AVMs of the quadrigeminal plate

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No Conflict of Interest
Mesencephalon (midbrain) is structurally a relatively simple part of the brain in which the fundamental relationships between the basal and alar plates are preserved.

Basal plates form a neuron-rich area: the tegmentum (location of the somatic efferent nuclei of cranial nerves III and IV)

Alar plates form sensory part of midbrain (tectum) which subserves functions of vision and hearing.
In response to the localized expression of En-1 and Pax-7, neuroblasts migrating toward the roof form two prominent pair of bulges: the corpora quadrigemina.

Tectal (quadrigeminal) plate is the portion of the midbrain tectum upon which the superior and inferior collicula sit.
Caudal part (inferior colliculi) is simple in structure and functionally part of the auditory system.

Cranial part (superior colliculi) is more complex in architecture and are an integral part of the visual system and are a synaptic relay between the optic nerve and the visual occipital cortex.

Connexions between superior and inferior colliculi help coordinate visual and auditory reflexes.

**Brain Anatomy – Midbrain**

Includes:
- **Corpora Quadrigemina (tectal plate)**
  - **Superior Colliculus**: Visual reflex center (turns eyes and head in response to visual stimulus)
  - **Inferior Colliculus**: Auditory reflex center (turns eyes and head in the direction of a sound)
- **Cerebral Peduncles**
Collicular artery originates from P1
Turns around the pontomesencephalic sulcus -> gives off branches to the crus cerebri
Arrives at tectal plate and divides into a
superior branch for the superior colliculus
inferior branch or intercollicular artery for the inferior colliculus and that participates extensively to the tectal network « hiding the subjacent veins » (Duvernoy 1975)

Accessory collicular artery for the lateral part of the superior colliculus
anastomoses with the vessels of the collicular brachium (Pmchor art)
Balance between Pmed Chor a and Collicular a
Balance between Collicular a and Sup cereb a

*Arterial network of inf colliculus mainly from sup Cereb a (+/- from collic a)
*Vascular density at intercollicular sulcus
*Vascular network of sup colliculus mainly from collicular a
*superior edge of sup colliculus often co-vascularized by Pmed Chor a
Mesencephalon: posterior view

1: pulvinar
2: pineal gl
3: sup colliculus
4: inf colliculus
5: precentral v
6: v of brachium conj
7: sup cereb art
8: Collic art
9: post medial chor art
10: sup median Collic v

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Numerous anastomoses on each side but few anastomoses between left and right networks (avascular zone along vertical intercollicular sulcus)
Green: collicular arteries
red: medial superior cerebellar arteries
blue posteromedial choroidal arteries
Superficial veins of colliculi:
less numerous than arteries

Mainly occur at periphery of colliculi and form
-superior median collicular vein
-inferior medial collicular vein

Peripheral collicular veins:
Emerge from the surface of colliculi

Central collicular veins:
Emerge near the midline

Principal lateral veins and the central collicular veins drain

-part of red nucleus
-almost all trochlear and oculomotor nuclei
Colliculi
Venous network

1: Pineal gland
2: 
3: sup colliculus
4: inf colliculus
5: Sup med collicular v
6: intercollicular v

Naidich & Duvernoy
14: latero mesenceph v

17: v of sup cerebell ped
1: longitudinal segment of precentral cerebellum
2: dorsal segment of precentral cerebellum
3: sloping segment of precentral cerebellum
4: VG
5: ICV
6: BV
7: Sup colliculus
8: superior cerebellar peduncle
9: precentral lobule
10: v of the cerebellar culmen
10': v of the cerebellar culmen
11: intercollicular v
12: sup medial collicular v
13: Sup colliculus
14: Inf colliculus
15: Trochlear n
Trans-Mesencephalic Arteries and Veins
Angiographic Aspects in Tectal Vascular Lesions

P. Lasjaunias¹, K. Terbrugge², and I. S. Choi³

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² Department of Radiology, Toronto Western Hospital, Toronto, Ontario, Canada,
³ Department of Neuroradiology, Bellevue Hospital, New York City, N.Y., U.S.A.

Transmesencephalic supply seen in 21% of mesencephalic AVMs Lasjaunias 1994
Axial: Superior colliculus

Axial: Inferior Colliculus
Mesencephalo-diencephalic angioanatomy in arteriovenous malformations. Endovascular management of transmesencephalic vs subependymal supply in 954 cases between 1982 and 1994

J Xavier, S Suthipongchais, J Al-Warth, H Alvarez, G Rodosthenus P Lignanias

Service de Neuroradiologie Vasculaire Diagnostique et Therapeutique, Hopital Bichetre, 78, rue du General Leclerc, F 94273 Le Kremlin Bicetre, France

Anterior thalamic perforators arise proximally from the caudal division of ICA and run vertically or slightly horizontally

Posterior thalamo perforators arise from P1 and have a nearly vertical supendymal course parallel to the basilar artery main direction

Lateral A° projection: above P2 towards choroid region
AP A° projection: close to midline between PCAs

Mesencephalic perforating arteries have an horizontal course, perpendicular to the basilar artery main direction
AP A° projection: close to midline
Lateral A° projection: below/ at level of P2 (diff diag with circumflex a that have more post course)
Vascular Anatomy allows recognition and differential diagnosis of different vascular malformations

**Tectal AVSs**
- Collicular a
- Sup cerebellar a
- Transmesencephalic a
- Tectal vein
- Precentral cerebellar v
- VG
- Basal v of Rosenthal

**Choroidal AVSs**
- Anterior choroidal a
- Posterior choroidal a
- Subependymal a
- Choroidal v
- VG
- Basal v
- Reflux into contributors of VGAM

**Choroidal VGAMs**
- Long circumferential a
- Anterior choroidal a
- Posterior choroidal a
- Chor br of ACA
- Limbic circle
- Subependymal a
- Medial vein of prosencephalon
- Epsilon aspect of deep drainage
- Falcorial sinus

**Dural AVFs**
- Dural feeders
  - (MMA, APhA, occip a, Sup cerebellar, PCA, ICA…)
- VG
- Veins of the region
Brainstem AVMs: Rare lesions (2-6% of all brain AVMs)

**Rare and small (surgical) series of brainstem AVMs**

Drake 1986  
15 pts  
7 « midbrain » series about PF AVMs: 61/66 Hb (but no precision of localization)

Solomon & Stein 1986  
12 pts  
4 « tectal plate region » all Hb

Yasargil 1988  
14 pts  
9 « dorsal/dorsolateral mesencephalon » revealing Hb in 6 operated pts no info about revealing sympt in non operated pts
Yasargil brainstem AVMs

There are 6 operated and 8 unoperated cases in the present series.

Three of the operated cases were in the dorsal mesencephalon. Two of these were small and located around the left superior colliculus. The third was located within the pineal body. In these cases a hematoma had developed a plane of cleavage and extended down towards the habenular commissure. These 3 cases were operated upon successfully and the only residual deficit was a temporary Parinaud's syndrome in one case (No. 1, 2, 3, Table 4.132, Figs 4.174 and 4.175 in Vol. IIIB, see also Figs 3.31, 3.32 and 3.100, Vol. IIIA). It is interesting to note that these AVMs were barely seen on preoperative angiography and were identified by the early appearance of a dorsal mesencephalic vein.

Three other cases were in the dorsolateral mesencephalon

One case (No. 6, Table 4.132) was unusual, as multiple AVMs were present around the mesencephalon,

In 2 further cases the AVM was in the right paracollicular area involving the nerves IV and V, but fortunately epipial and partially subpial. The feeders were coagulated until the draining vessels turned to dark blue then the lesion was removed. The postoperative course was uneventful in both cases.

Out of 189 patients with deep central AVMs, 153 have been operated and 36 not operated. In 6 patients with parasplenic, 4 with callosal, 1 with trigonal-plexal and 3 with dorsal mesencephalic AVM, the lesions could have been surgically removed but the patients or their relatives refused surgery.

Of nonoperated patients, a total of 19 underwent irradiation

<table>
<thead>
<tr>
<th>No.</th>
<th>Age</th>
<th>Sex</th>
<th>Diagnosis</th>
<th>Grade</th>
<th>Status</th>
<th>Treatment</th>
<th>Follow-up</th>
</tr>
</thead>
</table>
A 60-year-old male patient presented with subarachnoid hemorrhage and Parietal syndrome had a small enhancing nodule located over the left superior colliculus shown on CT (white arrow). B Postoperative CT 2 months after removal of the lesion. C-D Frontal and lateral vertebral angiography showed a barely visible nidus (black and white arrow) with draining vein. Omolateral vertebral angiography only early filling of the v. Galen and the straight sinus was seen. The 5 x 5 mm AVM was explored and removed through a supracerebellar approach. No additional neurological deficit after operation. Total disappearance of Parietal syndrome within 6 months.

Fig. 3.3A-D

1. The 60-year-old patient had subarachnoid hemorrhage.
2. Cerebral and lateral vertebral angiography were normal. AP vertebral angiography showed a small nidus with an early draining vein located over the left frontal mesencephalon (arrow). Surgery confirmed a small AVM located over the left superior colliculus. The AVM was radically removed.
3. Postoperative vertebral angiography.
4. Preoperative CT showed a small enhancing lesion in the area of the left superior colliculus (arrow).
5. The postoperative CT showed no parenchymal deficit in the mesencephalon. The postoperative course was uneventful. No Parietal syndrome.

Yasargil
<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>Number of pts</th>
<th>Main Location</th>
<th>Outcome of AVMs</th>
<th>Additional Information</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drake</td>
<td>1986</td>
<td>15</td>
<td>7 « midbrain »</td>
<td></td>
<td>series about PF AVMs: 61/66 Hb</td>
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<td>Solomon &amp; Stein</td>
<td>1986</td>
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<td>4 « tectal plate region »</td>
<td></td>
<td>all Hb</td>
</tr>
<tr>
<td>Yasargil</td>
<td>1988</td>
<td>14</td>
<td>9 « dorsal/dorsolateral mesencephalon »</td>
<td>revealing Hb in 6 operated pts</td>
<td>no info about revealing sympt in non operated pts</td>
</tr>
<tr>
<td>Lawton &amp; Spetzler</td>
<td>1995</td>
<td>10</td>
<td>NA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spetzler &amp; Martin</td>
<td>1986</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nozaki</td>
<td>2006</td>
<td>25</td>
<td>9 « dorsal midbrain »</td>
<td>9 initial Hb. 3 repeated Hb</td>
<td></td>
</tr>
<tr>
<td>Kelly &amp; Steinberg</td>
<td>2008</td>
<td>29</td>
<td>NA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Han &amp; Lawton</td>
<td>2015</td>
<td>29</td>
<td>6 « posterior midbrain »</td>
<td>Hb in 23 pts (79,3%)</td>
<td></td>
</tr>
<tr>
<td>Madhugiri &amp; Steinberg</td>
<td>2017</td>
<td>39</td>
<td>11 « posterior midbrain »</td>
<td>Hb in 36 pts (92,3%)</td>
<td></td>
</tr>
</tbody>
</table>
Haemorrhage SAH / IVH

No precise information about the annual hemorrhagic rate of tectal AVMs

Annual Hb risk Brainstem AVMs 15,1% - 17,5%
Koga 2011, Nozaki 2005, Han 2015, Madhugiri 2017

Annual Rebleeding risk Brainstem AVMs 14,2%
Nozaki 2005

Severe Neurological Symptoms
somnolence -> coma
III IV palsy
ataxia
hemiparesis
hemihypoesthesia
vertical gaze palsy
cerebellar syndrome
auditory troubles
...

Intraparenchymatous
Posterior fossa AVMs

75-95% cerebellar
5-25% brainstem  *Drake 1986; George 1992; Spetzler 2015*

The supracerebellar infratentorial approach is usually used for AVMs of the tectal plate and the superior surface of the cerebellum, provided they do not extend above the incisura (need for a posterior interhemispheric approach)

*Solomon RA and Stein B; Neurosurgery 1986
Han SJ and Lawton M; J Neurosurg 2015*

Occipital interhemispheric transtentorial approach

*Mc Laughlin N and Martin NA; World Neurosurg 2014*
Clinical & morphological outcomes after therapeutic management (brainstem! Tectum?)

**Surgery**

Better outcome if epipial than subpial

**Total excision vs retrograde venenoidal microsurgical obliteration**

« occlusion in situ technique »

- Postsurgical outcome influenced by presurgical condition
- Rate of worsening/death greatest with posterior midbrain and anterior pontine AVMs
- Post surgical permanent complication rate is 13.8-25%

*Han & Lawton 2015*

Steiger HJ, Hänggi D 2009

Han & Lawton 2015,
Madhugiri & Steinberg 2017
(already Yasargil 1988...)

*Han & Lawton 2015*

Nozaki 2006

Han&Lawton
Example of OP lateral medullary AVM
Rare and small (endovascular) series of brainstem AVMs

A midbrain arteriovenous malformation at quadrigeminal plate completely obliterated by embolization

Nakahara & al; No Shinkei Geka 1993

18 y male. Hb => tetraparesis. Recovery 6 months with residual R hemisensory disturbance and Parinaud
2 sessions of E° with EVAL after provocative tests (Amytal and Xylocain). Left oculomotor nerve palsy. Cure of AVM

Endovascular treatment of brainstem arteriovenous malformations: safety and efficacy

HM Liu & al, Neuroradiology 2003

1 dorsal midbrain
1 central and dorsal midbrain
1 pulvinar and dorsal midbrain

Hb
Hb
Hydrocephalus

All transmesencephalic feeders
All intranidal AA

E° glue partial treatment (2x 75% occlusion, 1x 90% occlusion)
FU outcome: 2x good, 1x fair
Rare and small (endovascular) series of brainstem AVMs

A proposed grading system to evaluate the endovascular curability of deep-seated arteriovenous malformations
Th. Robert & al; J Neurol Sci 2017

5 groups. Group 5: midbrain (no specificity of anatomical area : centered in midbrain: N=15)
10 E°. 3 Cured
Radiationtherapy in others

A challenging entity of endovascular embolization with Onyx for brainstem arteriovenous malformations.
Experience from 13 cases
Jin H &al; Interv Neuroradiol 2017

9 posterior midbrain AVMs.

8 Hb. 1 Headaches
1 intranidal AA on feeder

2 cured by E°. 7 partial E° + additional GammaKnife

No complication in the 2 cured pts and in 3 partially treated pts
1 death (brainstem infarction)
1 Hb with poor outcome (mRs 3)
2 transient diplopia (mRs 0 and 1 at mean FU 45,3 months)
More publications about **radiosurgery** and brainstem AVMs ... but rare specificity about tectal AVMs...

Microsurgery and radiosurgery in brain arteriovenous malformations

*Steiner L & al; J Neurosurg 1993*

57 brainstem AVMs (tectum?)
Complete obliteration in 71.4% of pts after 2 years FU
But no information about clinical characteristics, treatment parameters and adverse events

Prediction of results following Gamma Knife surgery for brain stem and other centrally located arteriovenous malformations: relation to natural course.

*Karlsson B & al; Stereotact Funct Neurosurg 1996*

Centrally located lesions carry higher risks of complications than those located peripherally
Positive relationship between the minimum dose given to the AVM nidus and the incidence of obliteration
The average dose to volumes that are large for radiosurgery is related to the incidence of complications
Results of radiosurgery for brainstem arteriovenous malformations

"Radiosurgery is considered as a valid treatment (...) it is also known to cause serious neurological deterioration when used for brainstem lesions"

“(…)the treatment of choice for brainstem AVMs located within the parenchyma”

“post treatment hemorrhage remains a significant problem with this technique…”

10 tectal AVMs

No description of the outcome of this specific type of brainstem AVMs, but:

“two patients with midbrain AVMs experienced symptomatic radiation injury 7 and 24 months after irradiation: ptosis and ataxia in one, eye movement disorder in the other one”

⇒ steroids. Resolution of symptoms within 1 year. No permanent deficit related to radiosurgery

GammaKnife radiosurgery for brainstem arteriovenous malformations: preliminary results

2 tectopineal AVMs

Overall results with no focus on the specific lesions
Figure 3  Neuroimages in a 16 year old girl with intraventricular haemorrhage. Left vertebral angiograms (A, B) and T2 weighted MR image (C) before radiosurgery show ruptured AVM in the tectum. T2 weighted MR image (D) obtained 6 months after radiosurgery shows asymptomatic brain stem oedema. Left vertebral angiograms (E, F) and T2 weighted MR image (G) obtained a year after radiosurgery show total obliteration of the AVM and a reversal of the oedema.
Stereotactic radiosurgery for brainstem arteriovenous malformations: factors affecting outcome

*Maruyama K & al, 2004*

An older patient age, a lesion located in the tectum, and a higher radiosurgery-based score were significantly associated with greater neurological complications.

Stereotactic Linac based radiosurgery in the treatment of arteriovenous malformations located deep involving corpus callosum, motor cortex or brainstem

*Zabel-Dubois A & al*  *Int. J. Radiation Oncology Biol Phys 2006*

5 brainstem AVMs (localization?)
« Size of AVM and applied single dose were determined as significant factors affecting successful obliteration »
Stereotactic Linac based radiosurgery in the treatment of arteriovenous malformations located deep involving corpus callosum, motor cortex or brainstem

Zabel-Dubois A & al  Int. J. Radiation Oncology Biol Phys 2006

5 brainstem AVMs (localization?)
« Size of AVM and applied single dose were determined as significant factors affecting successful obliteration »

GammaKnife Radiosurgery for arteriovenous malformations of basal ganglia, thalamus and brainstem- a retrospective Study comparing the results with that for AVMs at other intracranial locations

Kiran NA & al; Acta Neurochir 2009

« Central AVMs »......

No distinction of type of lesions and localization

Only size and dose...

Javalkar V & al, Neurol India 2009  Effective modality of treatment for central AVMs

Yen CP and Steiner L  World Neurosurg 2011  42 midbrain AVMs
« When the risks of surgery or embolization are high or there are still patent AVMs, GKS should be considered as a treatment alternative if the nidus is located in the subpial or epipial area, and it should always be used when the nidus is deep in the parenchyma of the brainstem.
In the present series, GKS achieved a complete obliteration rate of 59%. To this result should be added the higher risk of radiation-induced complications. »
Stereotactic radiosurgery for arteriovenous malformations located in deep critical regions

Nagy G & al, Neurosurgery 2012

After single treatment, obliteration was achieved in 65% of the brainstem, in 69% of the supratentorial, and 40% of the peritectal AVMs. Obliteration of lesions <4 cm was better in the brainstem (70%) and in the supratentorium (80%), but not in the peritectal region (40%).

Management outcome of brainstem arteriovenous malformations: the role of radiosurgery


“The distribution of brainstem locations in our cohort was 63.3% midbrain...”

Etc.... Etc...

Brainstem....Sometimes mesencephalon...rarely tectal / quadrigeminal plate...
**In Foch, Nancy and network**

<table>
<thead>
<tr>
<th>26 pts with quadrigeminal area AVMs</th>
<th>20 Nancy</th>
<th>9 epipial</th>
<th>11 subpial</th>
</tr>
</thead>
<tbody>
<tr>
<td>6 Foch</td>
<td>4 epipial</td>
<td>2 subpial</td>
<td></td>
</tr>
</tbody>
</table>

**Revealing symptoms**

- 22 Hb (84,6%)
- 1 fortuitous discovery (associated temporal AVM)
- 3 neuro (gaze palsy/ paresis -> diplopia...)

**Feeders**

- Mostly Collicular a, Superior cerebellar a

**Venous drainage**

- Precentral cerebellar vein / Latero mesencephalic vein (epipial)
- (More complex venous drainages for subpial lesions)

**Associated AA or false AA**

- 1 / 26 (subpial). 0 in epipial

**Size of lesion**

- From punctiform (epipial) to small nidus
<table>
<thead>
<tr>
<th>Therapeutic strategies and outcomes</th>
<th>17 pts treated Nancy</th>
<th>6 pts treated Foch</th>
<th>Trtt in 23 pts/26</th>
</tr>
</thead>
<tbody>
<tr>
<td>E° 20 glue / 1 Onyx</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>E° alone</th>
<th>Rxtherapy alone</th>
<th>E°+Rxth</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cure (Total occlusion)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6*</td>
<td>1</td>
<td>5**</td>
<td>12</td>
</tr>
<tr>
<td>Incomplete occl</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6***</td>
<td>1</td>
<td>2</td>
<td>9</td>
</tr>
</tbody>
</table>

ReHb 5y . Further Trtt in discussion

Further trtt in discussion

Waiting for results |
| 2          |

Total |
| 12       |
| 2        |
| 9        |
| 23       |

* 1 recurrence 10 y later (=> diplopia. 2nd E° + Rxth)

** 1 recurrence 7 y later (=> Holmes tremor. 2nd Rxtn)

*** 2 early rebleeds at D4 and D10 (1 death; 1 waiting for complementary trtt)

1 pt Onyx. Occlusion Sup cereb a. Development angiogenesis + dural adh (waiting for Rxth)

3 lost FU
31 y F
Hb 2011
Headaches
Tinnitus
Gaze palsy
R hemihypoesthesia
L hemiparesis

Partial recovery
Diplopia, hypoesthesia
R upper limb

Radiosurgery 2012
->No effect
F 49 y
IVH
Tr in writing
Hemifacial pain
M 41 y
Left hemiplegia
Dysarthria
Oculomotor palsy
Decision for radiosurgery
(perform 2013)
F born 1967
Stagnation...

ADC map
Vasogenic edema
Rebleed 10 y later on the same site from a recurrent AVM....
F 21 y headaches, intermittent strabismus, sensations of loss of equilibrium
Symptoms: venous congestion of quadrigeminal plaque and mesencephalic structures involved in oculomotricity

Venous congestion of the posterior fossa

→ tonsilar prolapse

Girard N et al
Reversible tonsilar prolapse in VGAMs: report of 8 cases and pathophysiological hypothesis. Childs Nerv Syst 10: 141-147, 1994

→ hydromyelia
M born in 1956
Operated for strabism in 1972

Followed for >20 y. No hemorrhage
Therapeutic Abstention (subpial!)
Diplopia
Conclusions

Tectal/posterior midbrain/quadrigeminal AVMs small size

Epipial vs Subpial

Hemorrhagic presentation

Simple architecture rare associated AA
single (main) draining vein

Challenging lesions: Surgical treatment Cure if epipial... but risks morbi-mortality (specially if intraparenchyma)
Embolization Few cures
Danger of Onyx (contraindication?: reflux; penetration; toxicity DMSO)
Glue ok but needs occlusion foot of vein
(challenging and risky because of vascular network)
Angiogenesis
Transvenous approach??
Collicular v and precentral v

Diplopia

Residual IVth n palsy

Post E° (glue) and post radiosurg (no result)

Failure transvenous approach. Surgery?
Conclusions

Tectal/posterior midbrain/quadrigeminal AVMs small size

Epipial vs Subpial

Hemorrhagic presentation

Simple architecture rare/exceptional associated AA (specific biology of tectal plate?) one (two...) draining veins

Challenging lesions:

- **Surgical treatment**
  - Cure if epipial... but risks morbi-mortality (specially if intraparenchyma)
- **Embolization**
  - Few cures
  - Danger of Onyx (contraindication?: reflux; penetration; toxicity DMSO)
  - Glue ok but needs occlusion foot of vein (challenging and risky because of vascular network)
- **Angiogenesis**
- **Transvenous approach??**
- **Radiosurgery**
  - Risk of rebleeding in the latency period. Higher risks for tectal AVMs

Combination and association of treatments. Selection of patients. Need for long FUs