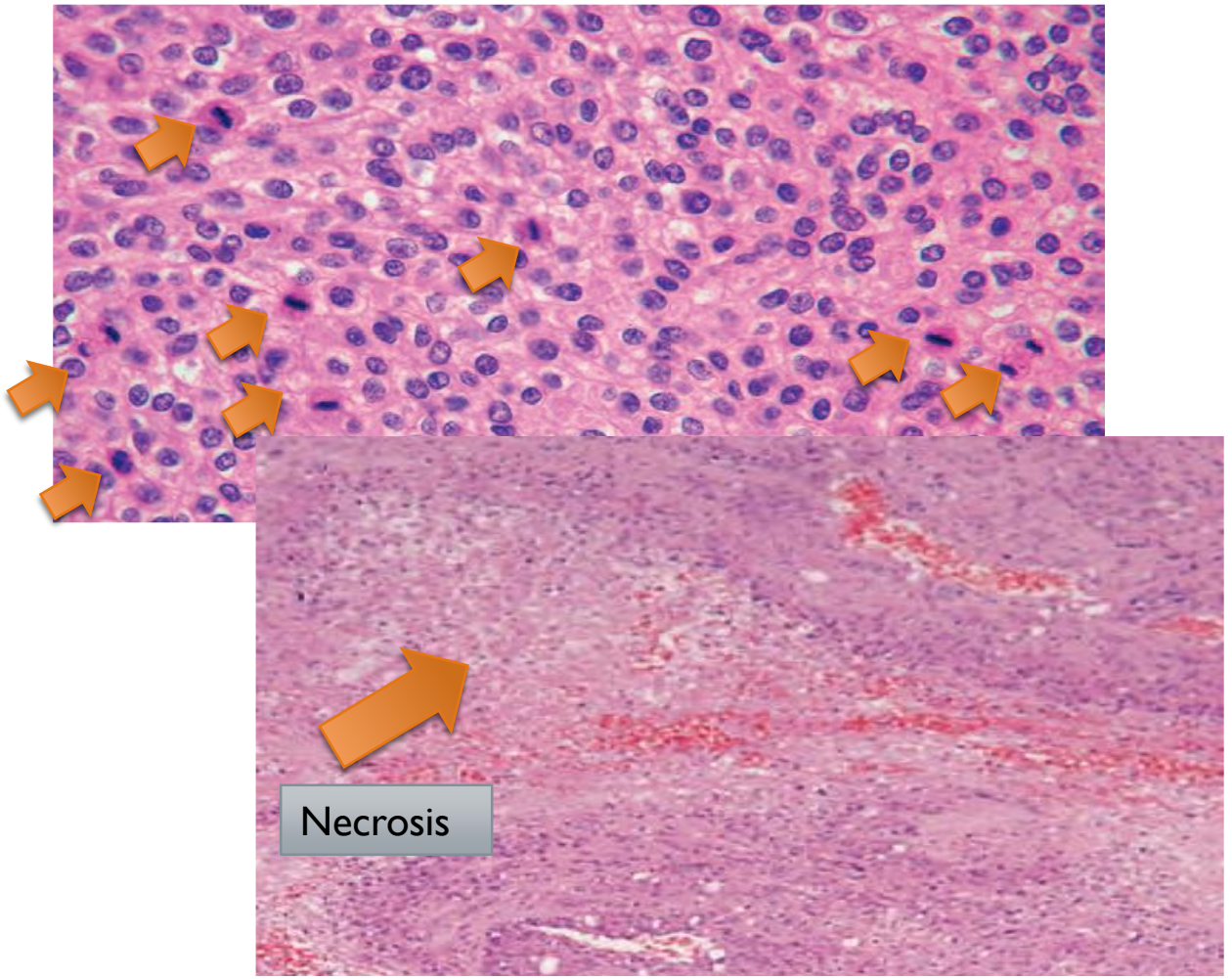




II. Atypische Meningiome Grad II.

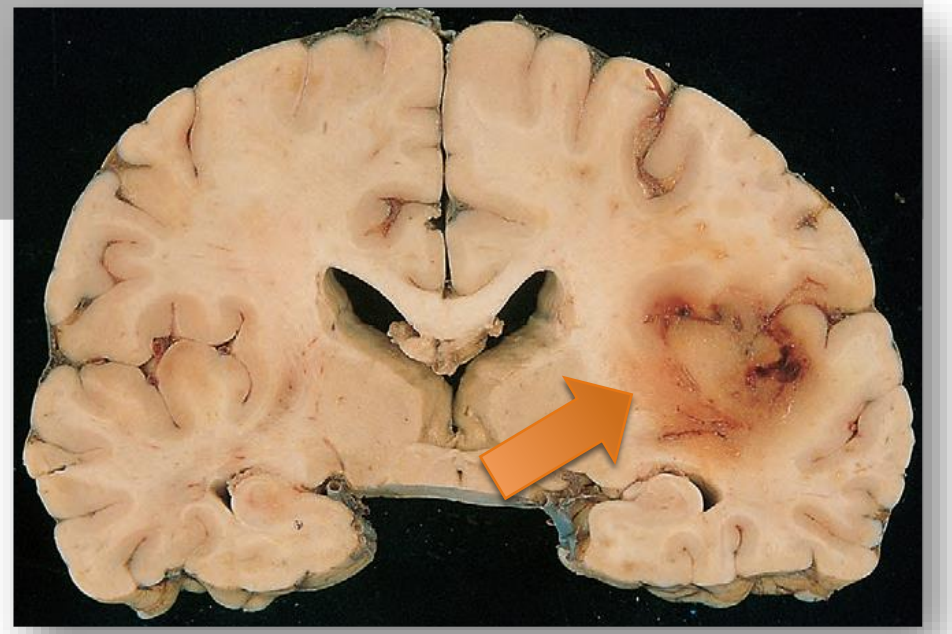


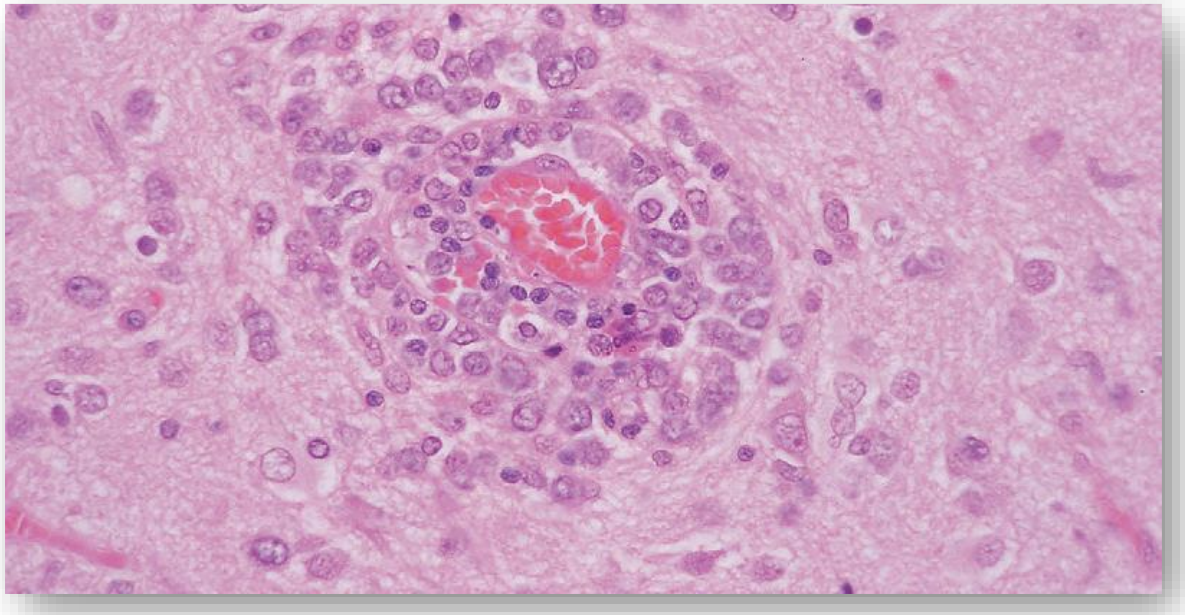
III. Anaplastische Meningiome Grad III.

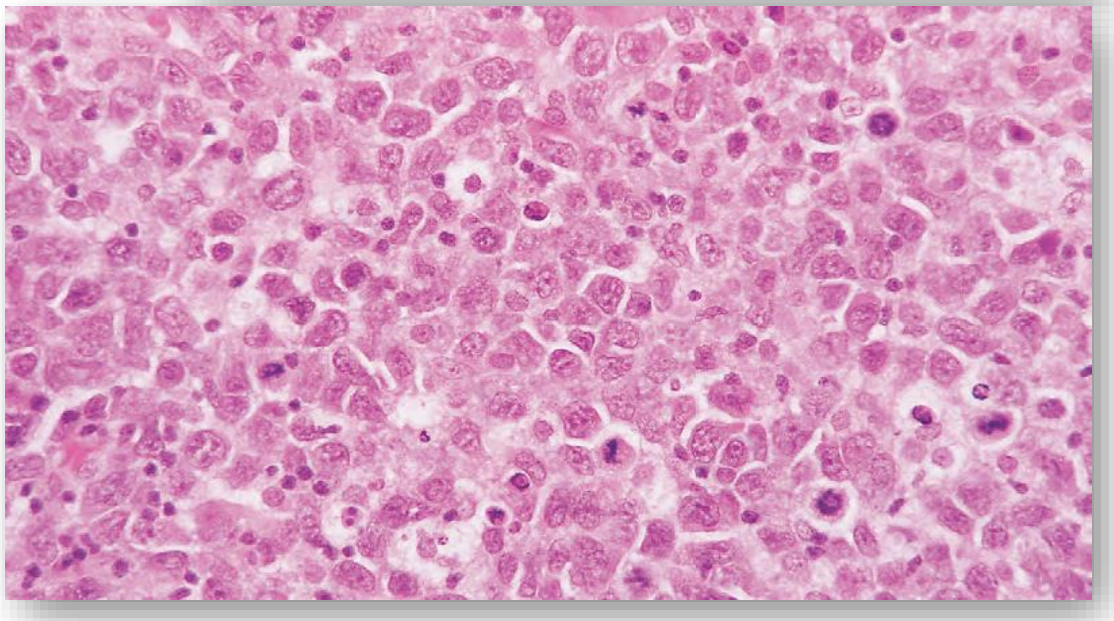


VI. PRIMÄRE ZNS LYMPHOME

- DLBCL typ
- Immunosuppression, AIDS
- EBV

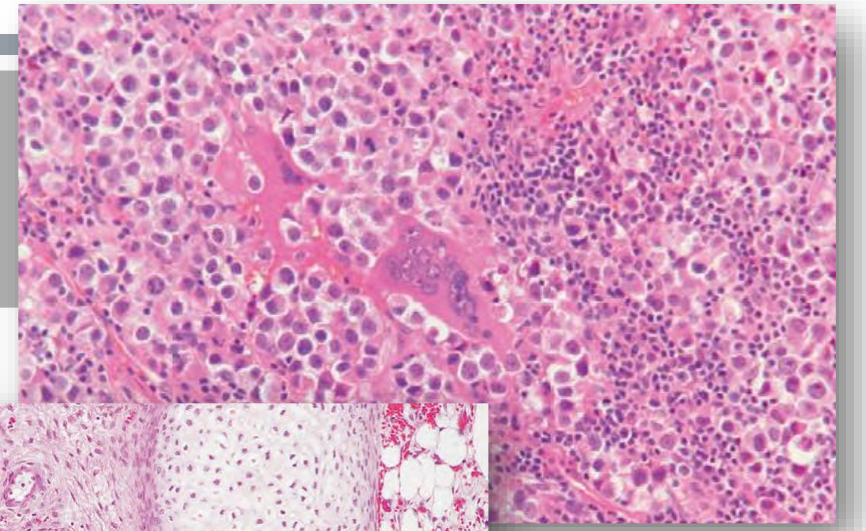


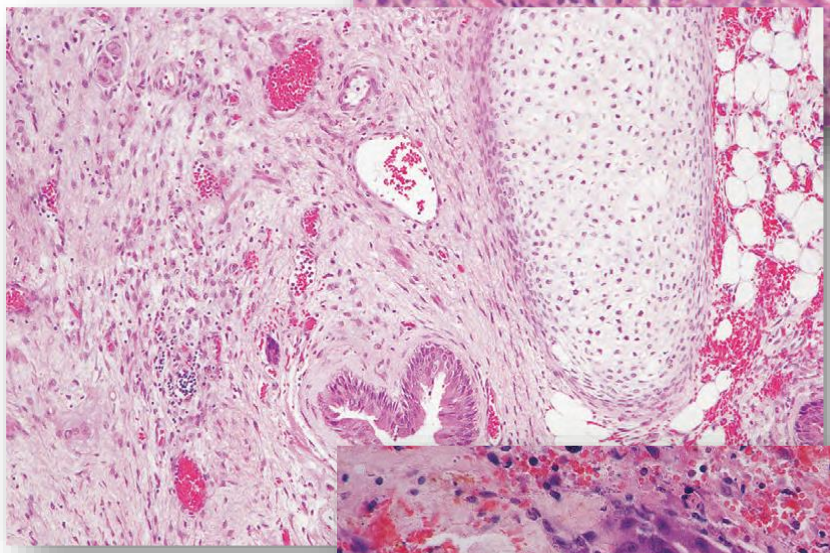


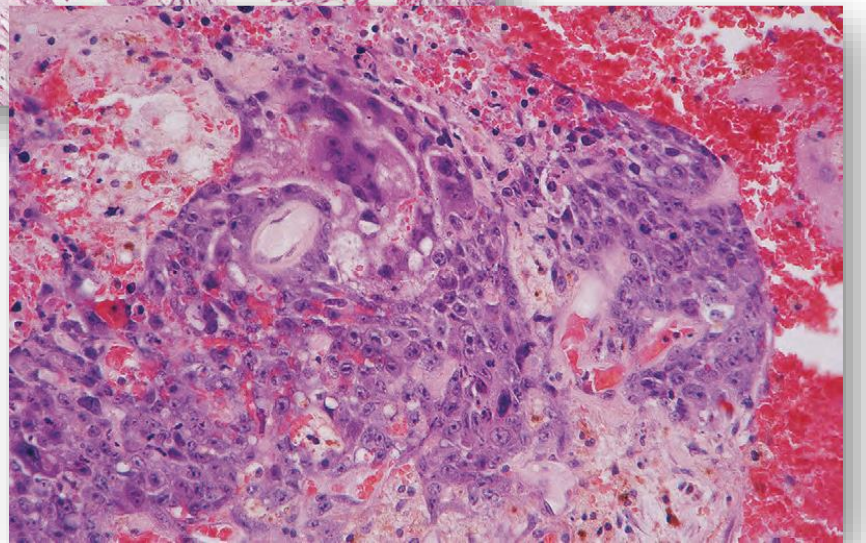


VII. ZNS KEIMZELLTUMOREN

- Germinome - 50%
- Teratome - 20%
- Yolk sac Tumoren
- Embryonale Karcinomen — 5%
- Choriokarcinomen
- gemischte Keimzelltumoren - 25%

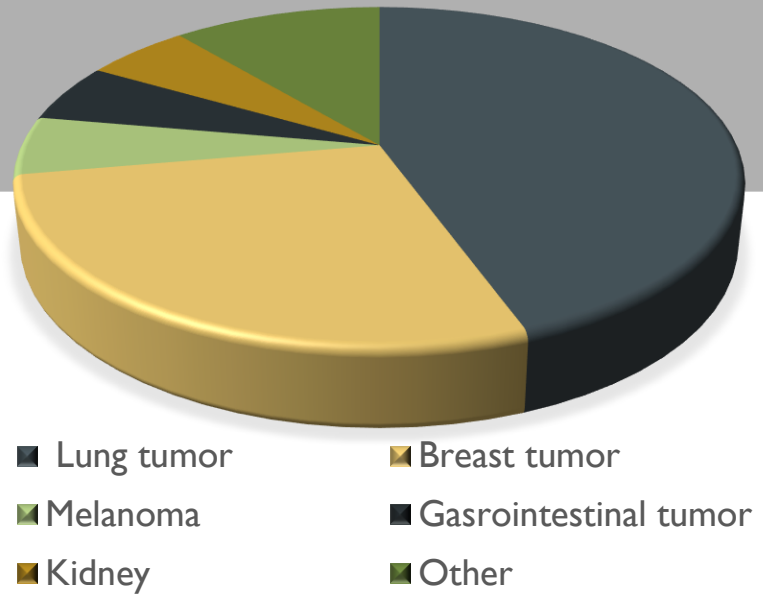


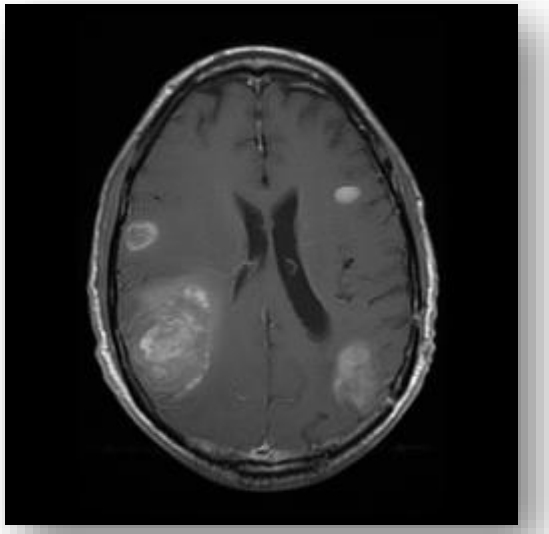


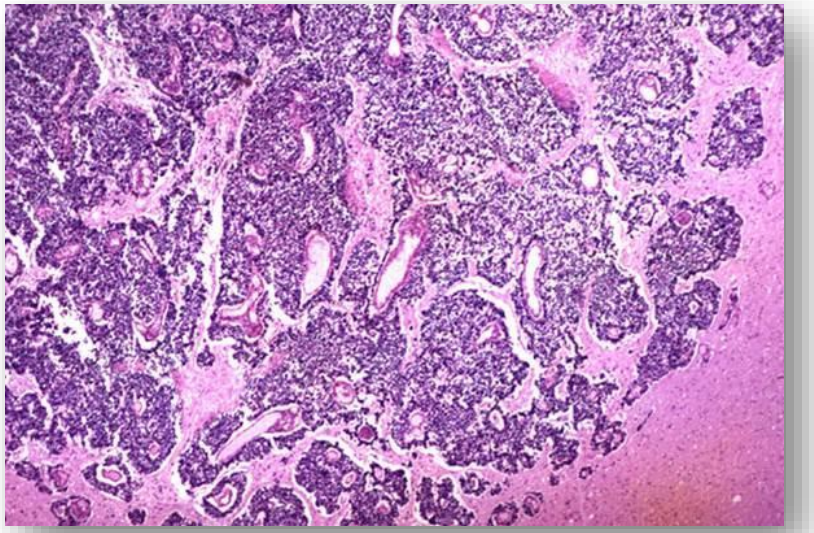


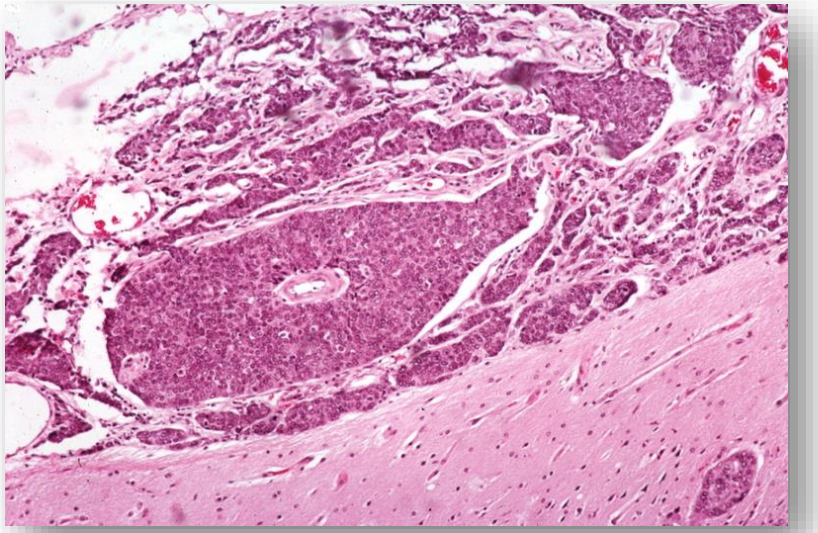
VIII. METASTASEN

- Graue-Weiße Substanz
- Oedema









NEURODEGENERATIVE ERKRANKUNGEN

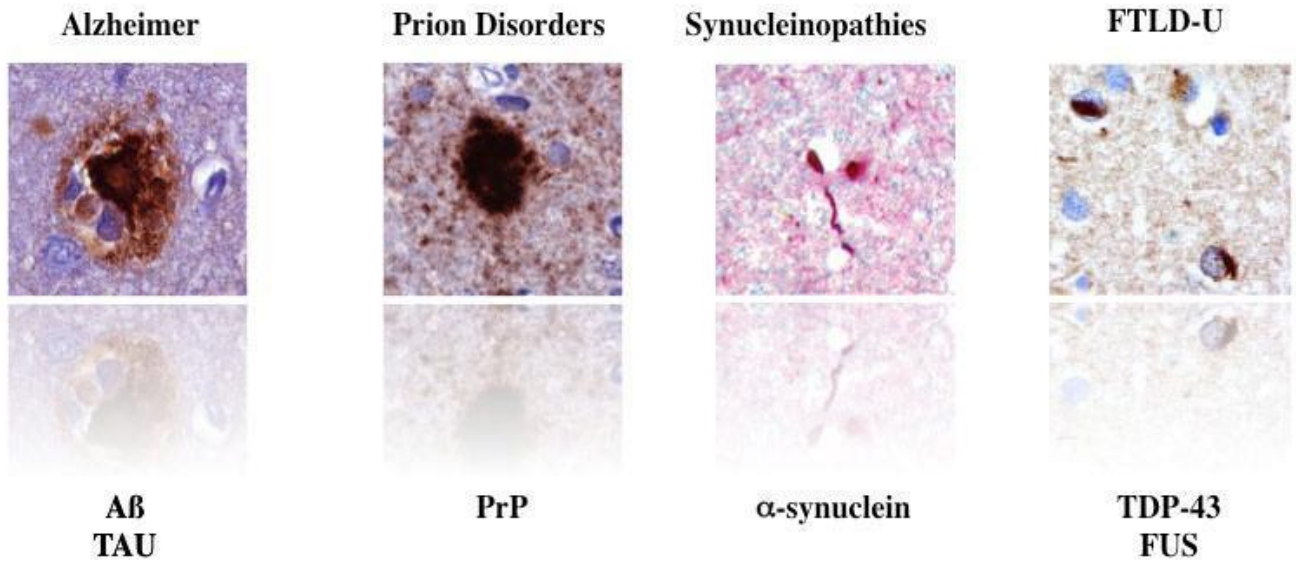
- **Untergang selektiver Nervenzellpopulationen**
- **Genetische Prädisposition**
- **Charakteristische neuropathologische Veränderungen**
- **Proteinaggregatbildung**

Klinische Manifestationen

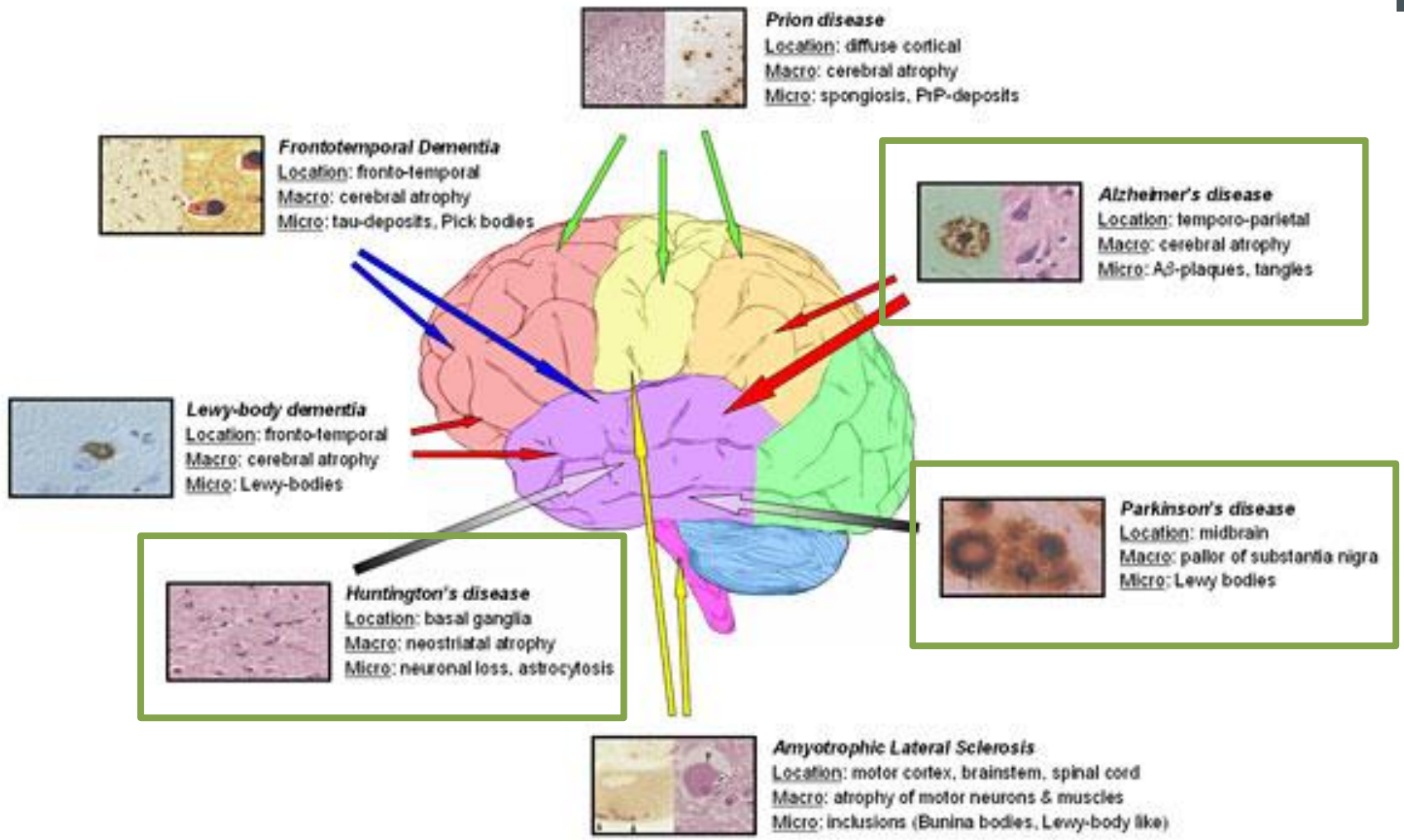
- **Demenz**
  - **M. Alzheimer**
  - **Demenz mit Lewy-Körperchen**
  - **M. Pick**
  - **Prionerkrankungen**
- **Bewegungsstörungen**
  - **M. Parkinson**
  - **M. Huntington**
- **Lähmungen**
  - **Amyotrophe Lateralsklerose (ALS)**
- **Ataxie**
  - **Spinozerebelläre Ataxien (SCA)**
- **Autonome Dysfunktion**
  - **Multisystematrophie (MSA)**

NEURODEGENERATIVE ERKRANKUNGEN

- **A-beta - APP-Fehlprozessierung**
  - **Aggregation des entstandenen Fragmentes**
- **Tau: - Hyperphosphorylierung**
  - **Aggregation des phosphorylierten Tau**
- **Prionprotein - Konformationsänderung**
  - **Aggregation der pathologischen Konformation**
- **Huntingtin; Ataxin/SCA, Frataxin - Trinukleotidrepeat-Expansion**
  - **Aggregation des elongierten Proteins**
- **Amylin - Fibrillogenet. Faktoren**
  - **Aggregation von Amylin (IAPP)**
- **Synuklein - ? Fibrillogenetische Faktoren ?**
  - **Aggregation von Synuklein**



NEURODEGENERATIVE ERKRANKUNGEN



- Robbins Basic Pathology, 9th Edition
- Intensivkurs – Allgemeine und spezielle Pathologie