Aneurysmal Subarachnoid Hemorrhage

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March 22, 2014
11:30am-12:45pm
CANP 37th Annual Conference

Disclosures

- none

Objectives

- Describe etiology and pathophysiology of aneurysmal subarachnoid hemorrhage (SAH).
- Discuss the grading scales used in the assessment of patients with aSAH.
- Understand the treatment modalities used for the prevention of morbidity and mortality that can follow SAH.

Overview

- Definition/epidemiology
- Disease process
- Current guidelines for management
- Case study

Epidemiology

- Adults over 30: 9 per 100,000
  translates to 30k per year in the US.
- Female > Male (1.6x)
- ~12% will die before reaching the hospital
- 45% overall mortality

- Warren R. Selman, Jeffry L. Sunshine, Robert W. Tarr, and Robert A. Ratcheson, Chapter 57 VascularDiseases of the Nervous System, C. Intracranial Aneurysms and Subarachnoid Hemorrhage. Neurology in Clinical Practice. Volume II, 4th Edition. Butterworth Heineman 2006.
- Huang J, van Gelder JM, The probability of sudden death from rupture of intracranial aneurysms: a meta- analysis. Neurology. 2002 Nov;51(5): 1101-5.

Basic Definitions

- Aneurysm

- What is it?
- Risk factors:
  Heredity
  HTN
  Tobacco

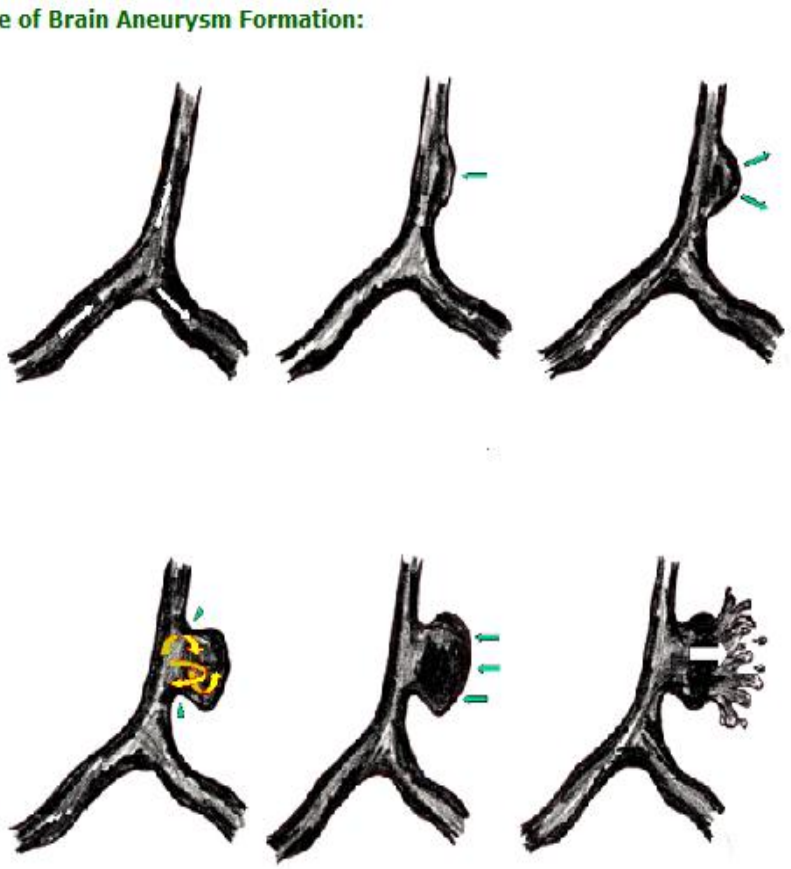
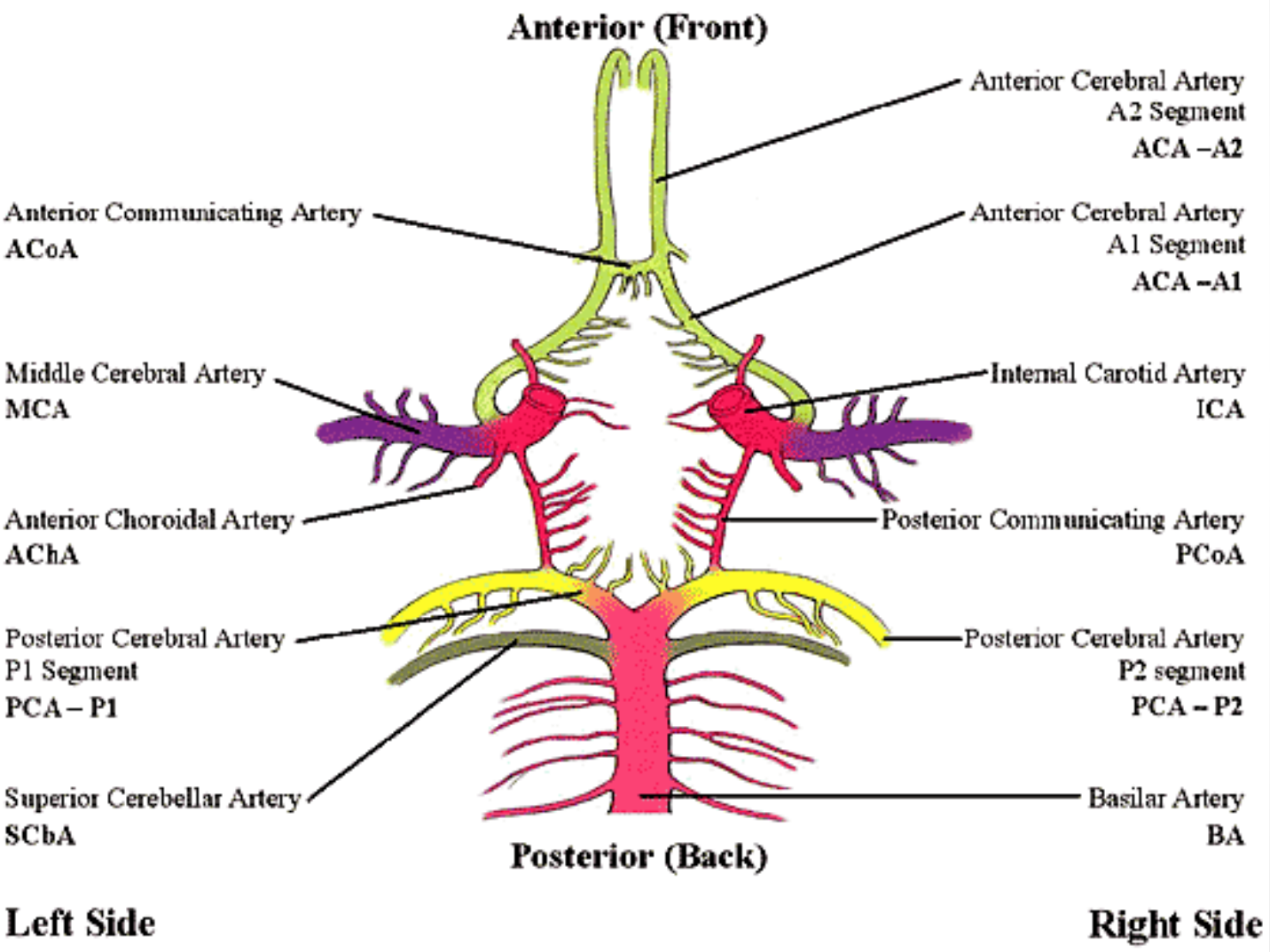


Figure:

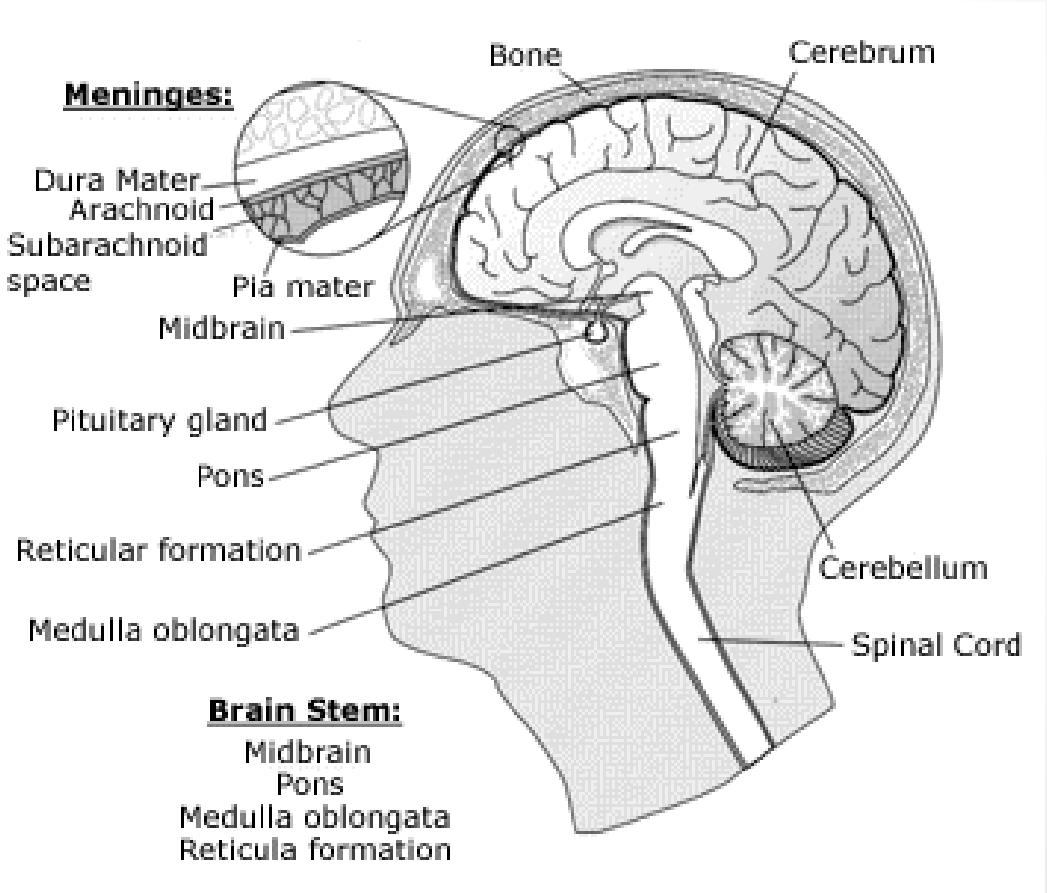
www.brain-aneurysm.com

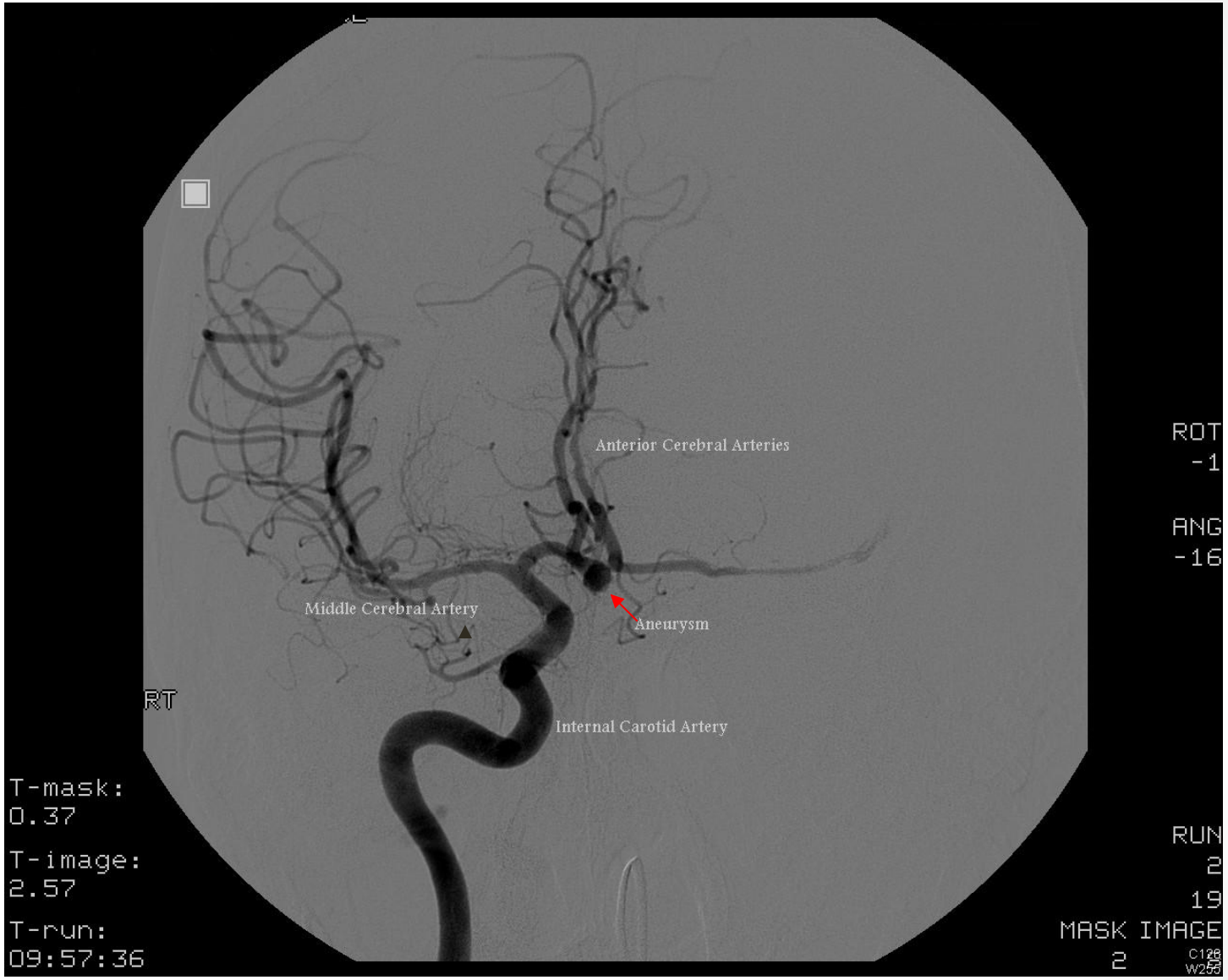
The Circle of Willis



Basic Definitions

- Subarachnoid Hemorrhage




Anterior Cerebral Arteries
Middle Cerebral Artery
Aneurysm
Internal Carotid Artery
RT
ROT
-1
ANG
-16
T-mask:
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RUN
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MASK IMAGE
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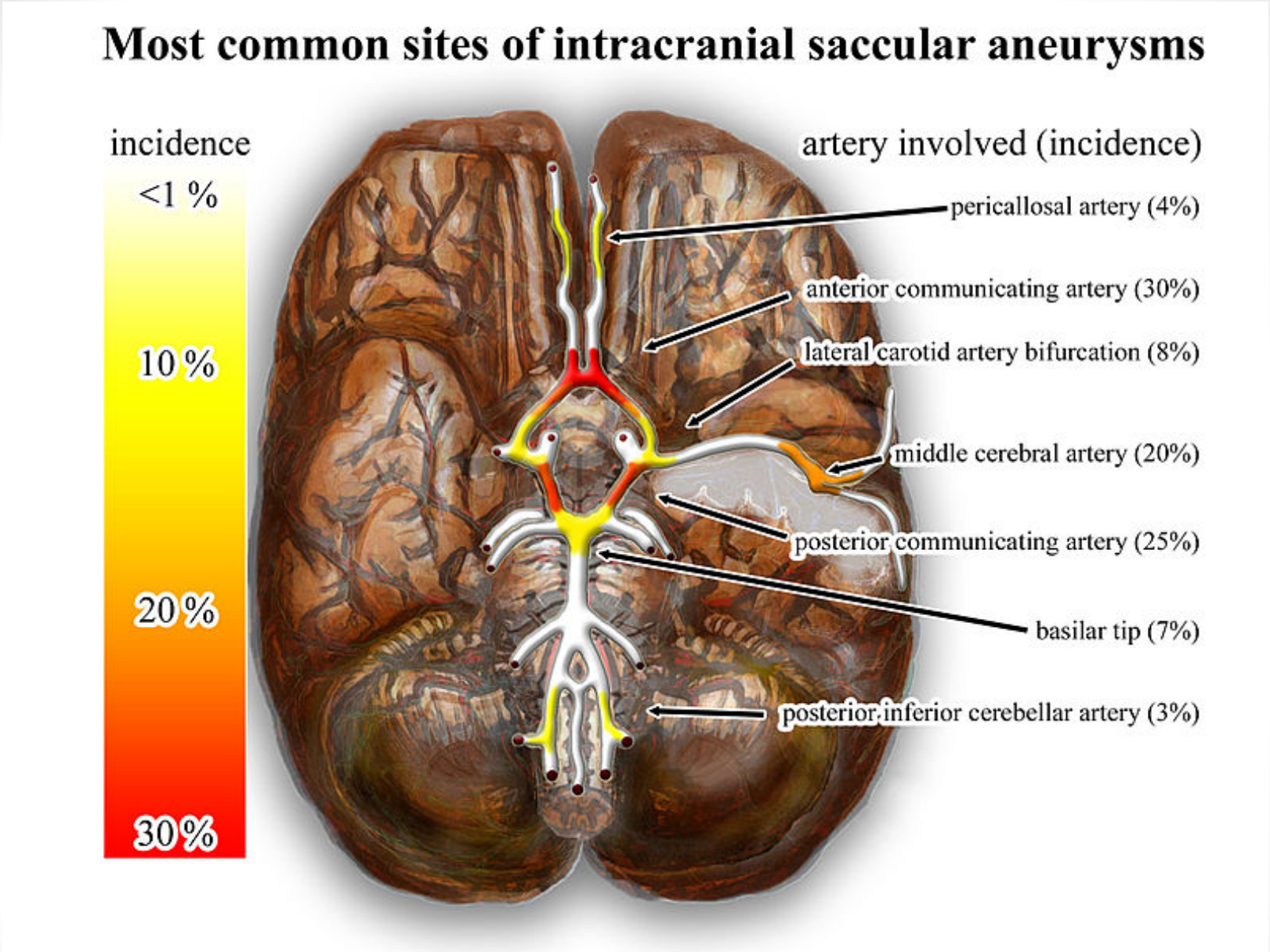


http://en.wikipedia.org/wiki/File:Wikipedia_intracranial_aneurysms_-_inferior_view_-_heat_map.jpg

Author: Nicholas Zaorsky, M.D.

What are the current guidelines?

- Guidelines for the management of aneurysmal subarachnoid hemorrhage: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke.* 2012;43:1711–1737.

Table 1. Applying Classification of Recommendation and Level of Evidence

ESTIMATE OF CERTAINTY (PRECISION) OF TREATMENT EFFECT	SIZE OF TREATMENT EFFECT: CLASS I *Benefit >>> Risk* Procedure/Treatment **SHOULD** be performed/ administered	CLASS IIa *Benefit >> Risk* *Additional studies with focused objectives needed* **IT IS REASONABLE** to perform procedure/administer treatment	CLASS IIb *Benefit ≥ Risk* *Additional studies with broad objectives needed; additional registry data would be helpful* Procedure/Treatment **MAY BE CONSIDERED**	CLASS III *No Benefit* or CLASS III *Harm* Procedure/Test / Treatment COR III: No benefit — Not Helpful / No Proven Benefit COR III: Harm — Excess Cost w/o Benefit or Harmful / Harmful to Patients
LEVEL A Multiple populations evaluated* Data derived from multiple randomized clinical trials or meta-analyses	■ Recommendation that procedure or treatment is useful/effective ■ Sufficient evidence from multiple randomized trials or meta-analyses	■ Recommendation in favor of treatment or procedure being useful/effective ■ Some conflicting evidence from multiple randomized trials or meta-analyses	■ Recommendation's usefulness/efficacy less well established ■ Greater conflicting evidence from multiple randomized trials or meta-analyses	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Sufficient evidence from multiple randomized trials or meta-analyses
LEVEL B Limited populations evaluated* Data derived from a single randomized trial or nonrandomized studies	■ Recommendation that procedure or treatment is useful/effective ■ Evidence from single randomized trial or nonrandomized studies	■ Recommendation in favor of treatment or procedure being useful/effective ■ Some conflicting evidence from single randomized trial or nonrandomized studies	■ Recommendation's usefulness/efficacy less well established ■ Greater conflicting evidence from single randomized trial or nonrandomized studies	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Evidence from single randomized trial or nonrandomized studies
LEVEL C Very limited populations evaluated* Only consensus opinion of experts, case studies, or standard of care	■ Recommendation that procedure or treatment is useful/effective ■ Only expert opinion, case studies, or standard of care	■ Recommendation in favor of treatment or procedure being useful/effective ■ Only diverging expert opinion, case studies, or standard of care	■ Recommendation's usefulness/efficacy less well established ■ Only diverging expert opinion, case studies, or standard of care	■ Recommendation that procedure or treatment is not useful/effective and may be harmful ■ Only expert opinion, case studies, or standard of care
Suggested phrases for writing recommendations	should is recommended is indicated is useful/effective/beneficial	is reasonable can be useful/effective/beneficial is probably recommended or indicated	may/might be considered may/might be reasonable usefulness/effectiveness is unknown/unclear/uncertain or not well established	COR III: No Benefit: is not recommended; is not indicated; should not be performed/ administered/ other; is not useful/ beneficial/ effective COR III: Harm: potentially harmful; causes harm; associated with excess morbidity/mortality; should not be performed/ administered/ other
Comparative effectiveness phrases†	treatment/strategy A is recommended/indicated in preference to treatment B treatment A should be chosen over treatment B	treatment/strategy A is probably recommended/indicated in preference to treatment B It is reasonable to choose treatment A over treatment B		

A recommendation with Level of Evidence B or C does not imply that the recommendation is weak. Many important clinical questions addressed in the guidelines do not lend themselves to clinical trials. Although randomized trials are unavailable, there may be a very clear clinical consensus that a particular test or therapy is useful or effective.

*Data available from clinical trials or registries about the usefulness/efficacy in different subpopulations, such as sex, age, history of diabetes, history of prior myocardial infarction, history of heart failure, and prior aspirin use.

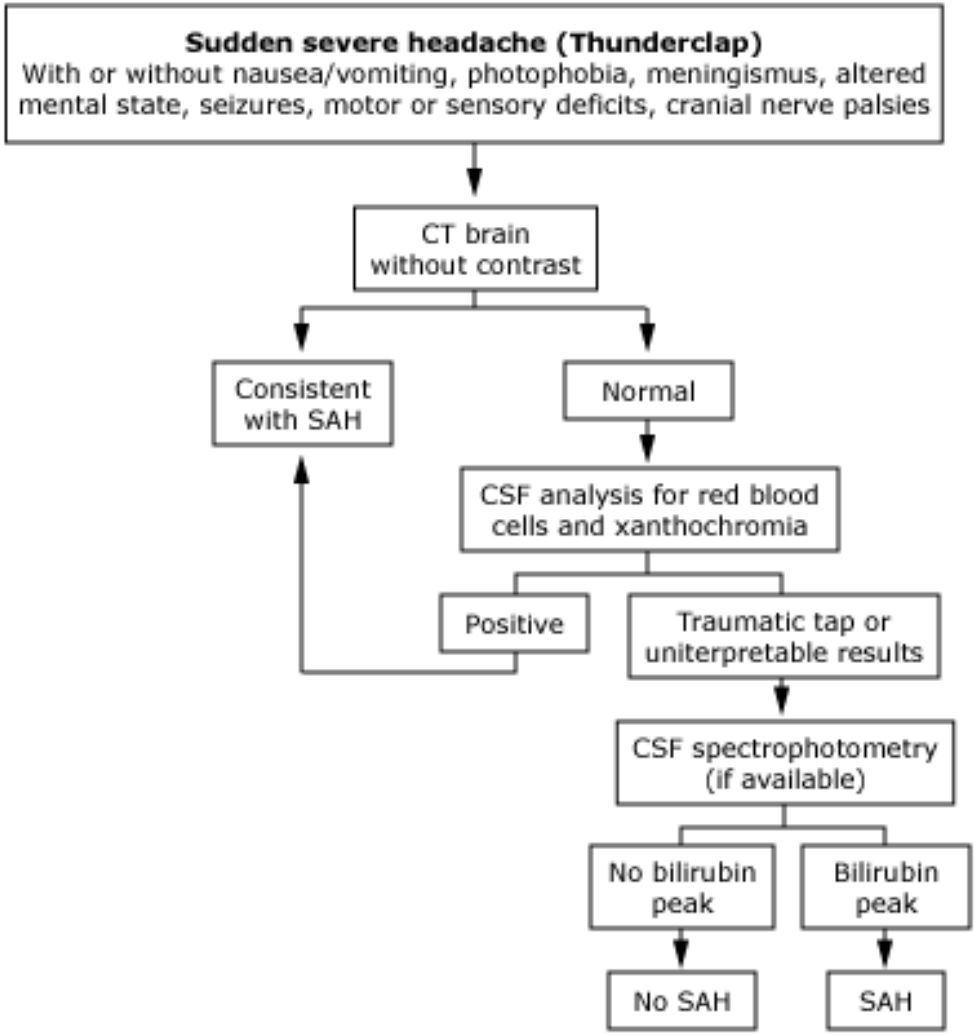
†For comparative effectiveness recommendations (Class I and IIa; Level of Evidence A and B only), studies that support the use of comparator verbs should involve direct comparisons of the treatments or strategies being evaluated.

Diagnosis and assessment

- **Clinical Manifestations and Diagnosis of aSAH:**
- **1. aSAH is a medical emergency that is frequently misdiagnosed. A high level of suspicion for aSAH should exist in patients with acute onset of severe headache *(Class I; Level of Evidence B).***
- **2. Acute diagnostic workup should include noncontrast head CT, which, if nondiagnostic, should be followed by lumbar puncture *(Class I; Level of Evidence B).***
- **3. CTA may be considered in the workup of aSAH. If an aneurysm is detected by CTA, this study may help guide the decision for type of aneurysm repair, but if CTA is inconclusive, DSA is still recommended (except possibly in the instance of classic perimesencephalic aSAH) *(Class IIb; Level of Evidence C).*** (New recommendation)
- **4. Magnetic resonance imaging (fluid-attenuated inversion recovery, proton density, diffusion-weighted imaging, and gradient echo sequences) may be reasonable for the diagnosis of aSAH in patients with a nondiagnostic CT scan, although a negative resultdoes not obviate the need for cerebrospinal fluid analysis *(Class IIb; Level of Evidence C).*** (New recommendation)
- **5. DSA with 3-dimensional rotational angiography is indicated for detection of aneurysm in patients with aSAH (except when the aneurysm was previously diagnosed by a noninvasive angiogram) and for planning treatment (to determine whether an aneurysm is amenable to coiling or to expedite microsurgery) *(Class I; Level of Evidence B).*** (New recommendation)

Presenting Symptoms…

Evaluation of suspected subarachnoid hemorrhage



SAH: subarachnoid hemorrhage.

UpToDate®

Presenting Symptoms:

N. F. Kassell, *et al.*

TABLE 6

Neurological status on admission

Neurological Feature	Day 0		Day 1		Day 2		Day 3		Total	
	No.	Percent	No.	Percent	No.	Percent	No.	Percent	No.	Percent
level of consciousness										
fully alert	708	41.1	557	50.5	268	64.1	190	68.3	1723	48.9
drowsy	557	32.4	393	35.6	121	28.9	65	23.4	1136	32.3
stuporous	221	12.8	87	7.9	22	5.3	18	9.9	348	9.9
comatose	235	13.7	67	6.1	7	1.8	5	8.9		
speech										
normal	1150	66.8	873	79.1	372	89.0	249	89.6	2644	75.1
dysphasic	167	9.7	97	8.8	17	4.1	13	4.7	294	8.3
no verbal response	404	23.5	134	12.1	29	6.9	16	16.6	583	16.6
orientation										
normal	902	52.4	670	60.7	305	73.0	204	73.4	2081	59.1
impaired	819	47.6	434	39.3	113	27.0	74	26.6	1440	40.9
response to commands										
appropriate	1141	66.3	866	78.4	374	89.5	240	86.3	2621	74.4
inappropriate	580	33.7	238	21.6	44	10.5	38	13.7	900	25.6
motor response										
normal	1189	69.1	882	79.9	361	86.4	238	85.6	2670	75.8
mild focal deficit	224	13.0	129	11.7	43	10.3	25	9.0	421	12.0
severe focal deficit	123	7.1	56	5.1	9	2.2	10	3.6	198	5.6
abnormal flexor	48	2.8	16	1.4	1	0.2	3	1.1	68	1.9
abnormal extensor	92	5.3	16	1.4	1	0.2	1	0.4	110	3.1
no response	45	2.6	5	0.5	3	0.7	1	0.4	54	1.5
meningeal signs										
none	225	13.1	75	6.8	31	7.4	24	8.6	355	10.1
headache	1142	66.4	791	71.6	313	74.9	215	77.3	2461	69.9
stiff neck	1281	74.4	939	85.1	345	82.5	209	75.2	2774	78.8
cranial nerve deficit										
none	1502	87.3	1003	90.9	371	88.8	239	86.0	3115	88.5
third	161	9.4	61	5.5	30	7.2	25	9.0	277	7.9
other	71	4.1	46	4.2	19	4.5	15	5.4	151	4.3
totals	1721	48.9	1104	31.4	418	11.9	278	7.9	3521	100.0

Why early CT?

TABLE 7

*Finding on first computerized tomography (CT) scan**

CT Findings	Day 0		Day 1		Day 2		Day 3		Day 4		Day 5		Total	
	No.	Percent	No.	Percent	No.	Percent	No.	Percent	No.	Percent	No.	Percent	No.	Percent
normal	51	3.3	76	7.2	68	15.2	62	22.2	20	26.3	9	27.3	288	8.3
decreased density	11	0.7	14	1.3	4	0.9	5	1.8	2	2.6	1	3.0	39	1.1
mass effect	124	8.0	57	5.4	14	3.1	11	3.9	2	2.6	2	6.1	211	6.1
aneurysm	73	4.7	50	4.8	33	7.4	11	3.9	5	6.6	0	0.0	173	5.0
hydrocephalus	248	16.0	170	16.2	51	11.4	47	16.8	6	7.9	3	9.1	527	15.2
intraventricular hematoma	312	20.1	178	17.0	51	11.4	23	8.2	10	13.2	2	6.1	578	16.7
intracerebral hematoma	294	18.9	171	16.3	70	15.7	43	15.4	14	18.4	6	18.2	601	17.4
subdural hematoma	28	1.8	7	0.7	6	1.3	2	0.7	1	1.3	0	0.0	45	1.3
subarachnoid hemorrhage†	1430	92.1	906	86.4	338	75.8	189	67.7	49	64.5	19	57.6	2940	85.2
diffuse	876	56.4	506	48.2	167	37.4	87	31.2	29	38.2	14	42.4	1684	48.9
thin	241	15.5	211	20.1	93	20.9	54	19.4	12	15.8	5	15.2	619	17.9
thick	587	32.6	321	30.6	116	26.0	68	24.4	15	19.7	6	18.2	1034	30.0
totals	1553	45.2	1049	30.5	446	13.0	279	8.1	76	2.2	33	1.0	3451	100.0

* Fifteen patients underwent CT scanning after Day 5; 70 patients did not have a CT scan.

† Classification was determined according to Fisher, *et al.*[11]

- How to describe our patients and risk stratify for complication

Basic Definitions

- Glascow Coma Scale

- Verbal 1-5
  1 None
  2 Moans
  3 Unintelligable words
  4 Confused Conversation
  5 Oriented

- Motor (1-6)
  1 None
  2 Decerebrate
  3 Decorticate
  4 Flexion Withdrawal
  5 Localize
  6 Follows Commands
- Eye Opening (1-4)
  1 None
  2 To Pain
  3 To Voice
  4 Spontaneous

Hunt and Hess Scale

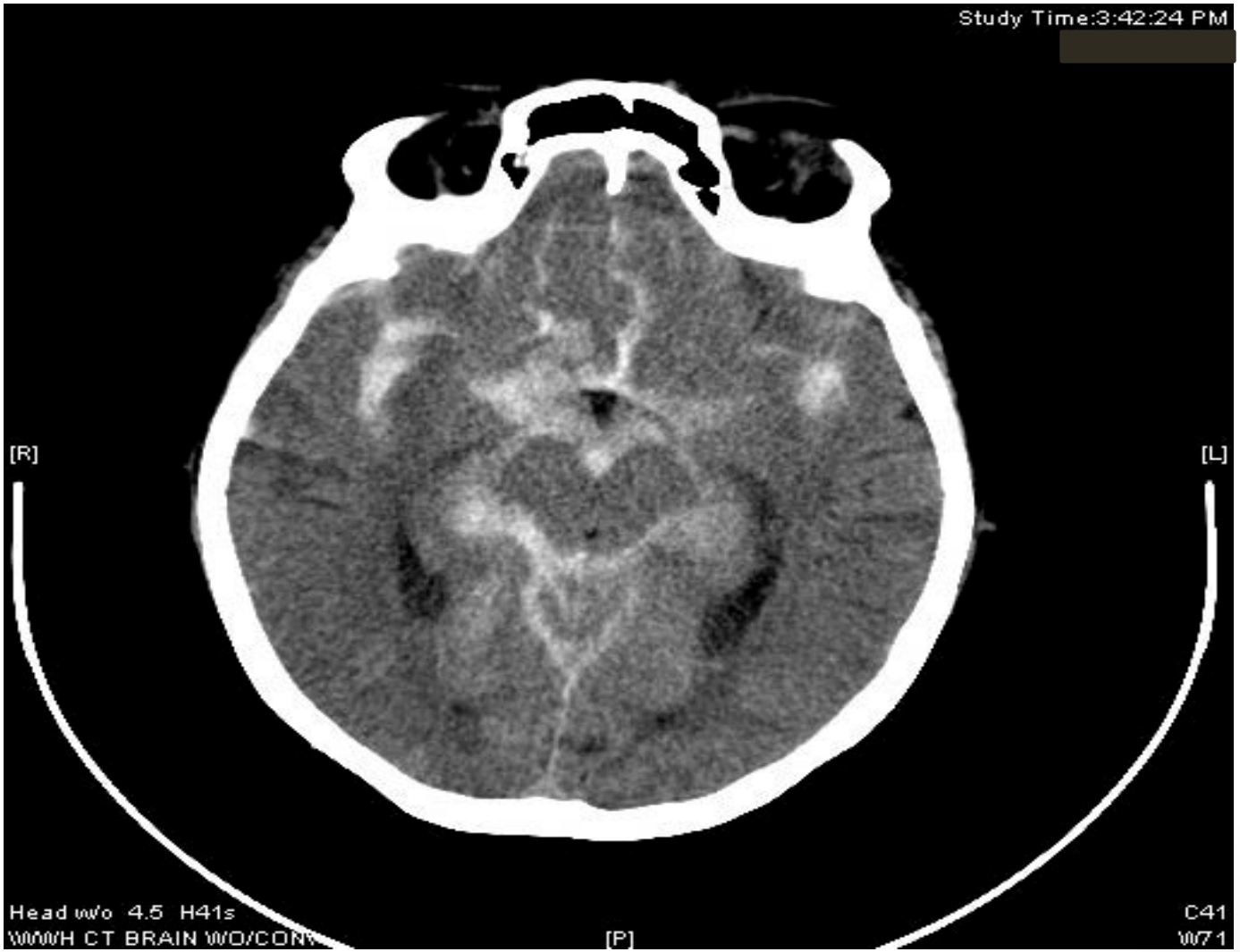
- Grade 0: Asymptomatic
- Grade I: Slight Headache, No Decicit
- Grade II: Severe Headache, No Deficit (may have cranial nerve palsy)
- Grade III: Drowsiness and mild deficit
- Grade IV: Stupor, Moderate to severe hemiparesis, and possible early rigidity
- Grade V: Deep coma, decerebrate rigidity, moribund appearance

Fischer Grade

- Based on imaging

- Grade 1: No Blood noted on Computed Tomography (CT) scan
- Grade 2: <1mm blood in subarachnoid space
- Grade 3: >1mm blood in subarachnoid space
- Grade 4: Blood in subarachnoid space with extension into ventricular or parenchymal space

Lets Practice Grading a SAH



Another scale to be aware of:

- World Federation of Neurologic Surgeons (WFNS)

- 1: GCS 15 no focal deficit
- 2: GCS 13-14, no focal deficit
- 3: GCS 13-14 with focal deficit
- 4: GCS 7-12 with or without focal deficit
- 5: GCS <7 with or without focal deficit

Management Guidelines

- **Medical Measures to Prevent Rebleeding After aSAH: Recommendations**

- **1. Between the time of aSAH symptom onset and aneurysm obliteration, blood pressure should be controlled with a titratable agent to balance the risk of stroke, hypertension-related rebleeding, and maintenance of cerebral perfusion pressure *(Class I; Level of Evidence B).*** (New recommendation)
- **2. The magnitude of blood pressure control to reduce the risk of rebleeding has not been established, but a decrease in systolic blood pressure to <160 mm Hg is reasonable *(Class IIa; Level of Evidence C).*** (New recommendation)
- **3. For patients with an unavoidable delay in obliteration of aneurysm, a significant risk of rebleeding, and no compelling medical contraindications, shortterm (<72 hours) therapy with tranexamic acid or aminocaproic acid is reasonable to reduce the risk of early aneurysm rebleeding *(Class IIa; Level of Evidence B).*** (Revised recommendation from previous guidelines)

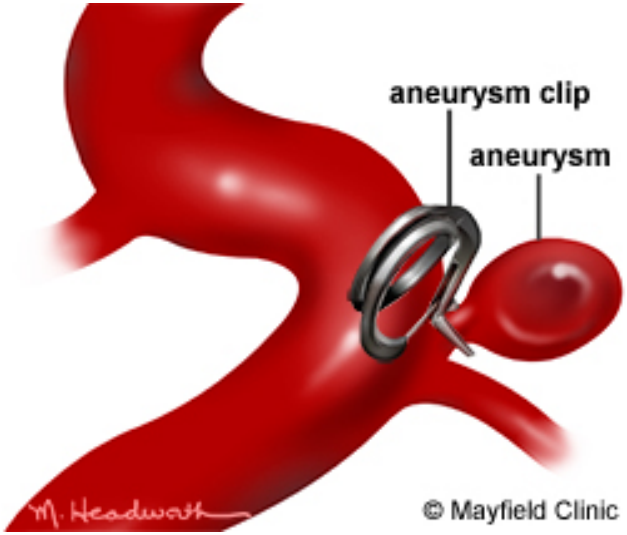
Surgical and Endovascular Methods of Treatment of Ruptured Cerebral Aneurysms:

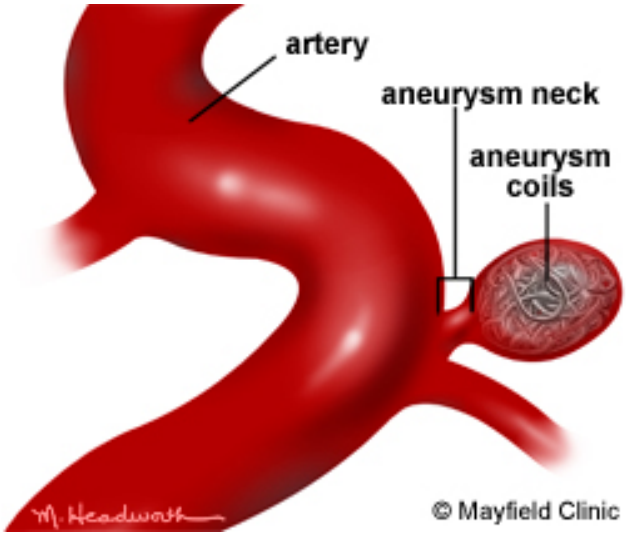
- **1. Surgical clipping or endovascular coiling of the ruptured aneurysm should be performed as early as feasible in the majority of patients to reduce the rate of rebleeding after aSAH *(Class I; Level of Evidence B).***
- **2. Complete obliteration of the aneurysm is recommended whenever possible *(Class I; Level of Evidence B).***
- **3. Determination of aneurysm treatment, as judged by both experienced cerebrovascular surgeons and endovascular specialists, should be a multidisciplinary decision based on characteristics of the patient and the aneurysm *(Class I; Level of Evidence C).*** (Revised recommendation from previous guidelines)
- **4. For patients with ruptured aneurysms judged to be technically amenable to both endovascular coiling and neurosurgical clipping, endovascular coiling should be considered *(Class I; Level of Evidence B).*** (Revised recommendation from previous guidelines)

Methods of Treatment cont.

- **5. In the absence of a compelling contraindication, patients who undergo coiling or clipping of a ruptured aneurysm should have delayed follow-up vascular imaging (timing and modality to be individualized), and strong consideration should be given to retreatment, either by repeat coiling or microsurgical clipping, if there is a clinically significant (eg, growing) remnant *(Class I; Level of Evidence B).*** (New recommendation)
- **6. Microsurgical clipping may receive increased consideration in patients presenting with large (>50 mL) intraparenchymal hematomas and middle cerebral artery aneurysms. Endovascular coiling may receive increased consideration in the elderly (70 years of age), in those presenting with poor-grade(World Federation of Neurological Surgeons classification IV/V) aSAH, and in those with aneurysms of the basilar apex *(Class IIb; Level of Evidence C).*** (New recommendation)
- **7. Stenting of a ruptured aneurysm is associated with increased morbidity and mortality, and should only be considered when less risky options have been excluded *(Class III; Level of Evidence C).*** (New recommendation)

Clip versus Coil

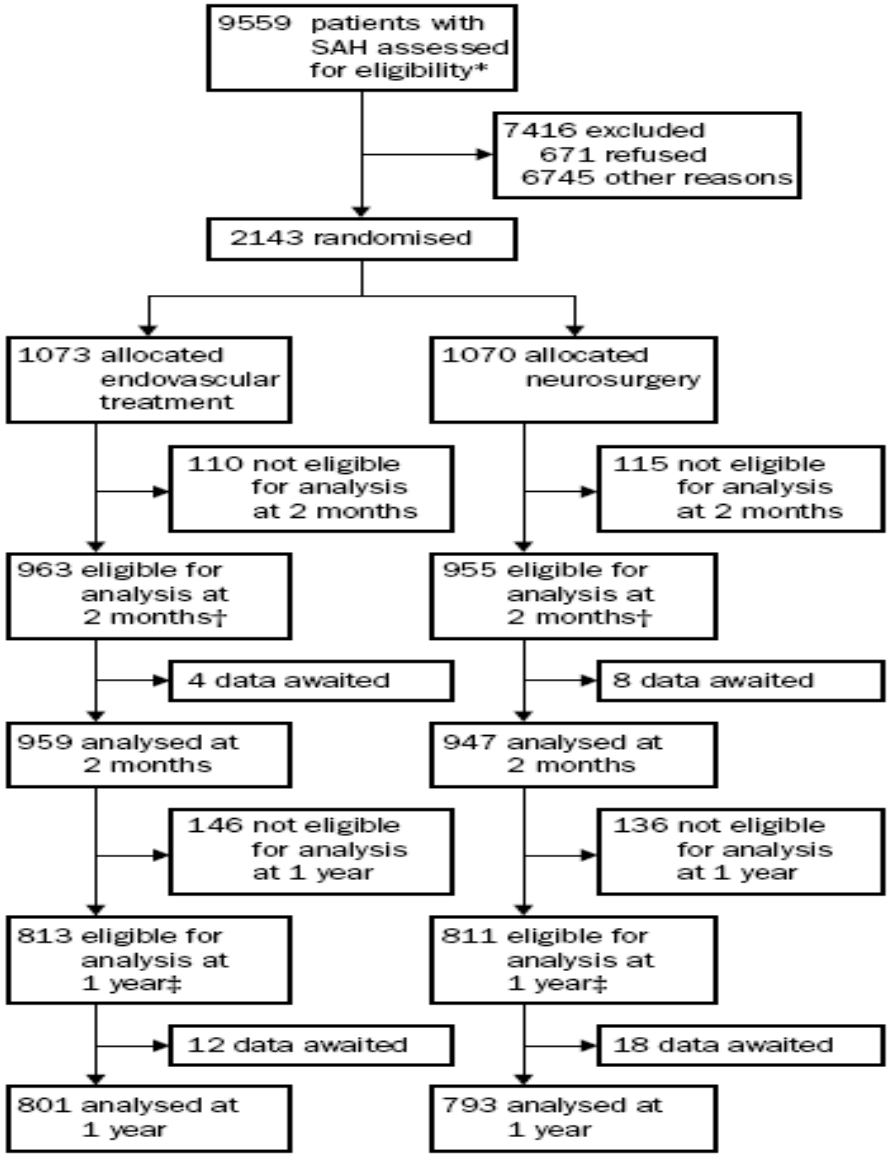




Treatment and Care

- Clip vs Coil
  Based on size and morphology of Aneurism
  Dome to neck ratio
  Medical stability of patient
- 2005 International Subarachnoid Aneurysm Trial (ISAT)
  Only multicenter, randomized trial
  2143 patients

ISAT cont.



Trial profile

SAH=subarachnoid haemorrhage. *Based on those centres that returned ascertainment logs. †Based on patients randomised before Dec 1, 2001. ‡Based on patients randomised before Feb 1, 2001.

Results of ISAT

- 23% (coil) vs 31% (clip) with death or severe disability at 1 year.
- Possible increased risk for re-bleed w endovascular therapy 5% vs 4%.

- Results favor coil embolization (but caveat).

Hospital Characteristics and Systems of Care: Recommendations

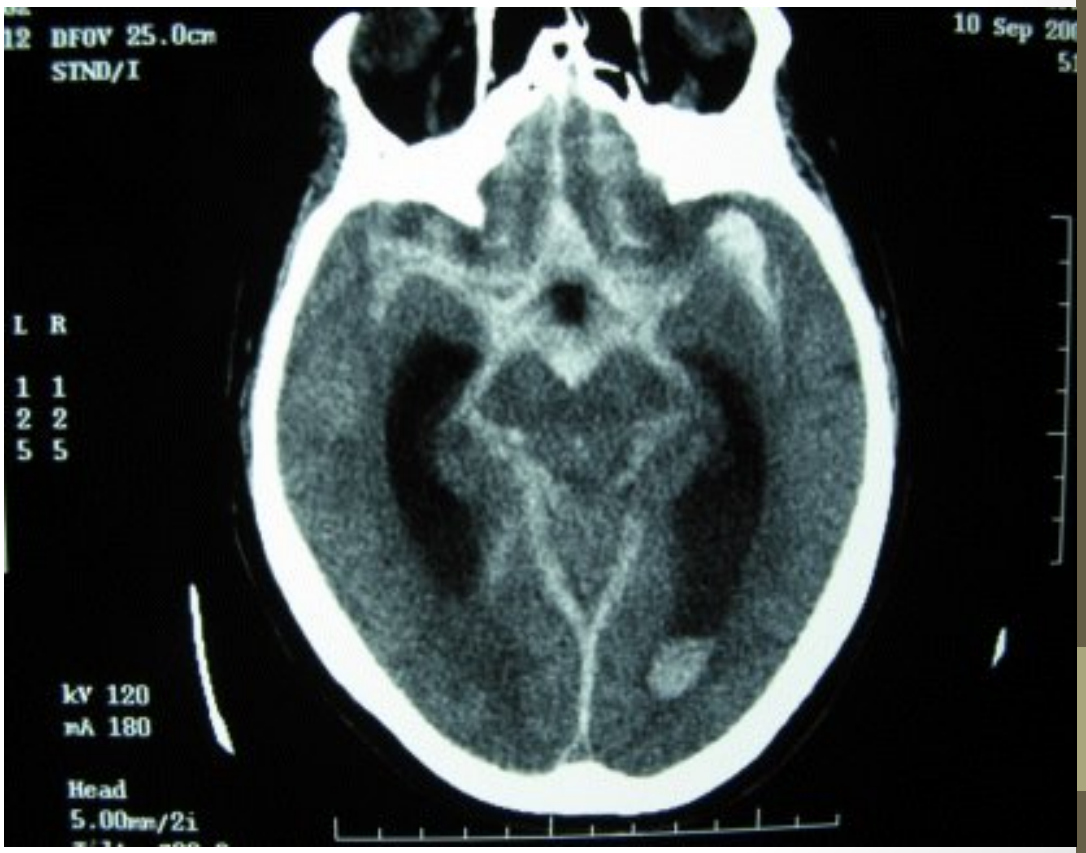
- **1. Low-volume hospitals (eg, <10 aSAH cases per year) should consider early transfer of patients with aSAH to high-volume centers (eg, >35 aSAH cases per year) with experienced cerebrovascular surgeons, endovascular specialists, and multidisciplinary neuro-intensive care services *(Class I; Level of Evidence B).*** (Revised recommendation from previous guidelines)
- **2. Annual monitoring of complication rates for surgical and interventional procedures is reasonable *(Class IIa; Level of Evidence C*).** (New recommendation)
- **3. A hospital credentialing process to ensure that proper training standards have been met by individual physicians treating brain aneurysms is reasonable *(Class IIa; Level of Evidence C).*** (New recommendation)

Complications

- Hydrocephalus
- Delayed Icshemic Deficits
- Medical Complications

Hydrocephalus

- Disturbance in CSF reabsorption

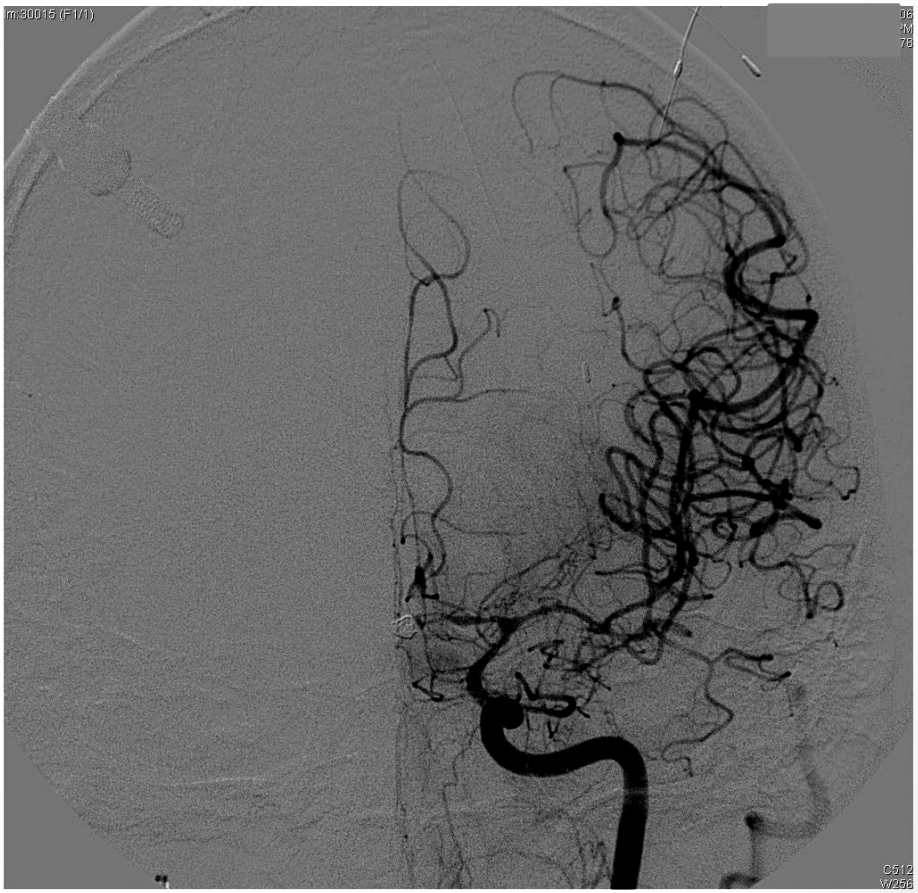


Management of Hydrocephalus Associated With aSAH: Recommendations

- **1. aSAH-associated acute symptomatic hydrocephalus should be managed by cerebrospinal fluid diversion (EVD or lumbar drainage, depending on the clinical scenario) *(Class I; Level of Evidence B).*** (Revised recommendation from previous guidelines)
- **2. aSAH-associated chronic symptomatic hydrocephalus should be treated with permanent cerebrospinal fluid diversion *(Class I; Level of Evidence C*).** (Revised recommendation from previous guidelines)
- **3. Weaning EVD over >24 hours does not appear to be effective in reducing the need for ventricular shunting *(Class III; Level of Evidence B).*** (New recommendation)
- **4. Routine fenestration of the lamina terminalis is not useful for reducing the rate of shunt-dependent hydrocephalus and therefore should not be routinely performed. *(Class III; Level of Evidence B).*** (New recommendation)

Delayed Ischemic Deficit

- Vasospasm
- Time of onset 5-7 days
- Duration may last up to 14 days
- Role of Nimodipine



Management of Cerebral Vasospasm and DCI After aSAH: Recommendations

- **1. Oral nimodipine should be administered to all patients with aSAH *(Class I; Level of Evidence A).*** (It should be noted that this agent has been shown to improve neurological outcomes but not cerebral vasospasm. The value of other calcium antagonists, whether administered orally or intravenously, remains uncertain.)
- **2. Maintenance of euvolemia and normal circulating blood volume is recommended to prevent DCI *(ClassI; Level of Evidence B).*** (Revised recommendation from previous guidelines)
- **3. Prophylactic hypervolemia or balloon angioplasty before the development of angiographic spasm is not recommended *(Class III; Level of Evidence B).*** (New recommendation)
- **4. Transcranial Doppler is reasonable to monitor for the development of arterial vasospasm *(Class IIa; Level of Evidence B).*** (New recommendation)

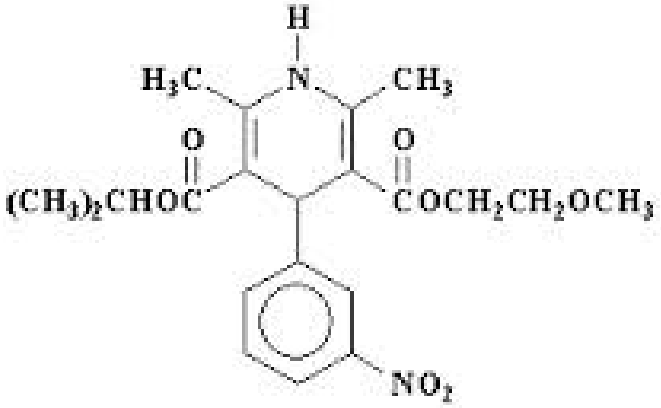
Cerebral Vasospasm and DCI cont:

- **5. Perfusion imaging with CT or magnetic resonance can be useful to identify regions of potential brain ischemia *(Class IIa; Level of Evidence B).*** (New recommendation)
- **6. Induction of hypertension is recommended for patients with DCI unless blood pressure is elevated at baseline or cardiac status precludes it *(Class I; Level of Evidence B).*** (Revised recommendation from previous guidelines)
- **7. Cerebral angioplasty and/or selective intra-arterial vasodilator therapy is reasonable in patients with symptomatic cerebral vasospasm, particularly those who are not rapidly responding to hypertensive therapy *(Class IIa; Level of Evidence B*).** (Revised recommendation from previous guidelines)

Nimodipine

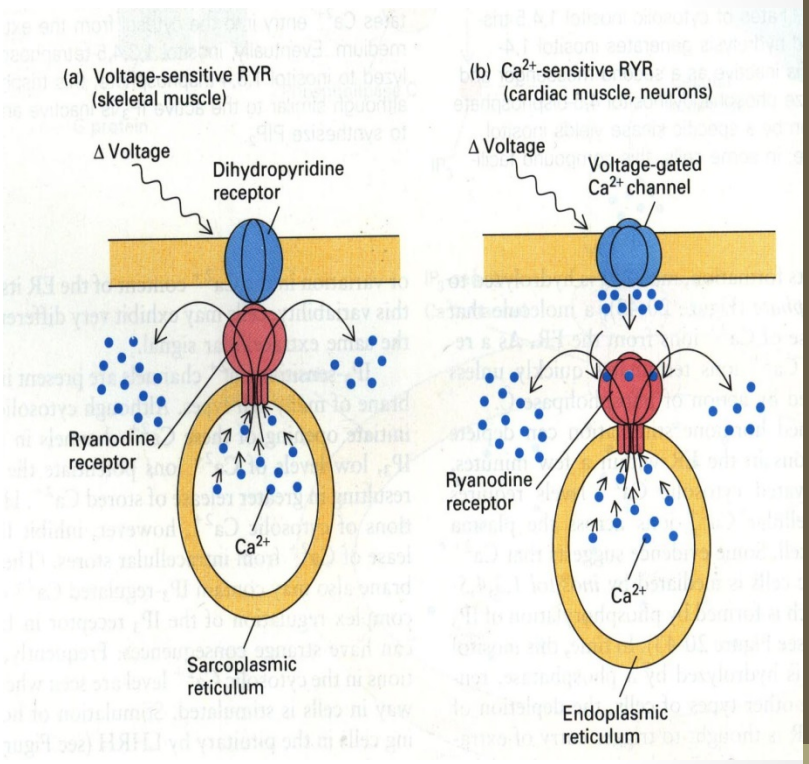
Nimodipine

$C_{21}H_{26}N_2O_7$



Nimodipine

- Dihydropyridine calcium channel blocker
- Protein bound
- Time to peak activity – 1 hour
- Medication half life – 8-9 hours
- Side Effects: hypotension, nausea, diarrhea, cramping



Prevention of Delayed Ischemic Deficit (DID)

- Data in early 80s demonstrated efficacy of drug in limiting DID despite continued angiographic vasospasm
- Strength of Effect began to be called into question

Mechanisms:

- Vasodilation
- Cytoprotective/Free radical scavenger
- Potentiation of collateral flow

Early data:

- NEJM 1983
- 125 patients randomized, double blind, placebo control
- 21 day outcomes.
- Death or severe disability occurred 8 of 60 in placebo vs 1 of 56 in Nimodipine arm. P=0.03

Efficacy of prophylactic nimodipine for delayed ischemic deficit after subarachnoid hemorrhage: a metaanalysis

FRED G. BARKER II, M.D., AND CHRISTOPHER S. OGILVY, M.D.

Brain Tumor Research Center, Department of Neurological Surgery, University of California at San Francisco, San Francisco, California; and Cerebrovascular Surgery, Neurosurgical Service, Massachusetts General Hospital, and Department of Surgery, Harvard Medical School, Boston, Massachusetts

1996

Methods

Medline Search and abstract review

Trials needed to be randomized and compare nimodipine vs placebo in a prophylactic setting

Endpoints:

- Glasgow Outcome Scale – some scales converted
- New deficit
- Death
- Radiological Infarct rate – CT based
- Hunt and Hess Grade

Intention to treat analysis

*Outline of metaanalyses performed**

Metaanalysis No.	Outcome Measure
1	good outcome vs. all other grades
2	good or fair outcome vs. other grades
3	overall mortality
4	deficit or mortality attributed to vasospasm or DID
5	mortality attributed to vasospasm or DID
6	cerebral infarction rate (as assessed by CT)
7	deficit or mortality attributed to rebleeding
8	mortality attributed to rebleeding

* Abbreviations: CT = computerized tomography; DID = delayed ischemic deficit.

Studies that met specifications

*Characteristics of trials included in metaanalyses**

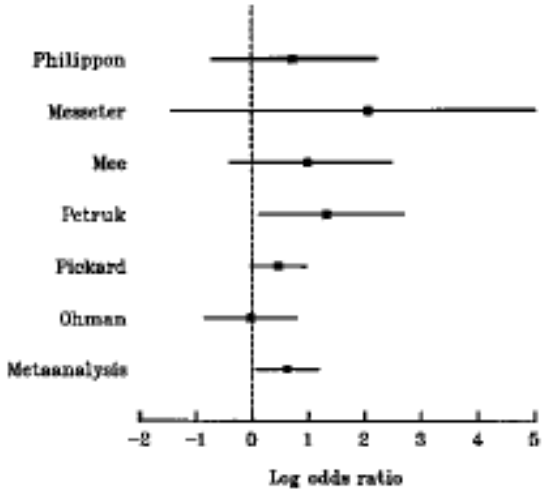
Author & Year	No. of Patients Randomized	No. of Patients Analyzed	No. of Patients Excluded	Method of Nimodipine Administration	Duration of Treatment (days)	Hunt & Hess[43] Grades Included
Allen, *et al.*, 1983	121	116	5	PO	21	I & II
Philippon, *et al.*, 1986	81	70	11	PO	21	I–III
Messeter, *et al.*, 1987	20	20	0	IC, IV	9	I–III
Mee, *et al.*, 1988	75	75†	0†	IC, PO	21	I–V
Petruk, *et al.*, 1988	188	154	34	PO	21	III–V
Pickard, *et al.*, 1989	555	554	1	PO	21	I–V
Öhman, *et al.*, 1991	215	213	2	IV, PO	21	I–III
total	1255	1202†	53†	—	—	—

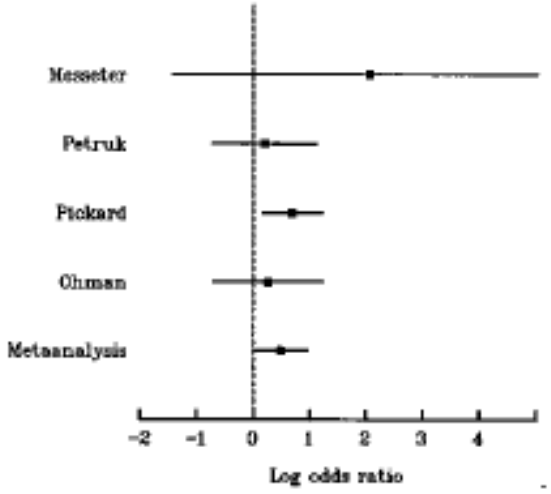
* Abbreviations: IC = intracisternal (during craniotomy); IV = intravenous; PO = oral; — = not applicable.
† Twenty-five patients were excluded from the trial reported by Mee, *et al.*, in Metaanalyses 4, 5, 7, and 8.

Nimodipine given orally in 5 trials

Meta-analysis Outcome

GOS 1
vs
GOS >1





GOS 1-2
vs
GOS >2

Higher chance of good outcome with nimodipine (P = 0.004 OR 1.86 99% CI 1.07-3.25)

Assuming 52% good outcome rate among placebos, one additional good outcome was gained with treatment of 7 patients

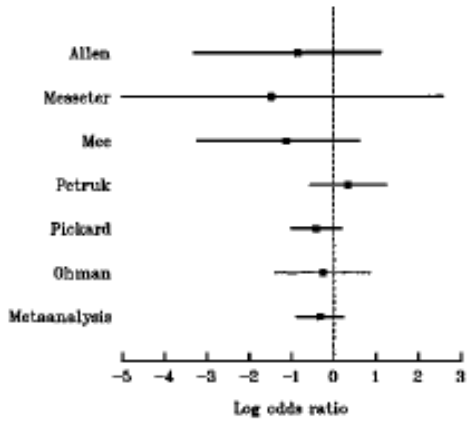
Higher chance of good to fair outcome with nimodipine

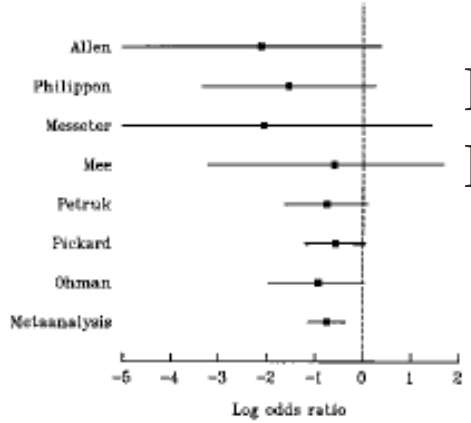
(p < 0.0007, OR 1.67 99% CI 1.13-2.46)

Assuming rate of deficit or death of 28% in placebo group, one additional good or fair outcome for treatment of 10 patients

Mortality and Deficit or Death

Mortality





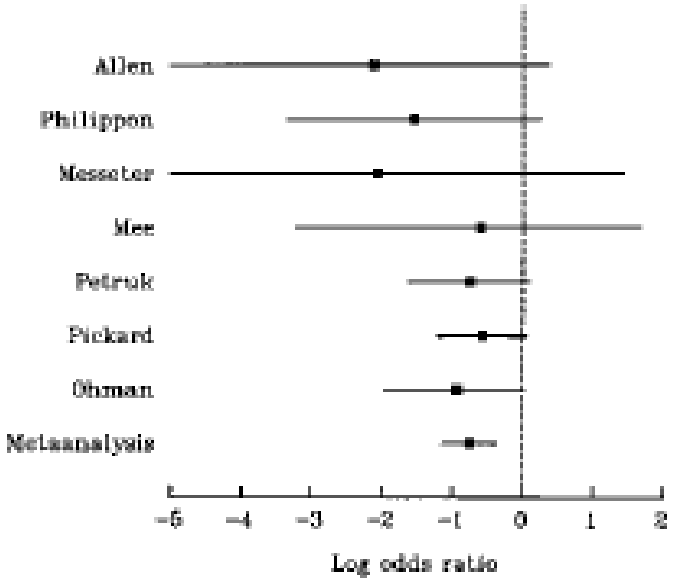
Deficit or Death Related to vasospasm

Nimodipine had no impact on mortality p=0.1

Nimodipine treated patients had a significantly lower incidence of death or deficit related to vasospasm ($p < 0.00001$, OR 0.46, CI 0.31-0.68)

Assuming a 28% rate of deficit or death attributable to delayed ischemia or vasospasm, treatment of 8 patients would result in one fewer death

Radiological Infarction

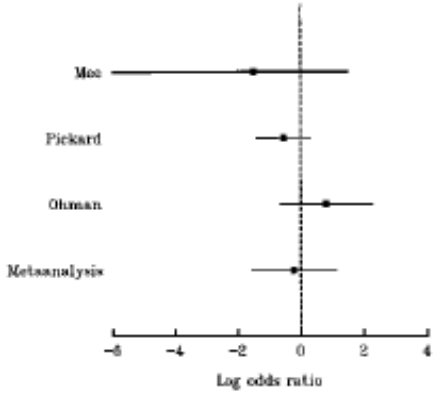


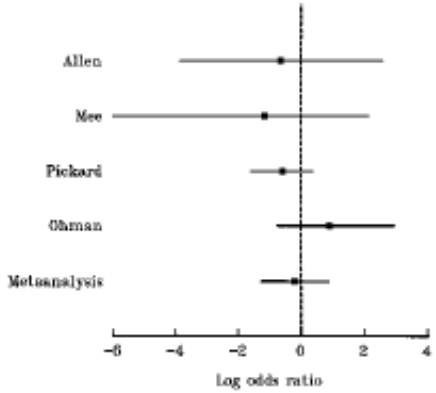
Nimodipine treated patients had less infarcts (p = 0.001, OR 0.58, CI 0.38-0.90)

Assuming a 32% radiographic infarction rate, treatment with nimodipine would prevent 1 stroke every 10 treated patients.

Re-bleeding

Permanent deficit or death from re-bleeding





Death due to Re-bleeding

No reduction in permanent deficit or death from rebleeding

No reduction in death due to rebleeding

Data from 2007 used for guidelines:

- Dorhout Mees S, Rinkel GJE, Feigin VL, Algra A, van den Bergh WM, Vermeulen M, van Gijn J. Calcium antagonists for aneurysmal subarachnoid haemorrhage. *Cochrane Database of Systematic Reviews* 2007, Issue 3. Art. No.: CD000277. DOI: 10.1002/14651858.CD000277.pub3.

Cochran Review

- The meta-analysis included 16 studies with a total of 3361 patients (1665 in the treatment group and 1696 in the control group).
- The number of patients per trial ranged from 20 (Messeter 1987) to 906 (Haley 1993).
- Comparison of:
  1. Poor Outcome
  2. Mortality
  3. Secondary Ischemia
  4. Rebleeding

Cochran Review, the results:

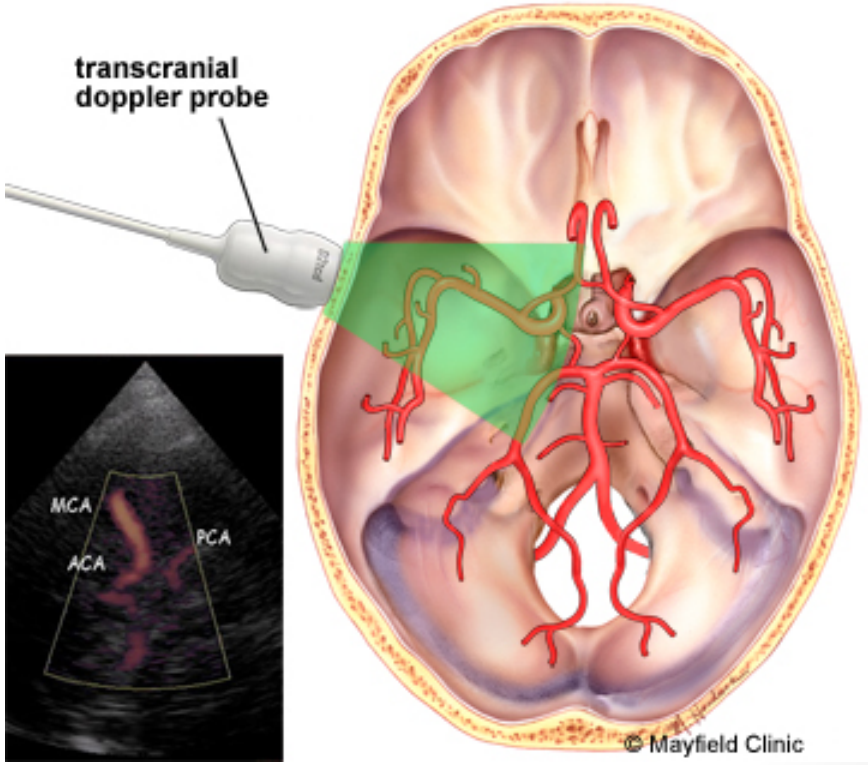
- Reduction of poor outcome (defined as death or dependency in activities of daily life) after SAH, though the reduction of case fatality alone is not statistically significant.
- Reduction in the frequency of secondary ischemia.
- Not associated with an increased frequency of rebleeding.
- Intravenous administration of calcium antagonists is more expensive and potentially hazardous in view of hypotensive effects, and is therefore not recommended.

Therefor:

- oral Nimodipine (60mg every four hours, to be continued for three weeks) as standard treatment in patients with aneurysmal subarachnoid hemorrhage.

ICU monitoring and therapies

- Neurologic Exam
- Role of cEEG
- CSF drainage
- Transcranial Doppler
- Triple H Therapy

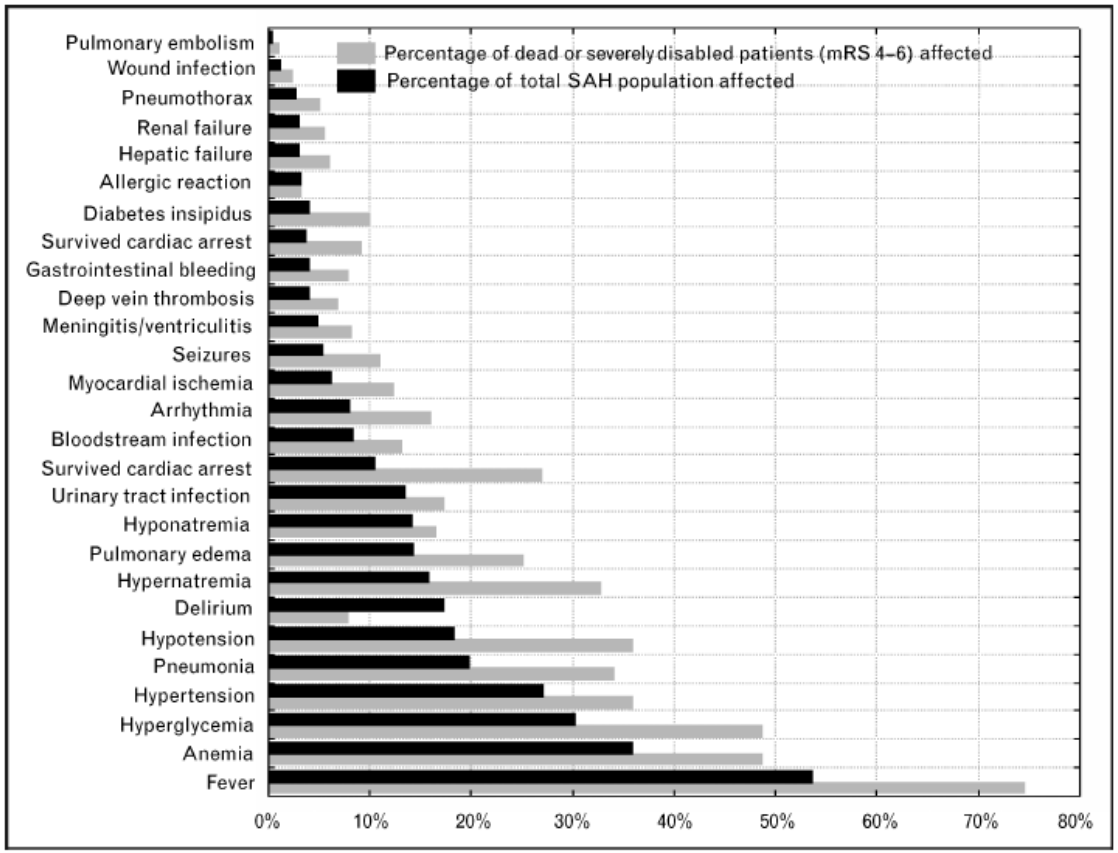


Medical Complications

- Therapies not without regard to patient

Medical Complications

Figure 2 Frequency of medical complications in the total subarachnoid hemorrhage (SAH) population (576 patients) and among patients with poor outcome (220 patients; mRS 4–6) at 3 months



Subarachnoid hemorrhage complications Wartenberg and Mayer 2006

Management of Medical Complications Associated With aSAH: Recommendations

- **1. Administration of large volumes of hypotonic fluids and intravascular volume contraction is not recommended after aSAH *(Class III; Level of Evidence B).***
- **2. Monitoring volume status in certain patients with recent aSAH by some combination of central venous pressure, pulmonary wedge pressure, and fluid balance is reasonable, as is treatment of volume contraction with crystalloid or colloid fluids *(Class IIa; Level of Evidence B).***
- **3. Aggressive control of fever to a target of normothermia by use of standard or advanced temperature modulating systems is reasonable in the acute phase of aSAH *(Class IIa; Level of Evidence B).*** (New recommendation)
- **4. Careful glucose management with strict avoidance of hypoglycemia may be considered as part of the general critical care management of patients with aSAH *(Class IIb; Level of Evidence B).***

Management of Medical Complications cont.

- **5. The use of packed red blood cell transfusion to treat anemia might be reasonable in patients with aSAH who are at risk of cerebral ischemia. The optimal hemoglobin goal is still to be determined *(Class IIb; Level of Evidence B).*** (New recommendation)
- **6. The use of fludrocortisone acetate and hypertonic saline solution is reasonable for preventing and correcting hyponatremia *(Class IIa; Level of Evidence B).***
- **7. Heparin-induced thrombocytopenia and deep venous thrombosis are relatively frequent complications after aSAH. Early identification and targeted treatment are recommended, but further research is needed to identify the ideal screening paradigms *(Class I; Level of Evidence B).*** (New recommendation)


VOLUME
1
2
3
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5
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8
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10
11

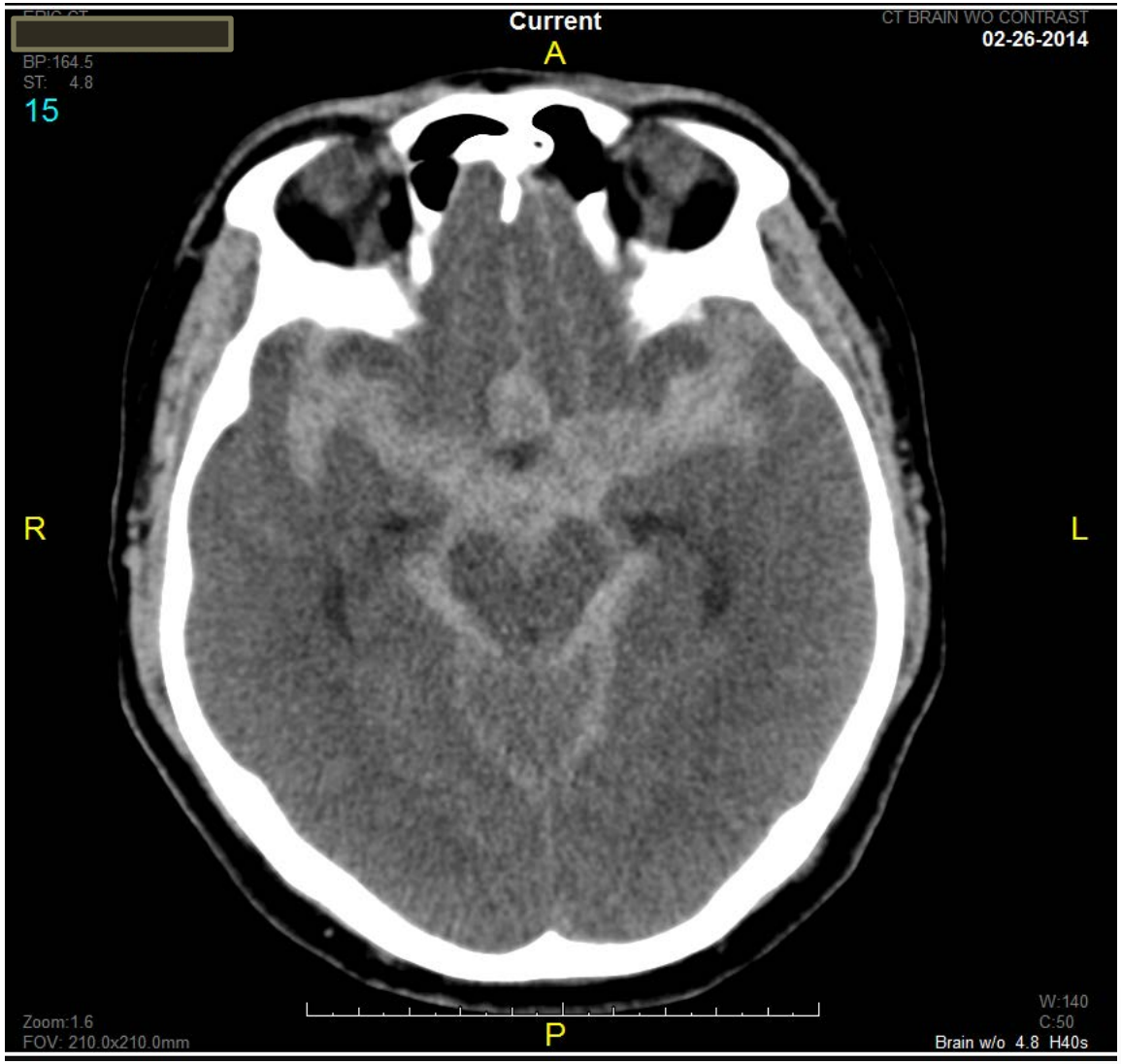
Case Study #1:

- 56yo Woman presents w acute onset HA started 1 hour ago.
- “Worst headache of my life”.
- Hx: HTN
- 10/10, frontal, onset less than 1 minute.
- Acute onset while doing her morning yoga

Work-up?

- What kind of imagining should we use to work-up?
  A) None, she has sinusitis, f/u w primary care provider in 1 week
  B) CT brain (non-contrast)
  C) MRI brain
  D) CT brain with contrast

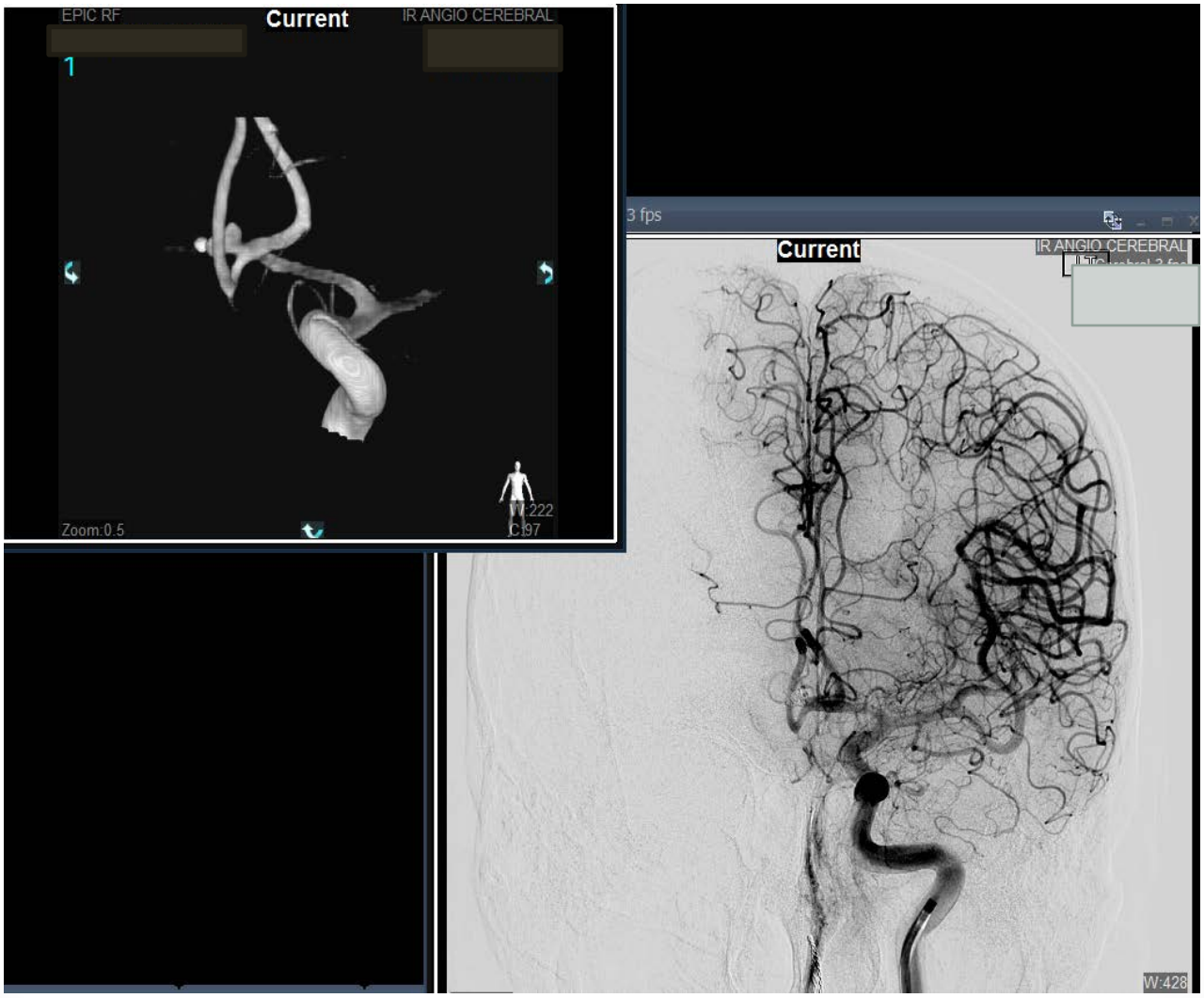
Image:


Current
A
CT BRAIN WO CONTRAST
02-26-2014
BP:164.5
ST: 4.8
15
R
L
P
Zoom:1.6
FOV: 210.0x210.0mm
W:140
C:50
Brain w/o 4.8 H40s

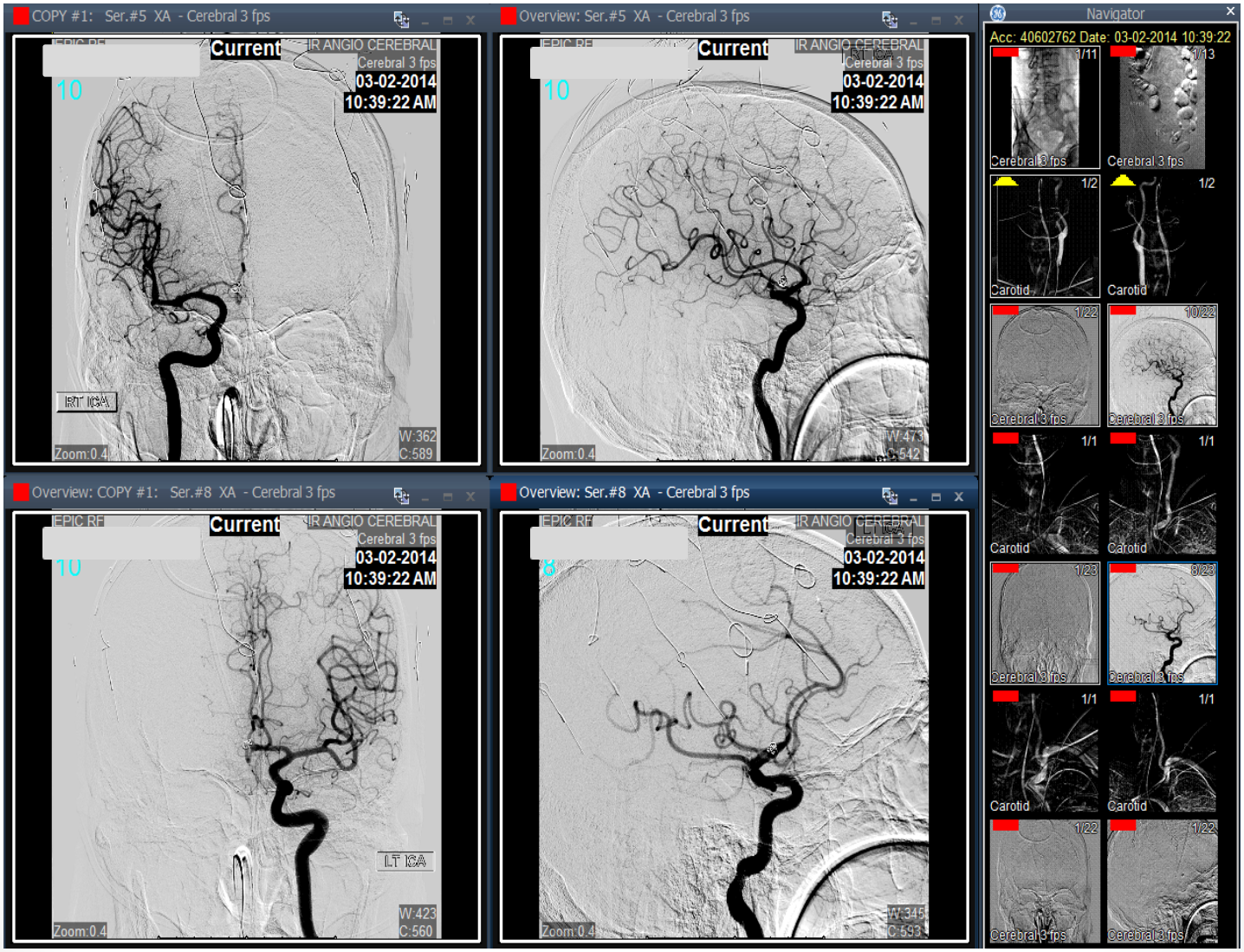
Next steps

- Blood pressure is 184/92, HR 72, NSR
- Is this ok?

Initial management



Initial management



What do we need to monitor for now?

- A) Nothing, aneurysm is stable, patient can be discharged to home.
- B) Hydrocephalus
- C) Delayed Ischemia related to cerebral vasospasm
- D) B and C

Therapies to prevent DIND:

- Nimodipine 60mg PO q 4 hrs x 21 days.
- We continue drainage of CSF via external ventricular drain. Daily transcranial doppler to assess velocities. What kind of fluid management should be implement?
- A) prophylactic hypertension using norepinephrine gtt to push SBP 160-200.
- B) PRBC and 0.45% NaCl to push volume status at least 2 liters positive balance.
- C) titration of ivf to maintain euvolemic status.

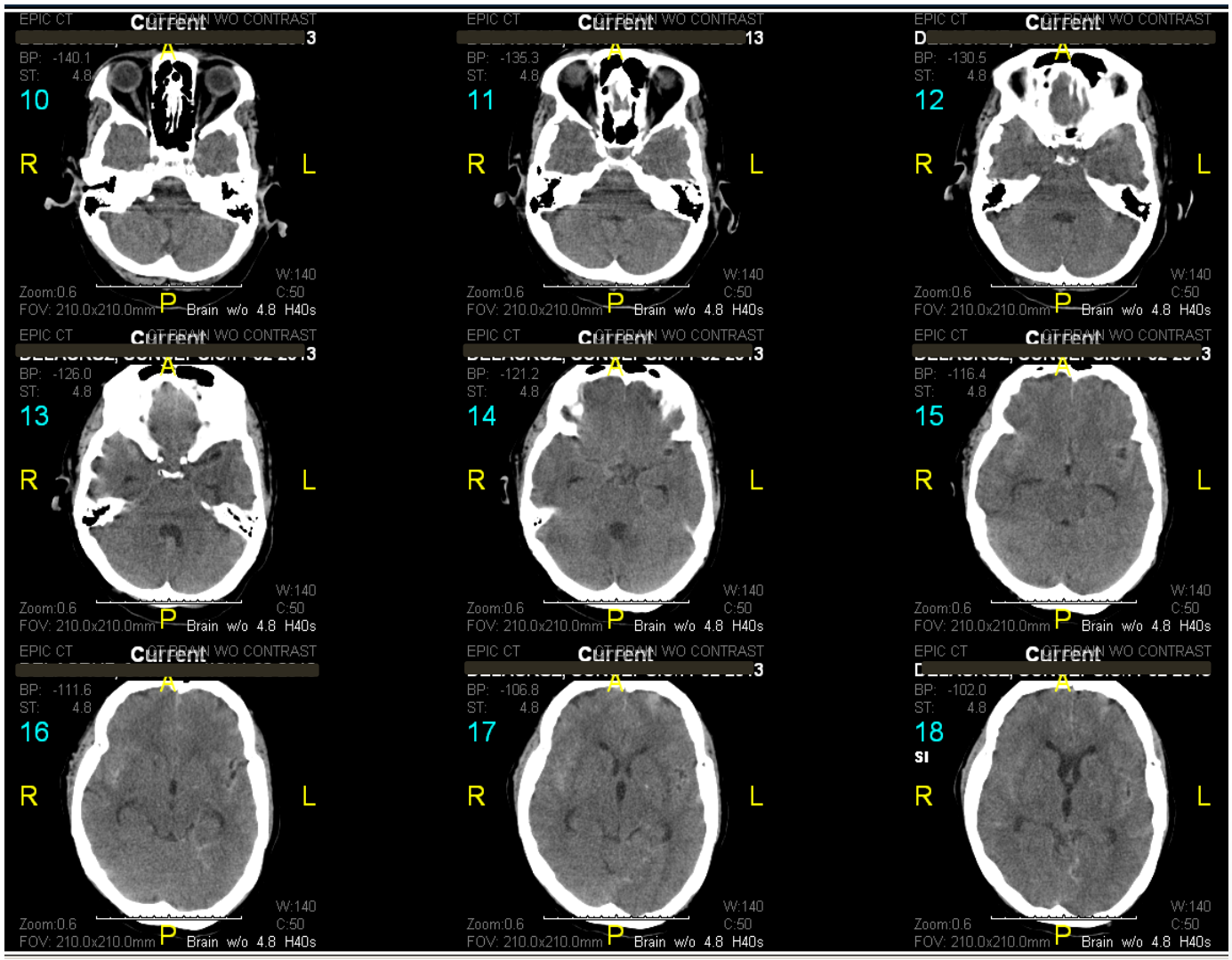
Outcome:

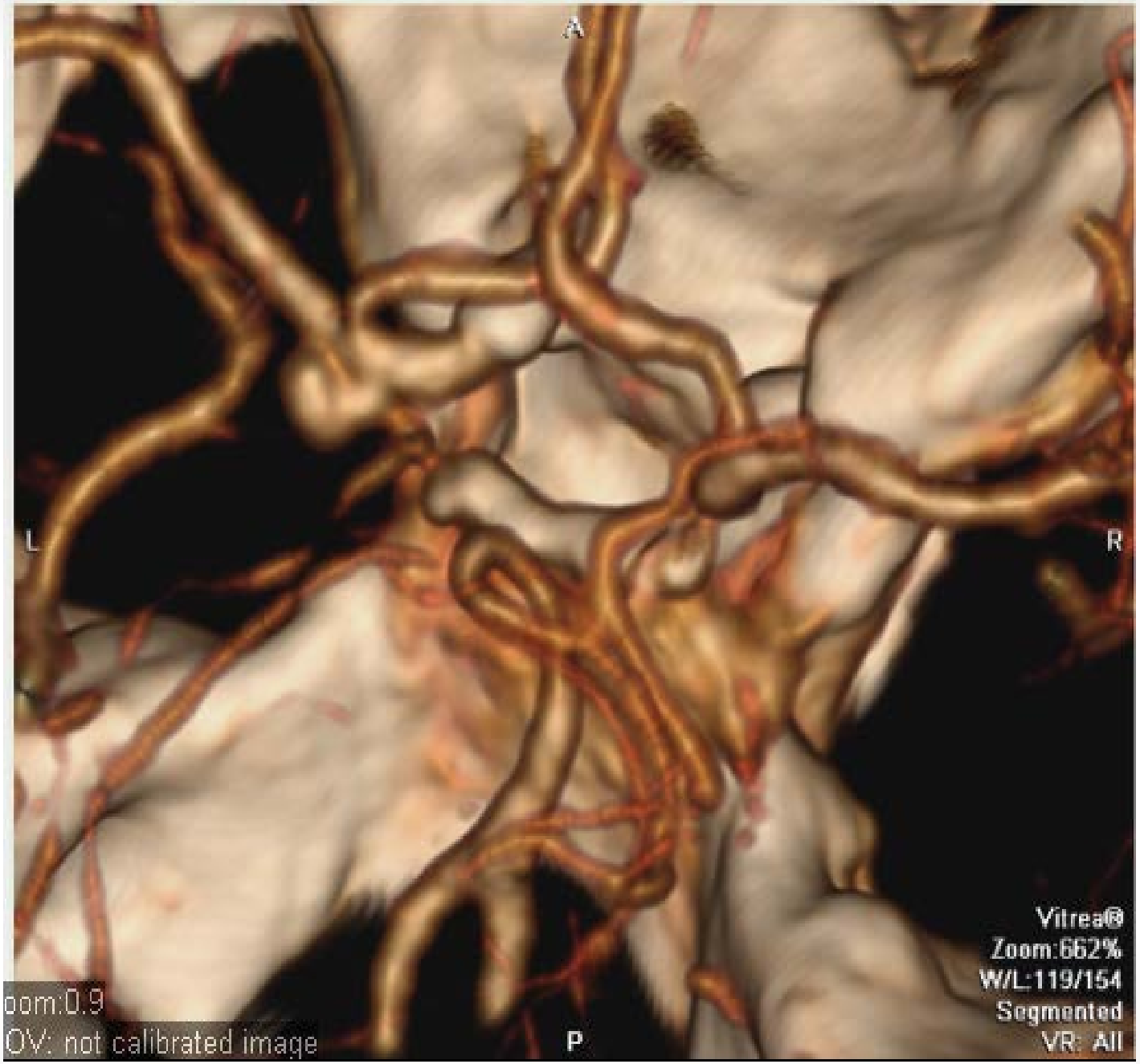
- Fluid balance maintained, Na balance maintained.
- Normothermia goal.
- Daily TCDs without elevation through post bleed day 14.
- Tapering and removal of EVD.
- PT/OT and transfer from ICU.

Case #2

- **HPI:** 67 y.o. female with a history of hypertension, who presents with a subarachnoid hemorrhage.
- The patient reports she has had headaches on and off for the last month, in the past week she has had gradual onset of a severe headache associated with nausea and vomiting. She lives alone and at first tried to self medicate with Aleve, aspirin, and Tylenol but with no relief. In the last two days, she's felt the headache has been especially unrelenting and she's had little PO intake due to nausea and this prompted her to call 911. She was taken to an outside hospital, where CT revealed subarachnoid hemorrhage with blood in the R sylvian fissure, parafalcine sulci bilaterally, and prepontine basilar cisterns; no IVH was seen. The patient was given platelets and ddAVP for reversal of platelet dysfunction from aspirin use and started on nicardipine gtt. She was then transferred to our facility for further management.
- She denies any numbness, weakness, vertigo, diplopia, or dysarthria.

Imagining:

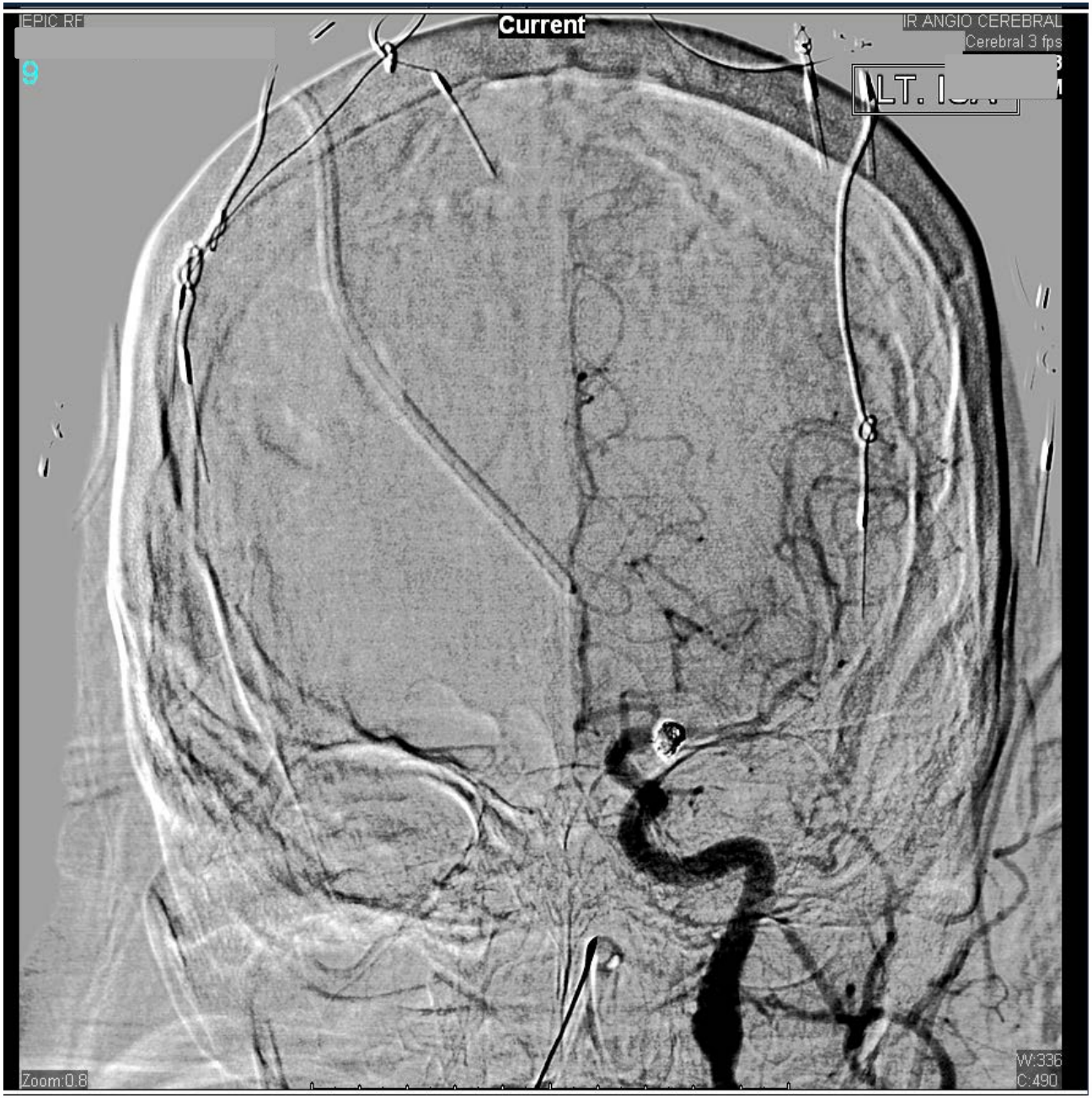

EPIC CT
Current
CT BRAIN WO CONTRAST
BP: -140.1
ST: 4.8
10
A
R
L
P
W:140
C:50
Zoom:0.6
FOV: 210.0x210.0mm
Brain w/o 4.8 H40s
BP: -135.3
ST: 4.8
11
BP: -130.5
ST: 4.8
12
BP: -126.0
ST: 4.8
13
BP: -121.2
ST: 4.8
14
BP: -116.4
ST: 4.8
15
BP: -111.6
ST: 4.8
16
BP: -106.8
ST: 4.8
17
BP: -102.0
ST: 4.8
18
SI


A
L
R
P
Vitrea®
Zoom:662%
W/L:119/154
Segmented
VR: All
oom:0.9
OV: not calibrated image

What happens next:

- She undergoes coil embolization of a L PCOM aneurysm. She is monitored in the ICU post embolization. Day 2 p embolization, she becomes increasingly sleepy.
- What should we be worried about?
- A) Nothing, she is in the icu where every 2 hours a nurse barges into the room to wake her up and ask her orientation questions.
- B) Hydrocephalus.
- C) Delayed cerebral ischemia due to cerebral vasospasm.
- D) B and C.

- She is intubated for airway protection, receives an external ventricular drain.
- TCDs demonstrate elevated velocities.
- What should we do regarding:
  Blood pressure goal?
  Fluid management?
  Other physiology issues to control?


EPIC RF
Current
IR ANGIO CEREBRAL
Cerebral 3 fps
9
Zoom:0.8
W:336
C:490

ICU management continues:

- Daily cerebral angiogram to administer intraarterial therapy.
- Despite aggressive measures…


Current
MRI BRAIN WO CONTRAST
localizer
ep2d_diff_3scan_trace
ep2d_diff_3scan_trace_ADC
Prior
CT BRAIN WO CONTRAST
Head Topo 0.6 T20s
Brain w/o 4.8 H40s
Navigator - Total 2 studies
Sag T1
Ax TSE T2
AX FLAIR
Ax SE T1
AX GRE
Cor Obl T2 TSE

Outcomes

- Unfortunate

Dr. Bleck clarifies

- … a realistic evidence based rating scale:
- Class 0: Things I believe
- Class 0a: Things I believe despite the available data
- Class 1: Randomised controlled clinical trials that agree with what I believe
- Class 2: Other prospectively collected data
- Class 3: Expert opinion
- Class 4: Randomised controlled clinical trials that don't agree with what I believe
- Class 5: What you believe that I don't.

BMJ. Jul 22, 2000; 321(7255): 239.

Thanks