

MMD 6513 – ANESTHÉSIE ET SYSTÈME NERVEUX MALADIES NEUROVASCULAIRES

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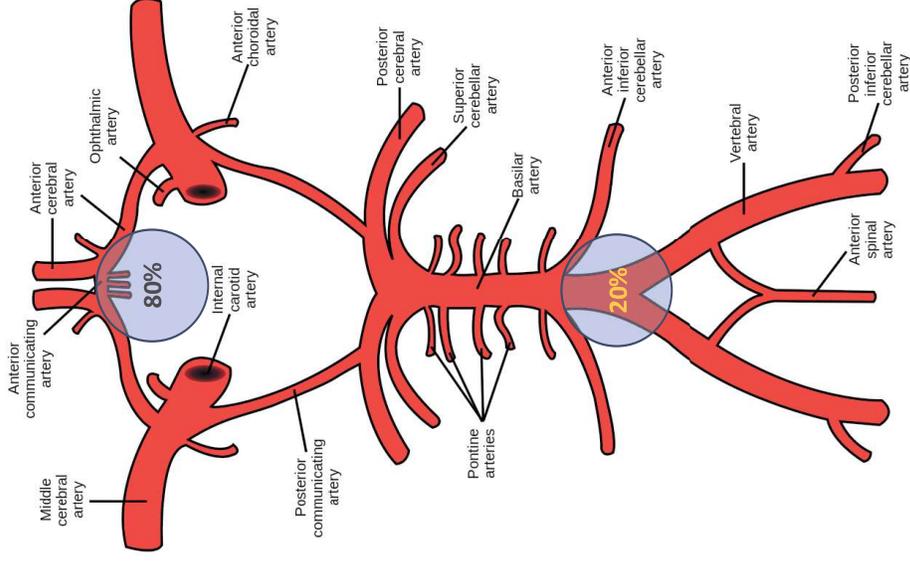
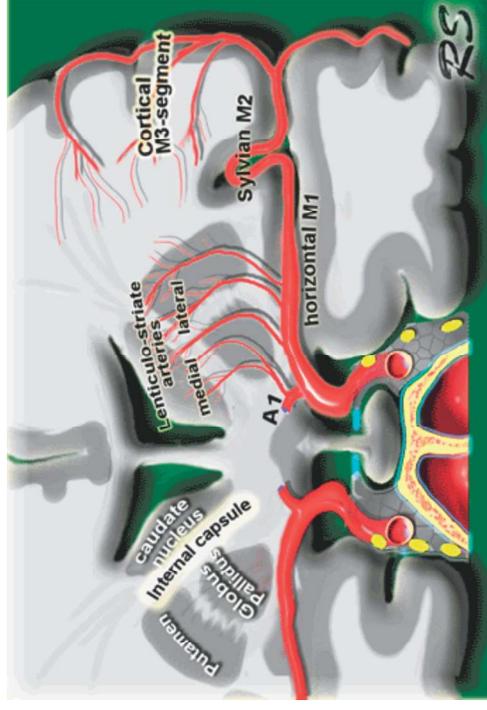
HIVER 2026

Objectif / plan

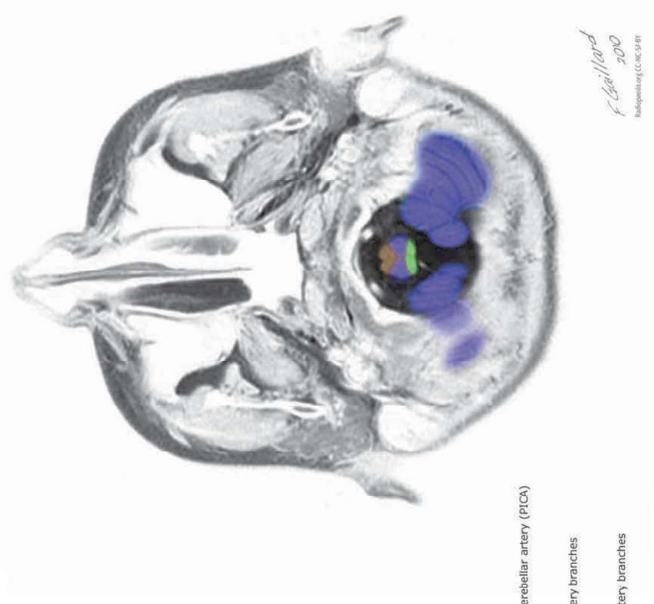
- Accident vasculaire cérébral (ischémique)
- Hémorragie intracérébrale
- Hémorragie sous-arachnoïdienne



Rappel de neuroanatomie vasculaire



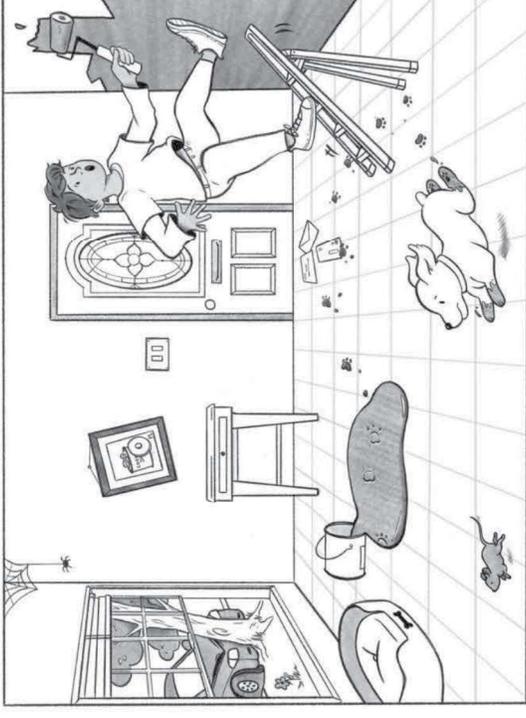
Cerebral Vascular Territories



- Posterior inferior cerebellar artery (PICA)
- Anterior spinal artery branches
- Posterior spinal artery branches

NIH Stroke Scale (NIHSS)

- Examen neurologique standardisé
- 15 items, total 0-42 points
 - État de conscience 0-3
 - Fonction visuelles
 - Langage
 - Fonction motrices
 - Fonction sensibles et négligence
 - Fonction cérébelleuse
- NIHSS > 10 ➔ 80% occlusion proximale



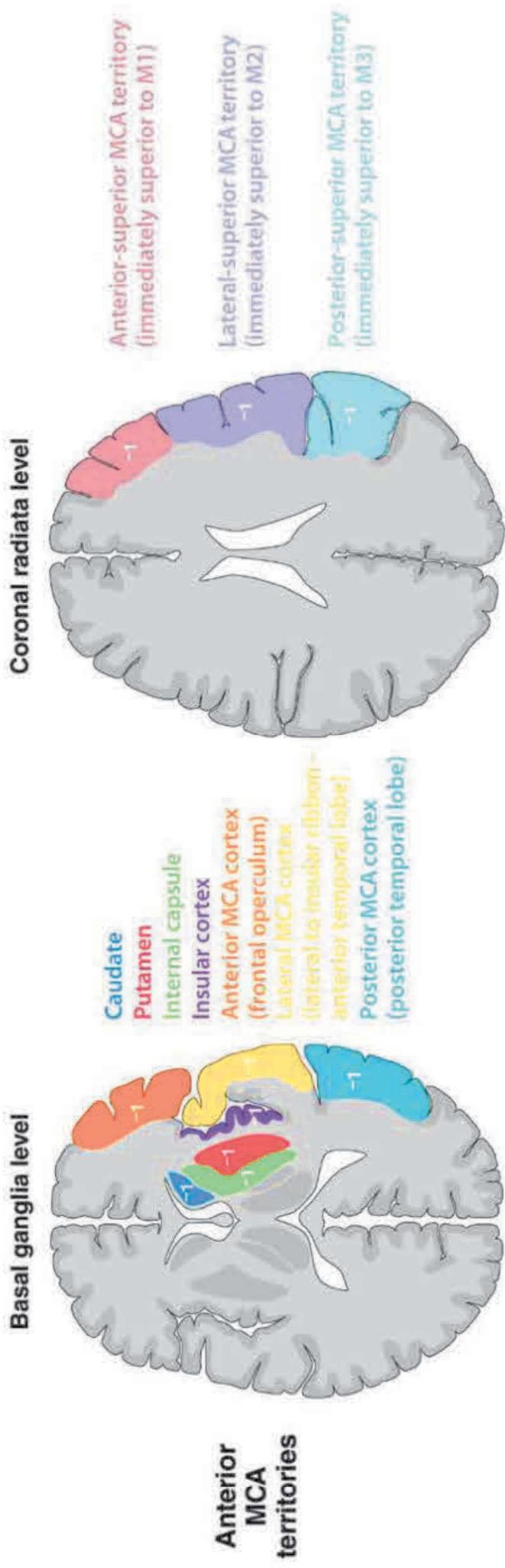
0-4 AVC mineur

5-15 AVC modéré

16-20 AVC mod-sévère

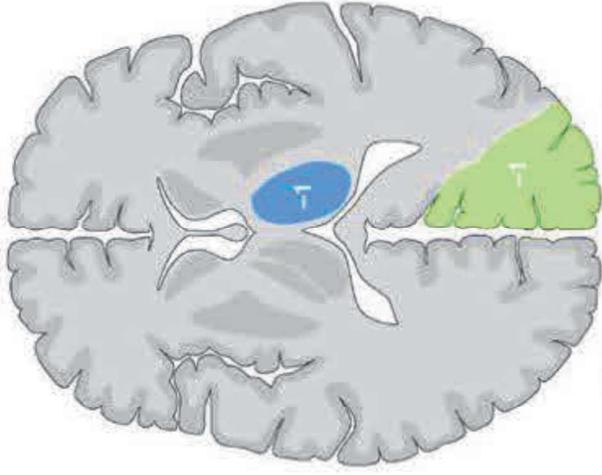
21-42 AVC sévère

Alberta Stroke Program Early CT Score (ASPECTS)



PC-ASPECTS

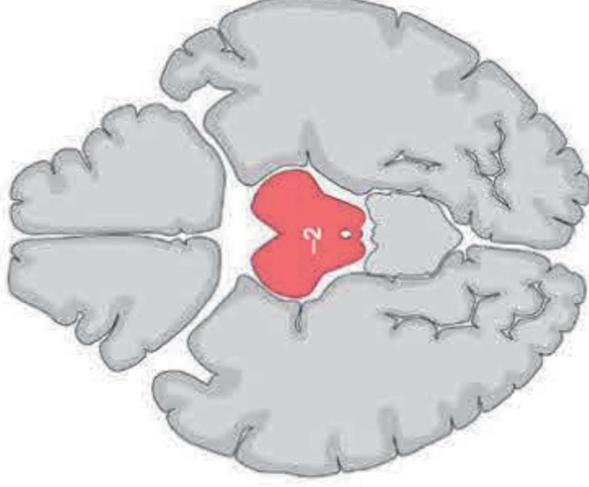
Thalami level



Thalami (1 point each)

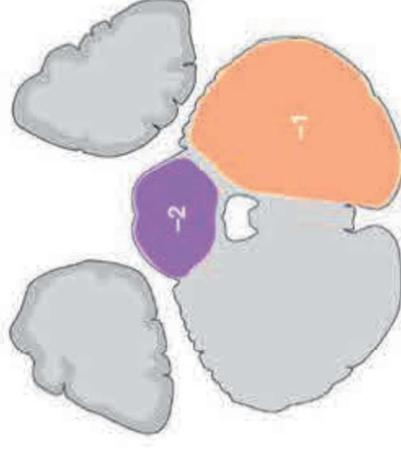
Occipital lobes (1 point each)

Midbrain level



Midbrain (2 points)

Pons level



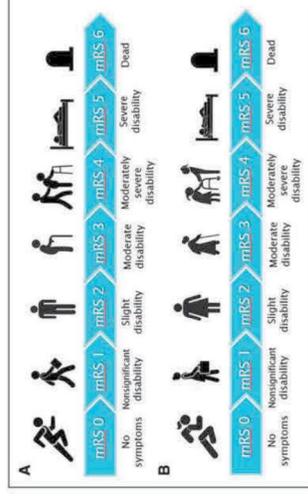
Pons (2 points)

Cerebellar hemispheres (1 point each)

Posterior
circulation
territories

MODIFIED RANKIN SCALE (MRS)

0. No symptoms
1. No clinically significant disability – able to carry out all usual activities despite some symptoms
2. Slight disability – able to look after own affairs without assistance but unable to carry out all previous activities
3. Moderate disability – requires some help but able to **walk unassisted**
4. Moderately severe disability – unable to attend to **bodily needs** without assistance and unable to walk unassisted
5. Severe disability – requires constant nursing care, bedridden, incontinent
6. Dead



Accident vasculaire cérébral ischémique

AHA/ASA GUIDELINE

2026 Guideline for the Early Management of Patients With Acute Ischemic Stroke: A Guideline From the American Heart Association/American Stroke Association

Endorsed by the American Association of Neurological Surgeons/Congress of Neurological Surgeons, Neurocritical Care Society, the Society for Academic Emergency Medicine, the Society of NeuroInterventional Surgery, and the Society of Vascular and Interventional Neurology.

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

The Canadian Journal of Neurological Sciences (2024), 51, 1–31
doi:10.1017/cjn.2022.344



Review Article

Canadian Stroke Best Practice Recommendations: Acute Stroke Management, 7th Edition Practice Guidelines Update, 2022

Manraj Heran¹, Patrice Lindsay², Gord Gubitz^{3,4}, Amy Yu^{5,6}, Aravind Ganesh⁷, Rebecca Lund², Sacha Arsenault⁸, Doug Bickford⁹, Donnita Derbyshire¹⁰, Shannon Doucette¹¹, Eszeddeeg Ghrooda¹², Devin Harris^{13,14}, Nick Kanya-Forstner^{15,16}, Eric Kaplovitch^{6,17}, Zachary Liederman^{6,17}, Shauna Martiniuk^{6,18}, Marie McClelland¹⁹, Genevieve Milot²⁰, Jeffrey Minuk²¹, Erica Otto²², Jeffrey Perry²³, Rob Schlamp²⁴, Donatella Tampieri²⁵, Brian van Adel²⁶, David Volders²⁷, Ruth Whelan²⁸, Samuel Yip²⁹, Norine Foley³⁰, Eric E. Smith⁷, Dar Dowlatshahi³¹, Anita Mountain³², Michael D. Hill⁷, Chelsy Martin² and Michel Shamy³¹

Society for Neuroscience in Anesthesiology and Critical Care Expert Consensus Statement: Anesthetic Management of Endovascular Treatment for Acute Ischemic Stroke*

Endorsed by the Society of NeuroInterventional Surgery and the Neurocritical Care Society

Pekka O. Talke, MD,* Deepak Sharma, MD, DM,† Eric J. Heyer, MD, PhD,‡
Sergio D. Bergese, MD,§ Kristine A. Blackham, MD,|| and Robert D. Stevens, MD¶

(J. Neurosurg. Anesthesiol. 2014;26:95–108)

Évolution spectaculaire de la littérature

- 1980 découverte t-PA
- 1995 NINDS
- 2008 ECASS III
- 2015 Thrombectomie
- 2018 Thrombolyse hors-délai (wake-up)
- 2018 Thrombectomie hors-délai
- 2020 Thrombectomie circulation postérieure
- 2022 Thrombectomie infarctus importants
- 2025 Occlusion distale?
- 2025 Thrombolyse IA post reperfusion par thrombectomie

⚠ TIME IS BRAIN ⚠

- Chaque minute d'occlusion → 1 900 000 neurones perdus

- THROMBOLYSE

Délai de 30 minutes → ↓ 10% probabilité de mRS 0-1 à 3 mois

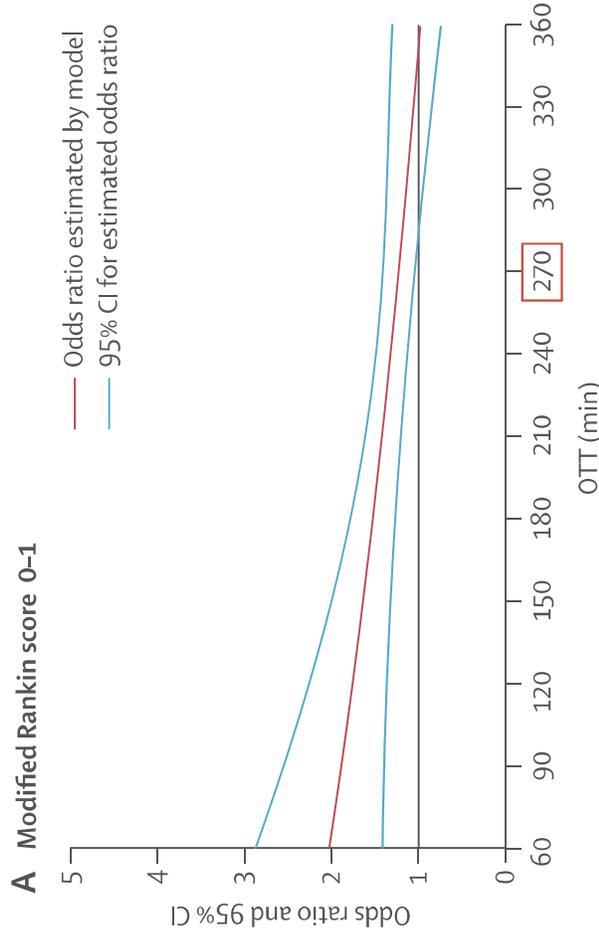
- THROMBECTOMIE

Délai de 9 minutes → ↑ 1% probabilité d'empirer mRS à 3 mois



Thrombolyse

- **NINDS NEJM 1995** 624 pts us r-tPA vs placebo < 3h
 - Meilleur évolution (NIHSS, Barthel, mRS, GOS) à 3 mois (mRS 0-1 39 vs 26%, NNT ~ 8)
- **ECASS III NEJM 2008** 821 pts EU, r-tPA entre 3-4.5h
 - ↑ mRS 0-1 à 90 jrs (52 vs 45%), plus sICH 2.4 vs 0.2%, mortalité idem (NNT ~ 14)



Impact du délai de traitement: thrombolyse

	Modified Rankin score 0–1 at 90 days,*		Odds ratio (95% CI)	p value	Estimated number needed to treat† for modified Rankin score 0–1
	Alteplase n/N (%)	Placebo n/N (%)			
0–90 min	67/161 (41.6%)	44/151 (29.1%)	2.55 (1.44–4.52)	0.0013	4.5
91–180 min	127/303 (41.9%)	91/315 (28.9%)	1.64 (1.12–2.40)	0.0116	9.0
181–270 min	361/809 (44.6%)	306/811 (37.7%)	1.34 (1.06–1.68)	0.0135	14.1
181–270 min (excluding EPITHET ⁷ data)	358/795 (45.0%)	303/794 (38.2%)	1.32 (1.04–1.66)	0.0202	14.9
271–360 min	215/575 (37.4%)	193/542 (35.6%)	1.22 (0.92–1.61)	0.1628	21.4
271–360 min (excluding EPITHET ⁷ data)	200/539 (37.1%)	184/512 (35.9%)	1.16 (0.87–1.54)	0.3063	28.7

- Réduction des bénéfices avec le temps...
- Augmentation de mortalité après > 4.5h de délai...
- Délai de 30-minutes = réduction de probabilité d'évolution favorable vers autonomie complète (mRS 0-1) à 3 mois de 10%

Thrombolyse: hors délai

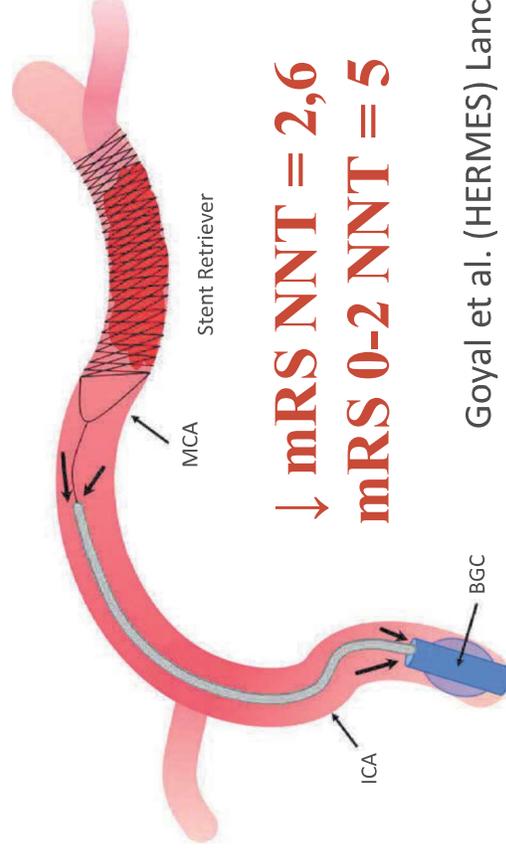
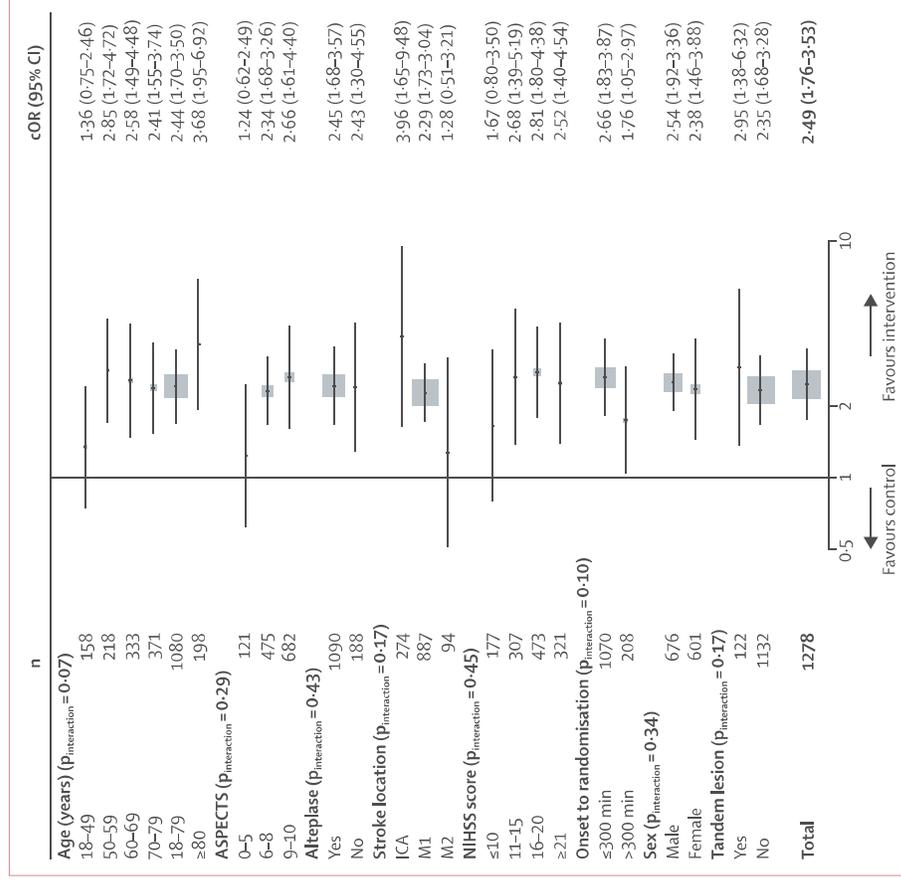
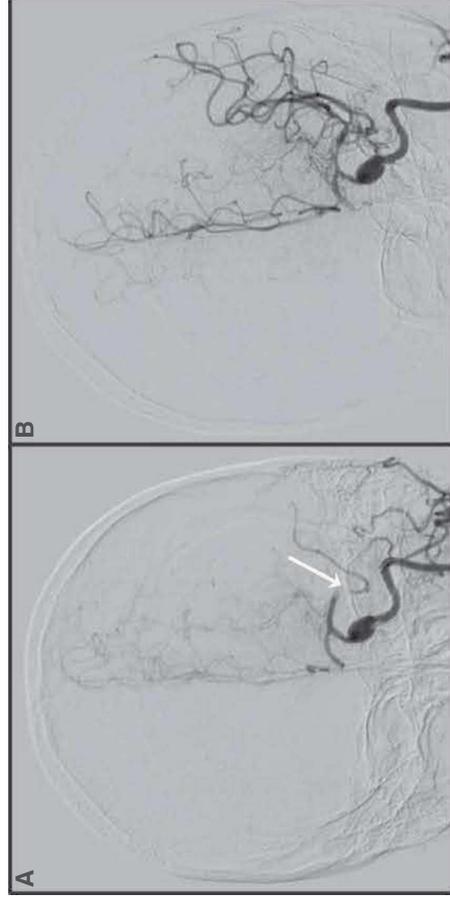
- **WAKE-UP NEJM 2018** 503/800 pts EU r-TPA vs placebo, wake-up stroke ou délai incertain avec imagerie favorable (mismatch DWI/FLAIR suggérant délai probable < 4.5h)
 - ↑ mRS 0-1 à 90 jrs **53 vs 42%**, augmentation mortalité (4 vs 1%), tendance plus sICH
 - (arrêt de financement)
- **EXTEND NEJM 2019**  225/310 pts r-TPA vs placebo, LKW 4.5-9h avec imagerie favorable (CTP ou IRM) délai LKW-r-TPA ~ 7h12
 - ↑ mRS 0-1 à 90 jrs 35 vs 30% (**OR ajusté âge+NIHSS 1.44**), tendance plus de sICH (6 vs 1%)
 - (perte equipoise)
- **ECASS-4: ExtEND Int J Stroke 2019** 119/264 pts EU r-TPA vs placebo, LKW 4.5-9h avec imagerie favorable (IRM) délai ~ 7h42
 - Distribution mRS idem, mortalité 12 vs 7% (NS)
 - (recrutement lent)
- **THAWS Stroke 2020** 131/300 pts JP r-TPA 0.6 mg/kg vs placebo, LKW > 4.5h avec imagerie favorable (idem à WAKE-UP)
 - Évolution favorable, mortalité, sICH idem
 - (perte equipoise)

Thrombectomie: résultats SPECTACULAIRES!

- **MR CLEAN NEJM 2015** 500 pts NL, LVO (CI,M1M2,A1A2), EVT < 6h (thrombectomie +/- thrombolyse IA (~10%)
 - Thrombolyse IV préalable permise (89%), NIHSS 18
 - ↑ mRS 0-2 à 90 jrs (33 vs 19%), OR 1.67 (1.21-2.30) d'améliorer mRS de 1 point avec tx
- **ESCAPE NEJM 2015** 316/500 pts CA, LVO (CI,M1M2), ASPECT 6-10, bonne collatérales, MT < 12h
 - Thrombolyse IV préalable permise (73-79%) NIHSS 16-17, délai LKW-rando ~ 3h
 - ↑ mRS 0-2 à 90 jrs (53 vs 29%), **OR 2.6 (1.7-3.8)**, réduction mortalité 10 vs 19%, sICH idem (**interrompue pour efficacité**)
- **EXTEND-IA NEJM 2015** 70/100 pts AUNZ, LVO (CI,M1M2), r-TPA IV reçu, mismatch favorable (RAPID), MT < 6h
 - NIHSS 17 vs 13 (en défaveur du groupe thrombectomie)
 - ↑ reperfusion à 24h et amélioration NIHSS à 3 jrs, ↑ mRS 0-2 à 90 jrs (71 vs 40%), sICH 0 vs 6 (**perte equipose**)
- **SWIFT PRIME NEJM 2015** 196/833 pts USEU, LVO (CI,M1), r-TPA IV, mismatch favorable (RAPID), MT < 6h
 - NIHSS 17, ASPECTS 9
 - mRS shift favorable, ↑ mRS 0-2 à 90 jrs (60 vs 35%), mortalité idem (**interrompue pour efficacité**)
- **REVASCAT NEJM 2015** 206/500 pts ES, LVO (CI,M1M2), r-TPA IV sans recanalisation ou CI, ASPECTS > 6, MT < 8h
 - NIHSS 17, ASPECTS 7-8
 - mRS shift favorable, ↑ mRS 0-2 à 90 jrs (44 vs 28%), sICH idem (4,9 vs 1,9%) (**perte equipose**)
- **THRACE Lancet Neurology 2016** 414 pts FR, LVO (CI,M1), r-TPA IV reçu < 4h, TX < 5h
 - Pas de critères d'imagerie (ASPECT 0-4 ~14%), NIHSS 18-17, délai r-TPA-rando < 20 min (tous thrombolyés pré-rando)
 - Pas d'évaluation de réponse à r-TPA → 29% des patients n'ont pas eu (besoin) de thrombectomie → ↑ mRS 0-2 à 90 jrs (53 vs 42%), mortalité et sICH idem

Thrombectomie

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Antoine Halwagi, MD FRCPC



↓ **mRS NNT = 2,6**
mRS 0-2 NNT = 5

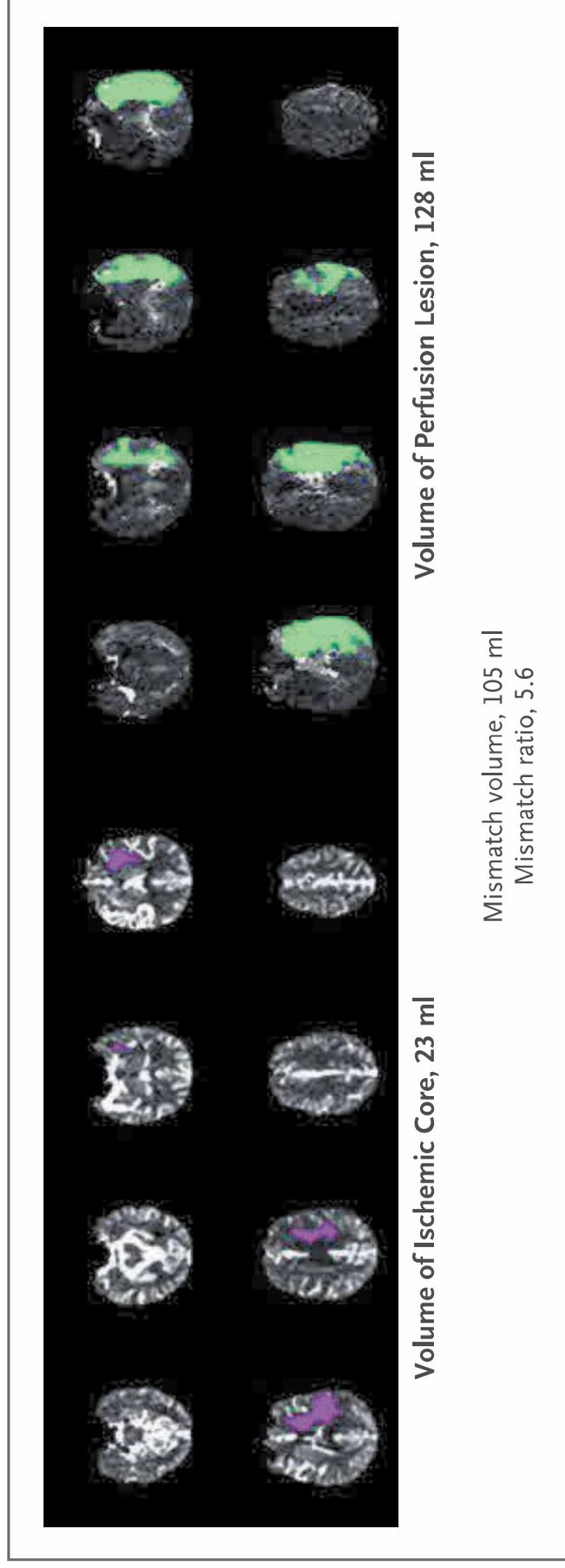
Goyal et al. (HERMES) Lancet 2016
Chowdhury et al. Can J Anesth 2022

Thrombectomie: hors délai

- **DAWN NEJM 2018**  206/500 pts LVO (CI, M1), NIHSS ≥ 10 , LKW 6-24h pré-rando, Mismatch clinico-radiologique (NIHSS vs core CTP ou DWI RAPID, sous groupes âge/core)
 - NIHSS 17, délai LKW-rando 12-13h, core 7.6 vs 8.9 mL, r-tPA 5 vs 13%
 - Réduction du volume d'infarctus cérébral, plus de recanalisation
 - \uparrow mRS et mRS 0-2 à 90 jrs (49 vs 13%), sICH 6 vs 3% NS, mortalité 19 v 18% NS
 - (interruption à 1ère analyse intérimaire pour d'efficacité préspecifié)
- **DEFUSE 3 NEJM 2018** us 182/476 LVO (CI, M1), NIHSS ≥ 6 , LKW 6-18h, core < 70mL Mismatch radiologique (CTP ou DWI/perfusion) ratio ≥ 1.8 ou pénombre ≥ 15 mL
 - NIHSS 16, ASPECT 8, délai LKW-rando 9.5h (pour les non-wake-up stroke), core 9 vs 10 mL
 - mRS shift favorable (OR 2.77), \uparrow mRS 0-2 à 90 jrs (45 vs 17%), sICH 7 vs 4% NS, \sim \downarrow mortalité (14 vs 26%)
 - (interruption pour efficacité suivant publication de DAWN)
- **MR CLEAN-LATE Lancet 2023** NL 502 pts LVO (CI, M1, M2), NIHSS ≥ 2 , LKW 6-24h, flot collatéral ACM CTA **pts remplissant critères DAWN/DEFUSE 3 exclus**
 - NIHSS 10, ASPECT 9 vs 8, délai LKW-rando ~ 11.5 h, $\sim 30\%$ M2
 - mRS médian 3 vs 4 (OR 1.67 1.20-2.32), mRS 0-2 à 90 jrs idem (39 vs 34%), sICH 7 vs 2%, mortalité idem
 - Résultats moins spectaculaires que DAWN et DEFUSE 3 mais significatifs néanmoins
- **RESILIENT-Extend (ISC 2024, non publié) BR 245 pts LVO, LSW 8-12h, CT/CTA seulement**
 - NIHSS 16, ASPECTS 7-8
 - \uparrow mRS 0-2 à 90 jrs (25 vs 14%), sICH idem

stryker

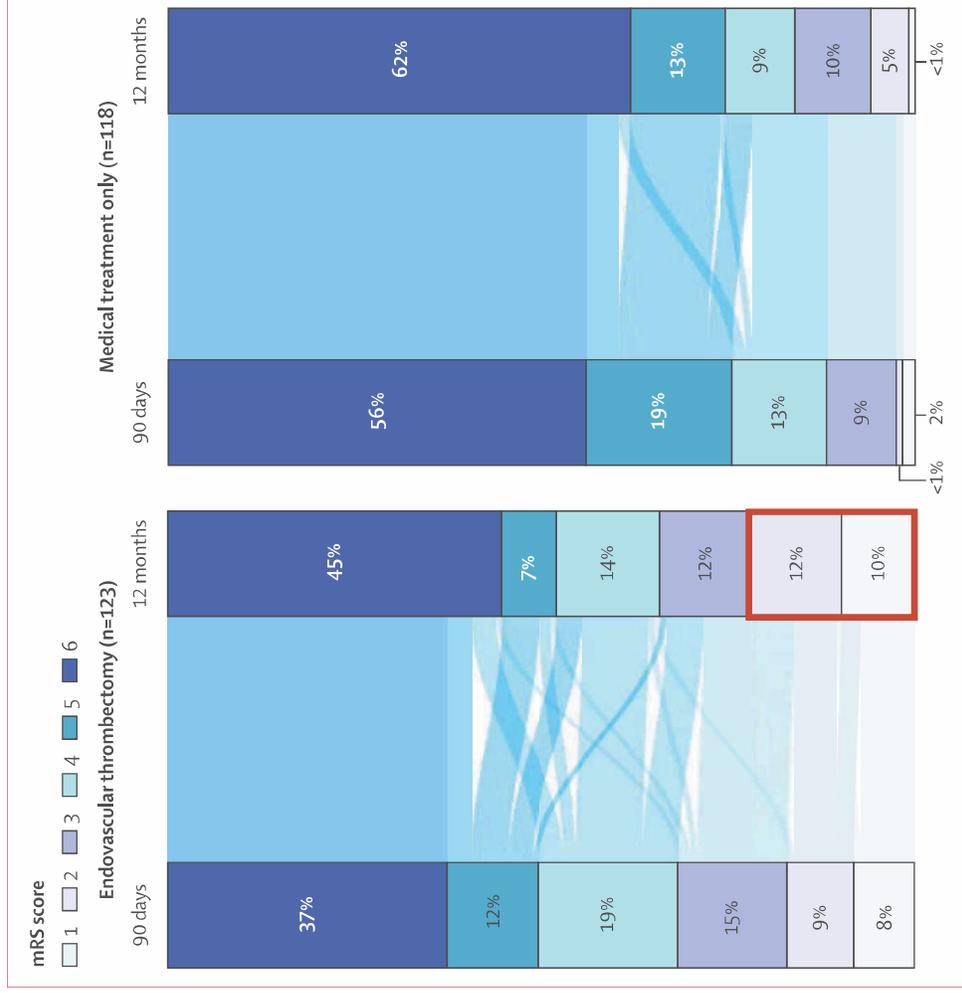
Exemple de quantification RAPID



Thrombectomie: infarctus étendu

- **RESCUE-Japan LIMIT NEJM 2022 JP 202** pts, NIHSS ≥ 6 , LVO (CI,M1), ASPECTS 3-5, LKW < 6h ou 6-24h si FLAIR-, délai tx < 60 min (exclu si effet de masse)
 - NIHSS 22, ASPECTS 3 vs 4 (IRM surtout), core 94 vs 110 mL, délai $\sim 3.7h$, r-TPA 27%
 - \uparrow **mRS 0-3 à 90 jrs (31 vs 13%), NNT ~ 6** , \uparrow ICH 58 vs 31%, sICH (9 vs 5%), DC (10 vs 14%) et mortalité idem
- **ANGEL-ASPECT NEJM 2023** CN 456/502 pts, LVO (CI,M1), ASPECT 3-5 ou core 70-100mL (CTP/ADC), LKW < 24h, NIHSS 6-30
 - NIHSS 16, ASPECT 3, core 62 mL, délai $\sim 7.6h$, r-TPA 28%
 - **mRS shift favorable (OR 1.37)**, \uparrow \uparrow mRS 0-2 à 90 jrs (30 vs 12%), \uparrow \uparrow mRS 0-3 à 90 jrs (47 vs 33%), ICH 49 vs 17%, sICH (6 vs 3%) idem, DC (7 vs 4%) idem et mortalité idem
 - **(interrompue pour efficacité après analyse intérimaire)**
- **SELECT2 NEJM 2023**  352/560 pts, LVO (CI,M1), ASPECTS 3-5 ou core > 50mL (CTP/ADC), LKW < 24h
 - NIHSS 19, ASPECTS 4, core 80 mL, délai LKW-rando $\sim 9.8h$, r-TPA $\sim 19\%$, AG 59%
 - **mRS shift favorable (OR 1.51)**, \uparrow mRS 0-2 à 90 jrs (20 vs 7%), \uparrow \uparrow mRS 0-3 à 90 jrs (38 vs 19%), sICH (1%) et mortalité ($\sim 40\%$) idem
 - Détérioration neuro précoce 24 vs 16% = oedème cérébral?
 - **(interrompue pour efficacité après analyse intérimaire précoce – re: publication RESCUE-Japan LIMIT)**
- **TENSION Lancet 2023** EUCA 253/665 pts, LVO (CI,M1), ASPECTS 3-5, LKW < 11h, NIHSS < 26 (**pas d'IRM,CTP,RAPID**)
 - NIHSS 19, CT/CTA $\sim 80\%$, r-TPA $\sim 36\%$, délai LKW-rando $\sim 2h$, délai LKW-revasc $\sim 4.4h$, AG 44%
 - **mRS shift favorable (OR 1.51)**, \uparrow mRS 0-2 à 90 jrs (17 vs 2%), \uparrow \uparrow mRS 0-3 à 90 jrs (31 vs 13%), sICH (1%), \downarrow mortalité (40 vs 51%), sICH (5%) et CD (8%) idem
 - **(interrompue pour efficacité après 1ère analyse intérimaire)**
- **LASTE NEJM 2024** FRES 333/450 pts, LVO (CI,M1), ASPECTS < 5, NIHSS > 6, LKW < 6.5h
 - NIHSS 21, ASPECT 2, core ~ 134 mL, délai LKW-rando $\sim 4.5h$, délai LKW-MT $\sim 5h$, r-TPA $\sim 35\%$
 - **mRS shift favorable (OR 1.63)**, \uparrow mRS 0-2 à 90 jrs (13 vs 5%), \uparrow \uparrow mRS 0-3 à 90 jrs (34 vs 12%), \downarrow mortalité (36 vs 56%), sICH (10 vs 6%) idem, CD (9 vs 12%) et détérioration précoce idem
 - **(interrompue suivant publication 1-3 et pour efficacité suivant analyse intérimaire précoce)**
- **TESLA JAMA 2024** us 300 pts, LVO (CI,M1), ASPECTS 2-5, LKW < 24h, NIHSS > 6 (**pas d'IRM,CTP,RAPID**)
 - NIHSS 19, délai rando $\sim 11-12h$, r-TPA $\sim 20\%$
 - **UW-mRS et mRS 0-2 à 90 jrs (15 vs 9%) idem**, \uparrow mRS 0-3 à 90 jrs (30 vs 20%), mortalité, sICH et CD idem, plus d'ICH

Évolution à 1 an



- mRS médian 5 vs 6
- mRS 0-2 22-24% vs 6%
- mRS 0-3 34-37 vs 17-18%
- Meilleure qualité de vie
 - Cognitif
 - Santé mentale
 - Mobilité
- Diminution mRS 5-6
- ↑ HIC, mais pas sICH
- Pas plus de CD...
- Enjeux organisationnels locaux?
 - MT sous AG?
 - Admissions aux SI? CD?

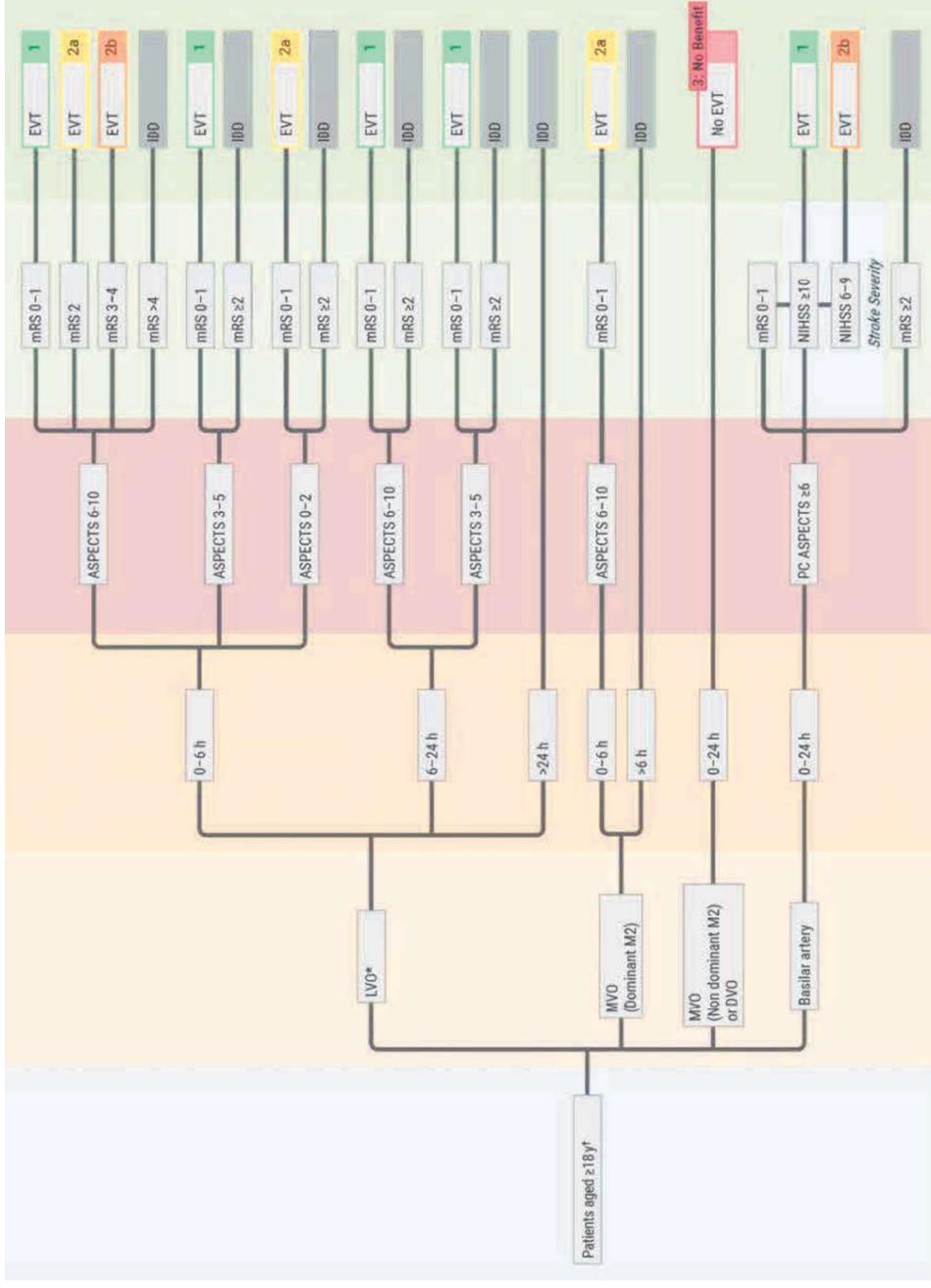
AVC circulation postérieure: thrombectomie

- **BEST Lancet Neurol 2020** CN 288/344 pts, occlusion VB, LKW < 8h
 - Occlusion basilaire ~ 90%, NIHSS ~ 30, r-TPA ~ 30%, délai revasc ~ 6.7h, TICI 2b-3 71%, **cross-over 22%**
 - mRS 0-3 à 90 jrs idem (42 vs 32%, PP 44 vs 25%)
 - (interrompte cross-over et pauvre recrutement)
- **BASICS NEJM 2021** EUBR 300/750 pts, occlusion basilaire, <85 ans, NIHSS > 10, délai < 6h; élargi après 91 pts: CI r-TPA, pas de limite âge/NIHSS
 - NIHSS 22, r-TPA ~ 80%, délai tx ~ 4.4h, TICI 2b-3 72%
 - mRS 0-3 à 90 jrs idem (44 vs 38%), plus de sICH 4.5 vs 0,7%, mortalité (59 vs 63%) idem
 - (modification de taille d'échantillon – publications LVO 2015 et pauvre recrutement)
- **BAOCHE NEJM 2022** CN 217/318pts, occlusion basilaire, délai tx 6-24h, NIHSS > 6, PC-ASPECTS > 5
 - Tous occlusion basilaire, NIHSS 20, PC-ASPECTS 8, r-TPA ~ 15%, délai revasc ~ 13h, TICI 2b-3 88%
 - ↑↑ mRS 0-3 à 90 jrs (46 vs 24%), sICH (6 vs 1%) et mortalité idem
 - (**interrompte pour efficacité à 1^{ère} analyse intérimaire**, modification outcome primaire)
- **ATTENTION NEJM 2022** CN 340 pts, occlusion VB, délai tx < 12h, NIHSS ≥ 10, PC-ASPECTS > 5
 - Occlusion basilaire ~90%, NIHSS 24,PC-ASPECT 9-10, r-TPA ~ 30%, délai revasc ~ 6.9h, TICI 2b-3 93%
 - ↑↑ mRS 0-3 à 90 jrs (46 vs 23%), ↓ **mortalité (37 vs 55%)**, sICH 5 vs 0%
 - ↑↑ mRS 0-3 à 1 an 46 vs 19%, ↑↑ **mRS 0-1 à 1 an (28% vs 8%)**, ↓ **mortalité (46 vs 64%)** (JAMA Neurol 2024)

En résumé...

- Considération de thrombolyse
 - AVC invalidant (NIHSS 0-5)
 - Délai début des symptômes < 4.5h
 - Délai début des symptômes 4.5-24h si imagerie neurovasc avancée favorable
- Considération de thrombectomie
 - AVC invalidant (NIHSS ≥ 6)
 - Occlusion intracrânienne proximale prouvée et traitable
 - ASPECTS ≥ 6
 - ASPECTS < 6 considérés au cas par cas...
 - Délai début des symptômes < 6h
 - Délai début des symptômes 6-24h avec imagerie neurovasc avancée favorable

Algorithme décisionnel pour thrombectomie



Thrombectomie sous AG

Effect of general anaesthesia on functional outcome in patients with anterior circulation ischaemic stroke having endovascular thrombectomy versus standard care: a meta-analysis of individual patient data

Bruce C V Campbell, Wim H van Zwam, Mayank Goyal, Bijoy K Menon, Diederik W J Dippel, Andrew M Demchuk, Serge Bracad, Philip White, Antoni Dávalos, Charles B L M Majoie, Aad van der Lugt, Gary A Ford, Natalia Pérez de la Ossa, Michael Kelly, Romain Bourcier, Geoffrey A Donnan, Yvo B W E M Roos, Oh Young Bang, Raul G Nogueira, Thomas G Devlin, Lucie A van den Berg, Frédéric Clarençon, Paul Burns, Jeffrey Carpenter, Olvert A Berkhemer, Dileep R Yavagal, Vitor Mendes Pereira, Xavier Ducrocq, Anand Dixit, Helena Quesada, Jonathan Epstein, Stephen M Davis, Olav Jansen, Marta Rubiera, Xabier Urra, Emilien Micard, Hester F Lingsma, Olivier Naggara, Scott Brown, Francis Guillemin*, Keith W Muir*, Robert J van Oostenbrugge*, Jeffrey L Saver*, Tudor G Jovin*, Michael D Hill*, Peter J Mitchell*, for the HERMES collaborators



Interpretation Worse outcomes after endovascular thrombectomy were associated with GA, after adjustment for baseline prognostic variables. These data support avoidance of GA whenever possible. The procedure did, however remain effective versus standard care in patients treated under GA, indicating that treatment should not be withheld in those who require anaesthesia for medical reasons.

Thrombectomie: modalité anesthésique

- Méta-analyse sur données individuelles de 7 RCT
 - 1764 patients, 131 centres
 - **Études évaluant thrombectomie vs traitement standard**
 - **Analyse rétrospective**
 - AG vs pas d'AG (= SC ou AL seule)
 - **Pas de protocole standardisé pour AG**
- **BIAIS DE SÉLECTION!**
 - AG 30%
 - AG – plus jeunes, plus de rtPA IV, mois de DLP/Db
 - SC – meilleur ASPECT (8 vs 7)
 - Délai début sx ad reperfusion = idem
 - Délai radomisation ad reperfusion = SC 85 vs AG 105 min
- AG n'élimine pas bénéfices de thrombectomie
- SC shift vers bon outcome (mRS ≤ 2) OR 2,62 ($p < 0.001$)

No GA vs GA*		
	Effect size OR (95%CI)	p value
Primary outcome		
Functional outcome at 90 days (mRS)†
Covariate adjusted common odds ratio	1.53 (1.14–2.04)	0.0044
Propensity-score stratification common odds ratio	1.44 (1.08–1.92)	0.012
Secondary outcomes		
Independent functional outcome (mRS 0–2)	1.65 (1.14–2.38)	0.0078
Excellent functional outcome (mRS 0–1)	1.68 (1.12–2.52)	0.013
Early neurological improvement (NIHSS reduction ≥ 8 points or reaching 0–1 at 24 h)‡	1.75 (1.23–2.48)	0.0020
Safety		
Death within 90 days	0.71 (0.44–1.14)	0.15
Symptomatic intracerebral haemorrhage§	0.95 (0.41–2.19)	0.90
Parenchymal haematoma	0.97 (0.60–1.58)	0.90

Thrombectomie circulation antérieure: SC vs AG

Étude	N	Maintient	SC → AG (%)	Délai (min) HOP-PCT NRI-PCT PCT-reperf	TICI 2b-3 (%)	Δ NIHSS à 24h	mRS (0-2) à 90 jrs (%)	Δ taille AVC (mL)	Note
SIESTA Schönenberger et al. DE (JAMA 2018)	150	SC = PPF/rémi AG = PPF/rémi	14%	66 vs 76 ----- 109 vs 90	81 vs 89	-3.8 vs -3.2	18 vs 37	N/A	AG plus de complications (HypoTo 33 vs 9%, pneumo 14 vs 4%)
AnStroke Hendén et al. SE (Stroke 2017)	90	SC = rémi TCI AG = sévo/rémi TCI	16%	----- 25 vs 34 74 vs 55	89 vs 91	-8 vs -9	40 vs 42	N/A	AG désavantagé (NIHSS, T carot, G) AG plus hypoTA > 20% (PAM 95 vs 91)
GOLIATH Simonsen et al. DK (JAMA Neurol 2017)	128	AG = PPF/fenta SC = PPF/rémi	6%	----- 15 vs 24 29 vs 34	60 vs 77	-7 vs -10	52 vs 67	19 vs 8	AG plus hypoTA (PAM 102 vs 90)
CANVAS Sun et al. CN (J Neurosurg Anesthesiol 2020)	43	SC = PPF/suf TCI AG = PPF/rémi TCI	20%	----- 15 vs 27 78 vs 102	65 vs 95	N/A	50 vs 55	N/A	AG plus hypoTA > 20% (PAM 108 vs 89) LMA

Thrombectomie circulation antérieure: SC vs AG

Étude	N	Maintient	SC → AG (%)	Délai (min) HOP-PCT NRI-PCT PCT-reperf	TICI 2b-3 (%)	Δ NIHSS à 24h	mRS (0-2) à 90 jrs (%)	Δ taille AVC (mL)	Note
Ren et al. CN (Front Neurol 2020)	90	SC = PPF/dex + F/M AG = PPF/rémi/dex	10%	----- 11 vs 11 39 vs 47	86 vs 88	-5 vs -5	?idem? Pas formellement rapporté!	N/A	Pneumonie 5 vs 21 mRS médian idem
GASS Maurice et al. FR (Anesthesiology 2022)	351 multi	SC = rémi TCI AG = PPF/rémi TCI TAs 140-185 SpO2 > 96	4%	60 vs 69 ----- 59 vs 51	75 vs 85	-5 vs -5	36 vs 40	N/A	AG plus hypoTA et hyperTA
AMETIS Chabanne et al. FR (JAMA Neurol 2023)	273 multi	Pragmatique TAs 140-180 SpO2 > 94 EtCO2 30-35 (AG)	11%	----- 9 vs 11 41 vs 35	78 vs 85	-7 vs -7	39 vs 33	N/A	mRS 0-2 à 90 jrs et absence de complications 36 vs 28
SEGA Chen et al. US (JAMA Neurol 2025)	260 multi	Pragmatique TAs 140-180 EtCO2 35-40 (AG)	14%	69 vs 78 13 vs 17 43 vs 41	95 vs 97	N/A	39 vs 48 Prob post de bénéfice 89%	N/A	Amélioration mRS OR 1.22 (0.79-1.87) Prob post de bénéfice (81%)

Thrombectomie sous AG

RESEARCH ARTICLE

General Anesthesia Compared With Non-GA in Endovascular Thrombectomy for Ischemic Stroke

A Systematic Review and Meta-analysis of Randomized Controlled Trial¹

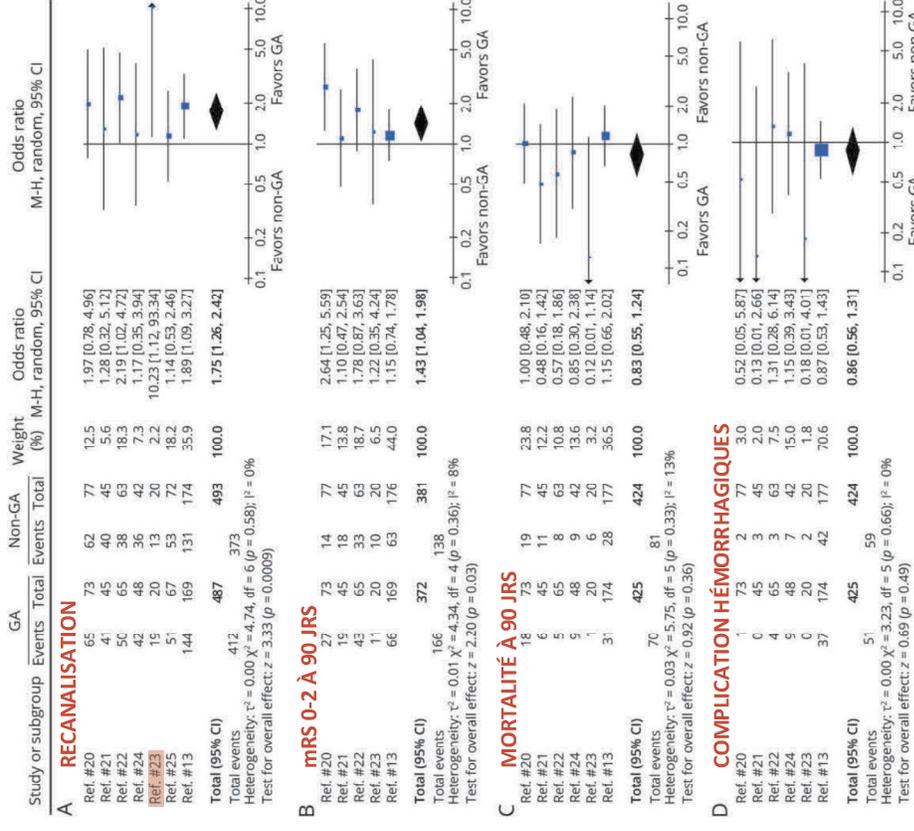
Douglas Campbell, BM, Elise Butler, MB ChB, Ruby Blythe Campbell, Jess Ho, MB ChB, and P. Alan Barber, MBChB, PhD, FRACP

Neurology® 2023;100:e1655-e1663. doi:10.1212/WNL.000000000000207066

atcampbell@camo.govt.nz

- ↑recanalisation de 9%
- ↑mRS 0-2 à 90 jrs de 8,4% (NNT 12)
- Limites de RCT non-centrée sur cette comparaison (MA données individuelles, ajustement)
- Biais de sélection inhérent
- Délai de traitement
- Gestion AG/HD péri-thrombectomie
- Présence d'anesth/intensiviste (SC/AG)

MMD 6513 – Anesthésie et système nerveux
Antoine Halwagi, MD FRCP



Pooled estimates were performed only if trials reported the endpoint and in the correct format. GA = general anesthesia.

Thrombectomie circulation postérieure: SC vs AG

Étude	N	Maintient	SC → AG (%)	Délai (min) HOP-PCT NRI-PCT PCT-reperf	TICI 2b-3 (%)	Δ NIHSS à 24h	mRS (0-2) à 90 jrs (%)	Δ taille AVC (mL)	Note
Hu et al. CN (Am J Transl Res 2021)	139 mono	SC = PPF/fenta AG = PPF/rémi	3%	143 vs 130 (procédure)	76 vs 73	N/A	N/A	29 vs 12	Étude non-infériorité mRS (2 vs 3) idem
CANVAS II Liang et al. CN (JAMA Neurol 2022)	93 mono	SC = PPF/rémi AG = PPF/rémi BIS >70 vs 40-60	30% (!!!) 	151 vs 185 15 vs 20 81 vs 99	77 vs 95	NS	55 vs 49 (ITT)	N/A	Peu rTPa (11-16%) Plus d'hypoTA en AG (23 vs 7%) Objectif TAsyst 140-180mmHg non atteint

Thrombectomie: RCT SC vs AG

- Limites des données disponibles
 - Maintenant quelques multicentriques (3)
 - (validité externe, variation performance – délai, succès...)
 - Anesthésistes (ou neurointensiviste) pour toute procédures (SC ou AG)
 - Plan anesthésiques variés (agent, monitoring, intubation vs LMA)
 - **Protocole standardisé avec cibles précises – éviter hypotension**
- AG semble sécuritaire... bénéfique?
 - Effet « neuroprotecteur » direct?
 - Procédure de meilleure qualité?
 - Effet différentiel selon agent utilisé (PPF vs volatiles)? Pas de données convaincantes
- Délais – AG = 10 minutes de plus mais délai sx à reperfusion similaire
 - Équipes rodées avec protocoles et objectifs clairs

Thrombectomie: modalité anesthésique

Modification des lignes directrices AHA/ASA

Privilégier sédation consciente plutôt qu'anesthésie générale (Stroke 2015)



Individualiser de technique anesthésique en fonction des facteurs de risques du patient, de considérations techniques de la procédure et d'autres caractéristiques cliniques (Stroke 2019)



Recommendations for Endovascular Techniques (Continued)		
COR	LOE	Recommendations
1	B-R	3. In patients with AIS undergoing EVT, either general anesthesia or procedural sedation are recommended to facilitate EVT. ⁷⁻⁹

Thrombectomie: modalité anesthésique

CA



Mise à jour 2025



5.4.2 Sedation for Endovascular Interventions

- i. For endovascular interventions, procedural sedation is generally preferred over intubation and general anesthesia for most patients undergoing EVT [Strong recommendation; Moderate quality of evidence]
- ii. General anesthesia is appropriate if medically indicated (e.g., for airway compromise, respiratory distress, depressed level of consciousness, severe agitation, or other indication potentially impairing the technical ability to perform the procedure, as determined by the treating physician). General anesthesia may also be considered when technical complexity is anticipated during the stroke intervention. In such cases, excessive and prolonged hypotension and time delays should be avoided [Strong recommendation; Moderate quality of evidence].
- iii. A process should be in place at EVT enabled centres to activate notification of Anesthesiology without delay when deemed necessary for patients who meet criteria for EVT [Strong recommendation; Moderate quality of evidence].

Anesthésie pour thrombectomie

- Modalité selon le patient: individualisation
- Situation urgente!!
- **Fenêtre de traitement élargie ≠ ralentir cadence du traitement**
- Information disponible limitée...
 - « Baseline ECG, troponin, creatinine, platelet count, coagulation studies (...) should not delay »

Rôle de l'anesthésiste

⚠ **TIME IS BRAIN** ⚠

NHISS

Imagerie

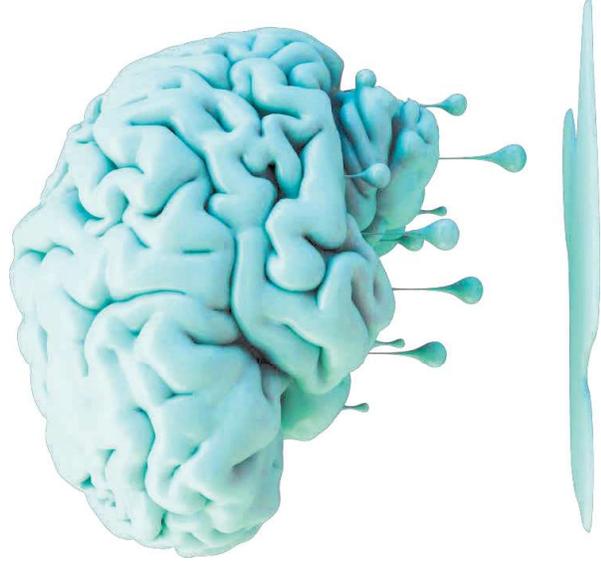
Thrombolyse

Thrombectomie

Door-to-needle

Door-to-puncture

GO! GO! GO!



Anesthésie pour thrombectomie

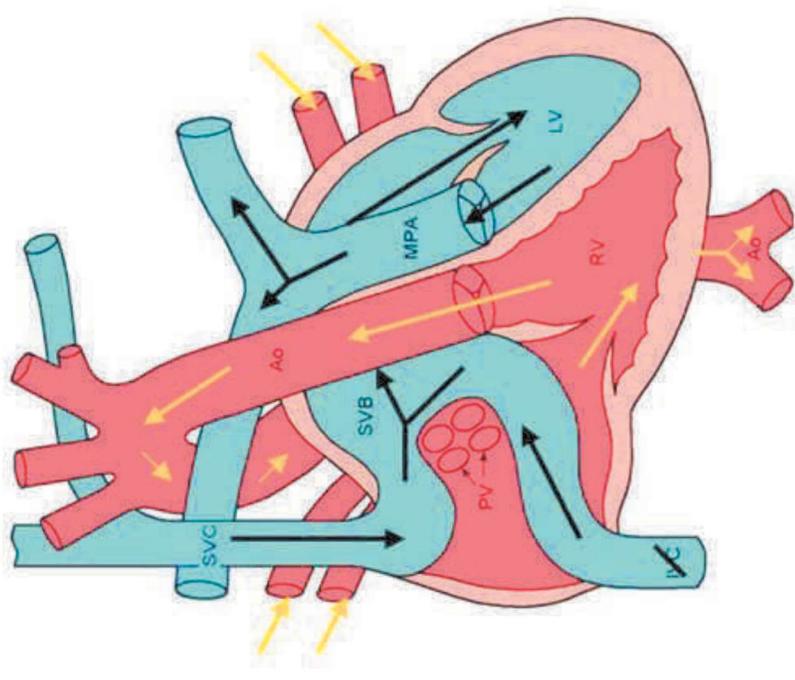
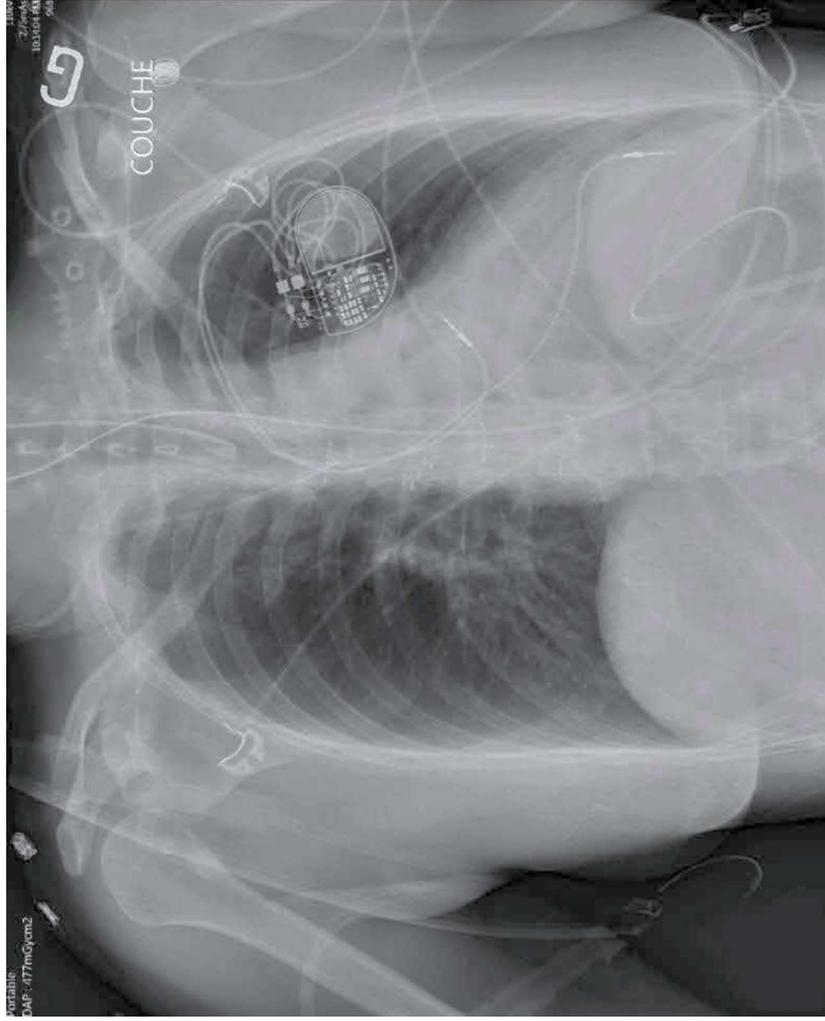
- Placé devant le fait accompli...
 - Procédure déjà en cours
 - Information limitée
 - Altération d'état de conscience
 - Hypoxémie, obstruction VRS, vomissements
 - Agitation
 - Accès au patient limité
- Terrain hostile (hors bloc)





TGV → correction de Mustard

MMD 6513 – Anesthésie et système nerveux
Antoine Halwagi, MD FRCPC



A-B-C en AVC: AIRWAY

- Cause possibles d'intubation / prise en charge airway
 - Cause intracrânienne – AEC (CGS \leq 8), dysfonction tronc (gestion OP, protection VRS), œdème malin, HTIC/engagement, status epilepticus et agitation extrême
 - Cause extracrânienne – hypoxémie (pneumonie, OAP), apnée/hypopnée (exacerbée par physionomie, sédation), instabilité HD (cardiopathie, choc)
 - Contextuel – pré-procédure (immobilité), agitation ou complication péri-procédurale, évolution clinique anticipée, sécurisation pré-transport...

A-B-C en AVC: AIRWAY

- Sédation procédurale vs **estomac plein** - pistes de réflexion
- Recommandations de jeûne s'appliquent aux procédures électives^{1,2,3}
 - En situation urgente, évaluer le risques d'inhalation selon FR
 - Procédure « urgente » en est un!
- Consensus de sédation procédurale en salle d'urgence⁴

Level B recommendations. Do not delay procedural sedation in adults or pediatrics in the ED based on fasting time. Preprocedural fasting for any duration has not demonstrated a reduction in the risk of emesis or aspiration when administering procedural sedation and analgesia.

- Sédation consciente en thrombectomie prévalente x des années

1- Procedural sedation – Dobson et al. Can J Anesth 2018

2- Guide d'exercice de l'anesthésie 2025 – Can J Anesth 2025

3- Lignes directrices de l'ASA 2017 – Anesthesiology 2017

4- Politique de l'ACEP – Ann Emerg Med 2014

ABSENCE DE JEÛNE
≠
CONTRE-INDICATION
ABSOLUE À SÉDATION
PROCÉDURALE

A-B-C en AVC: BREATHING

- Éviter hypoxémie
 - Viser SpO₂ > 97%, O₂ PRN
- ~~O₂ non indiqué si non hypoxémique?~~
- ~~Effets délétères de l'hyperoxie?~~
- **NOUVELLES RECOMMANDATIONS**
- **EN CONTEXTE DE THROMBECTOMIE**

Recommendations for Airway, Breathing, and Oxygenation Referenced studies that support the recommendations are summarized in the online data supplement .		
COR	LOE	Recommendations
1	C-LD	1. In patients with acute stroke and decreased consciousness or bulbar dysfunction, airway support and ventilatory assistance are recommended as needed to provide airway maintenance, protection and adequate ventilation and oxygenation. ¹⁻³
1	C-LD	2. In patients with AIS with hypoxia, supplemental oxygen should be provided to maintain oxygen saturation (SpO ₂) >94%. ^{4,5}
2b	B-R	3. In patients with AIS within 6 hours from onset, NIHSS score 10 to 20, CT ASPECTS of ≥6, and anterior circulation LVO (M1 or carotid terminus) with planned EVT (with or without IVT) normobaric hyperoxia (NBO) before EVT may be reasonable to improve functional outcomes at 90 days. ⁶⁻⁹
2b	B-NR	4. In patients with AIS due to arterial air embolism, hyperbaric oxygen (HBO) may be reasonable to improve clinical outcome. ¹⁰
3: No benefit	B-R	5. In patients with AIS without hypoxia who are ineligible for EVT, supplemental oxygen is not recommended to improve functional outcomes. ¹¹⁻¹⁶
3: No benefit	B-R	6. In patients with AIS, not associated with air embolism, HBO is not recommended to improve functional outcomes. ¹⁷

A-B-C en AVC: BREATHING

- RCT multicentrique CN
 - 282 patients, thrombectomie
 - LVO circulation antérieure (CI, M1)
 - LKW < 6h, NIHSS 10-20
 - ASPECTS ≥ 6

• FiO2 100% (VM 10 LPM ou via TET) vs sham (VM 1 LPM ou 30%) x 4h

- 20% AG

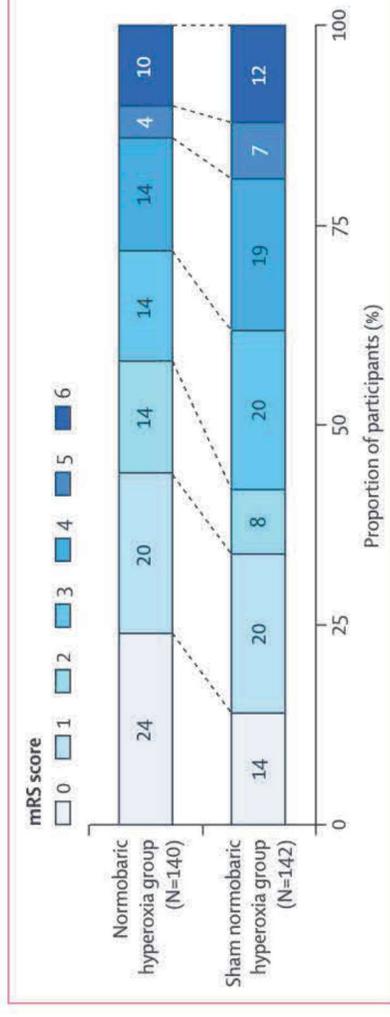
• mRS médian 2 vs 3 à

(OR 1.65 IC 1.09-2.5)

• ↓ taille infarctus, NIHSS à 24h

Normobaric hyperoxia combined with endovascular treatment for acute ischaemic stroke in China (OPENS-2 trial): a multicentre, randomised, single-blind, sham-controlled trial

Weili Li*, Jing Lan*, Ming Wei*, Lan Liu, Chengbei Hou, Zhifeng Qi, Chuanhui Li, Liqun Jiao, Qi Yang, Wenhao Chen, Shuling Liu, Xincan Yue, Qinglin Dong, Haicheng Yuan, Zongren Gao, Xiangbin Wu, Changming Wen, Tong Li, Changchun Jiang, Di Li, Zuquan Chen, Junfeng Shi, Wanchao Shi, Jinglin Yuan, Yijie Qin, Binglong Li, Marc Fisher, Wuwei Feng, Ke Jian Liu, Xuming Ji, on behalf of the OPENS-2 Investigators†



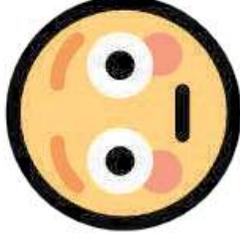
A-B-C en AVC: BREATHING

- Normoventilation (PaCO₂ 35-45 mmHg)
- Hyperventilation → réduction du DSC
- Hypoventilation → phénomène de vol?
- Impact différent pré vs post-reperfusion?
- En absence de monitoring avancé ou d'engagement (bridge)

→ pas de folies!

A-B-C en AVC: CIRCULATION

- Éviter hypoTA, hypovolémie et hypoperfusion
- Éviter réduction rapide ou excessive de TA



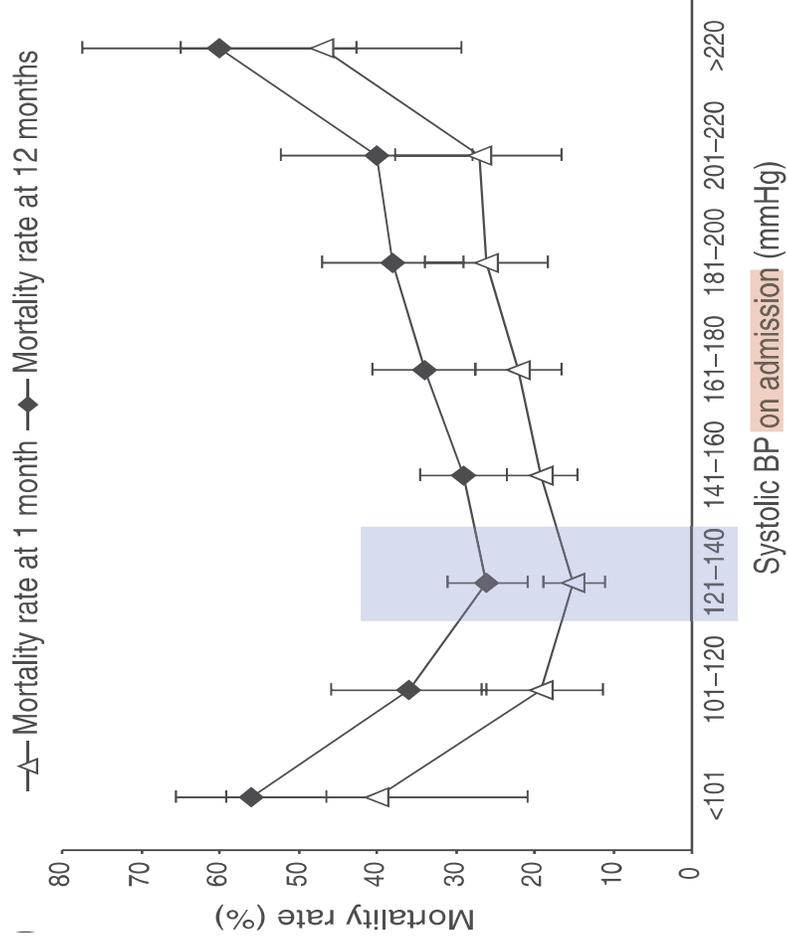
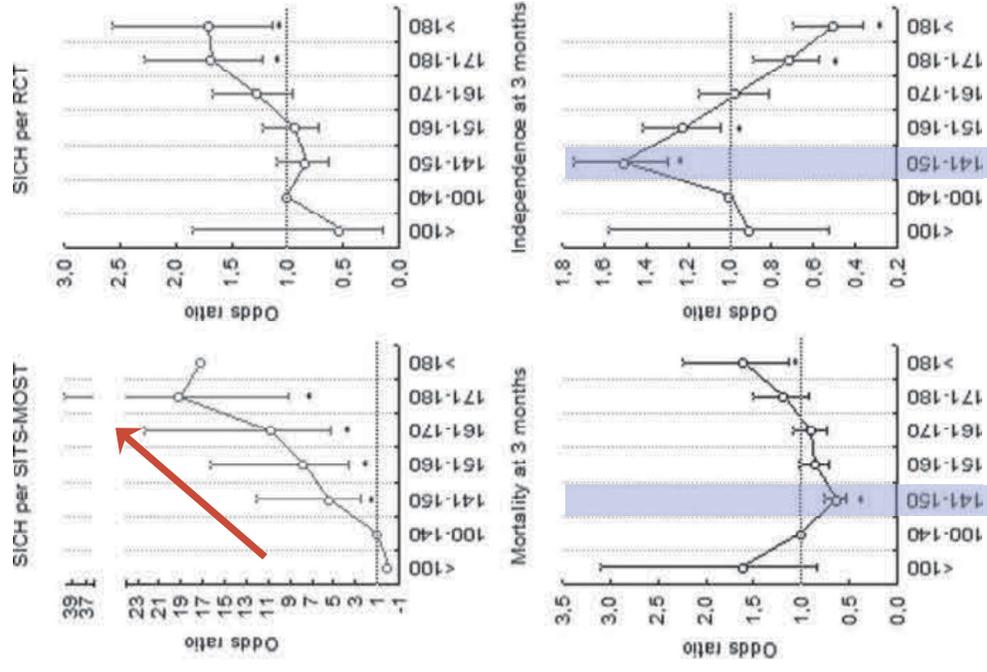
- PA marqueur d'ischémie-reperfusion ou cible thérapeutique?

A-B-C en AVC: CIRCULATION

- Quoi viser – PAsyst, PAdia, PAM?
- Comment mesurer – PNI vs PI, MS plégique ou non?
- Quand la contrôler – hyper-aigu, pré-intervention, post-intervention?
- Degré de collatéralisation et/ou reperfusion? Microthrombose veineuse post-capillaire?
- Étendue de l'AVC établi?
- Comorbidités?
- Ajustement en fonction de neuromonitoring avancé?
- Contrôle pendant combien de temps?
- Effet de variabilité de TA...
- Effet différentiel selon l'agent?

U

PA en AVC: la fameuse courbe en U



CIBLES DE PRESSION ARTÉRIELLE EN AVC AIGU

ABSENCE DE TRAITEMENT SPÉCIFIQUE

≥ 220 / 120 → ↓ 15-25% au cours des 1^{ers} 24-72^{1,2}
 < 220 / 120 → pas de Rx^{1,2}

ÉVITER HYPOTENSION – THÉRAPIE HYPERTENSIVE CHEZ PATIENTS SÉLECTIONNÉS?³

PRÉ-THROMBOLYSE

< 185 / 110^{1,2}

POST-THROMBOLYSE

< 180 / 105 x 24h^{1,2,4}

PRÉ-THROMBECTOMIE

< 185 / 110¹

DURANT THROMBECTOMIE

≤ 180/105¹
PA syst entre 140-180^{5,6}
 PAM 70-90 ?⁷

POST-THROMBECTOMIE

TOUS: < 180 / 105 x 24h¹
 TIC1 ≥ 2b: PA syst > 130-140^{8,9}

1- Prabhakaran et al. Stroke 2026 (AHA/ASA)

4- Anderson et al. Lancet 2019 (SETIN-HYPERTENSION)

7- Espelund et al. Stroke Vasc Interv Neurol 2024

2- Heran et al. Can J Neurol Sci 2024 (CAN)

5- Sharma et al. J Neurosurg Anesthesiol 2020 (SNACC)

8- Yang et al. Lancet 2022 (ENCHANTED2/MT)

3- Bang et al. Neurology 2019 (SETIN-HYPERTENSION)

6- Chen et al. Stroke 2023 (INDIVIDUATE)

9- Nam et al. JAMA 2023 (OPTIMAL BP)

Durant thrombectomie: cibles individualisées

- **Espelund et al. Stroke Vasc Interv Neurol 2024 DK (GOLIATH)**
 - 60 pts LVO + thrombectomie sous AG (étude faisabilité)
 - PAM +/- 10% pré-intervention vs PAM 70-90 mmHg (PI, LIDCOrapid)
 - mRS 0-2 à 90 jrs idem (70 vs 53%)
 - **Pas faisable > 50% du temps à l'extérieur de cible prévue malgré expertise**
- **INDIVIDUATE Chen et al. Stroke 2023 DE (SIESTA)**
 - 250 pts LVO + thrombectomie sous sédation (étude exploratoire)
 - TAsyst +/- 10% pré-intervention (urgence) vs TAsyst 140-180 mmHg
 - Écart de TAsyst moyen rapproché (155-175 vs 140-180 mmHg)
 - mRS 0-2 à 90 jrs idem (25 vs 24%), sICH et mortalité idem
- **DETERMINE Maier et al. (non-publié, ESOC 2025) FR**
 - 433 pts LVO + thrombectomie
 - PAM ~ 10% pré-EVT vs TAs 140-180 mmHg (NA) ad reperfusion ou fermeture artère fémorale
 - mRS 0-2 à 90 jrs idem (~44 vs 48%)

Post-thrombectomie efficace

- 5 études complétées ($TA_{syst} < 140$ mmHg vs $TA_{syst} < 180$ mmHg)
 - **BP-TARGET** Lancet Neurol 2021 FR négative
 - **ENCHANTED2/MT** Lancet 2022 CN **DANGER**
 - **OPTIMAL BP** JAMA 2023 KR **DANGER**
 - **BEST-II** JAMA 2023 US probablement futile
 - **DETECT** Stroke Vasc Interv Neurol 2024 CA pas faisable
 - **IDENTIFY** Lancet Reg Health West Pac 2025 CN négative
- **≥ 3 en cours...**
 - **CRISIS 1** NCT04775147 CN TAs <120 vs <140 post MT $TICI \geq 2b$
 - (205 pts enrôlés, terminée 01/2023, pas publiée)
 - **HOPE** NCT04892511 ES TAs 100-140 ($TICI$ 2c/3) 140-160 ($TICI$ 2b) vs $<180/105$
 - (814 pts enrôlés, fin prévue 09/2024, pas publiée)
 - **CLEVER** NCT05175547 US TAs 90-120 vs 90-160 mmHg ($mTICI \geq 2c$)
 - (80 pts enrôlés, terminée 02/2024, pas publiée)

« results strongly contribute to a dampened enthusiasm for spending limited research resources on a future, large, pivotal trial »

Post-thrombectomie efficace

- **BP-TARGET Lancet Neurol 2021** FR 324 pts LVO TICI $\geq 2b \rightarrow$ TAsyst 100-129 vs TAsyst 130-185 en < 1h x 24h (PNI, nicardipine, TAsyst moy de base ~ 154 mmHg)
 - TAsyst moyenne dans 1^{er} 24h: 128 vs 138 mmHg \rightarrow ICH idem, hypoTA idem
 - 30% des patients avaient TAsyst < 130 mmHg post reperfusion
 - Difficulté d'effectuer ces études: TAsyst dans les cibles 61-67% du temps, ~3h pour atteindre cible intensive
- **ENCHANTED2/MT Lancet 2022** CN 821/2257 pts LVO TICI $\geq 2b \rightarrow$ TAsyst 100-120 vs 140-180 en < 1h x 72h (PNI, MS parététique, urapidil, TAsyst moy de base ~ 159 mmHg)
 - TAsyst moyenne à 1h et 24h: 125 et 143 vs 139 mmHg (D = 18 mmHg)
 - **DANGER**: pire mRS shift (OR 1.37), augmentation mRS 3-6 53 vs 39%, sICH 6% idem
 - (interrompu pour motif de sécurité)
- **OPTIMAL BP JAMA 2023** KR 306/668 pts LVO TICI $\geq 2b \rightarrow$ TAsyst < 140 vs 140-180 en < 1h x 24h (PNI, MS non-parététique, nicardipine, TAsyst moy base ~ 155 mmHg)
 - TAsyst moyenne à 24h: 128 vs 139 mmHg (D = 10 mmHg)
 - **DANGER**: pire évolution (mRS 0-2 39 vs 54%), plus d'hypoTA < 100 (30 vs 17%), plus d'œdème malin (OR 6), sICH (9%) et mortalité idem
 - (interrompu suivant résultats ENCHANTED2/MT et futilité de rejeter hypothèse nulle) (pire si réduction de PA par Rx plutôt que spontanée)
- **BEST-II JAMA 2023** US 120 pts LVO TICI $\geq 2b \rightarrow$ TAsyst < 140 vs < 160 vs < 180 en < 1h x 24h (PNI, nicardipine, TAsyst moy base ~ 148 mmHg)
 - TAsyst moyenne à 24h: 122 vs 130 vs 129 mmHg, utilisation d'antiHTA IV à 24h: 73 vs 55 vs 25%
 - Étude de futilité: conclusion de futilité non-atteinte mais peu probable d'identifier un bénéfice de viser des TAsyst plus basses... (taille d'infarctus à 36h, UW-mRS)
 - « results strongly contribute to a dampened enthusiasm for spending limited research resources on a future, large, pivotal trial »
- **DETECT Stroke Vasc Interv Neurol 2024** CA 30 pts LVO TICI $\geq 2b \rightarrow$ TAsyst < 140 vs < 180 x 48h (labetalol/hydralazine, TAsyst moyenne base ~ 159 mmHg)
 - TAsyst moyenne à 48h: 131 vs 139 mmHg,
 - Étude de faisabilité: 69% des pts exclus car TAsyst < 150, 48% du temps TAsyst < 140 dans groupe standard, HTA moins prévalente qu'attendu post-reperfusion, recrutement lent
- **IDENTIFY Lancet Reg Health West Pac 2025** CN 383/600 pts LVO TICI $\geq 2b \rightarrow$ TAsyst < 130 vs < 180 x 24h (urapidil et autres, PNI, TAsyst moyenne base ~ 151 mmHg)
 - TA moyenne à 24h
 - Évolution défavorable (mRS 3-6) à 90jrs idem, sICH, œdème cérébral, mortalité idem
 - (interrompu suivant résultats ENCHANTED2/MT et OPTIMAL BP)

A-B-C en AVC: CIRCULATION

Considérer la cause d'hypotension...

- Iatrogène (anticiper avec vasopresseurs)
- Hypovolémie
- Dépression myocardique
- Arythmie
- Saignement externe
- Saignement rétro-péritonéal
- Dissection aortique

An intra-arterial cannula is useful if it can be placed without delaying the procedure.
(SNACC 2014)

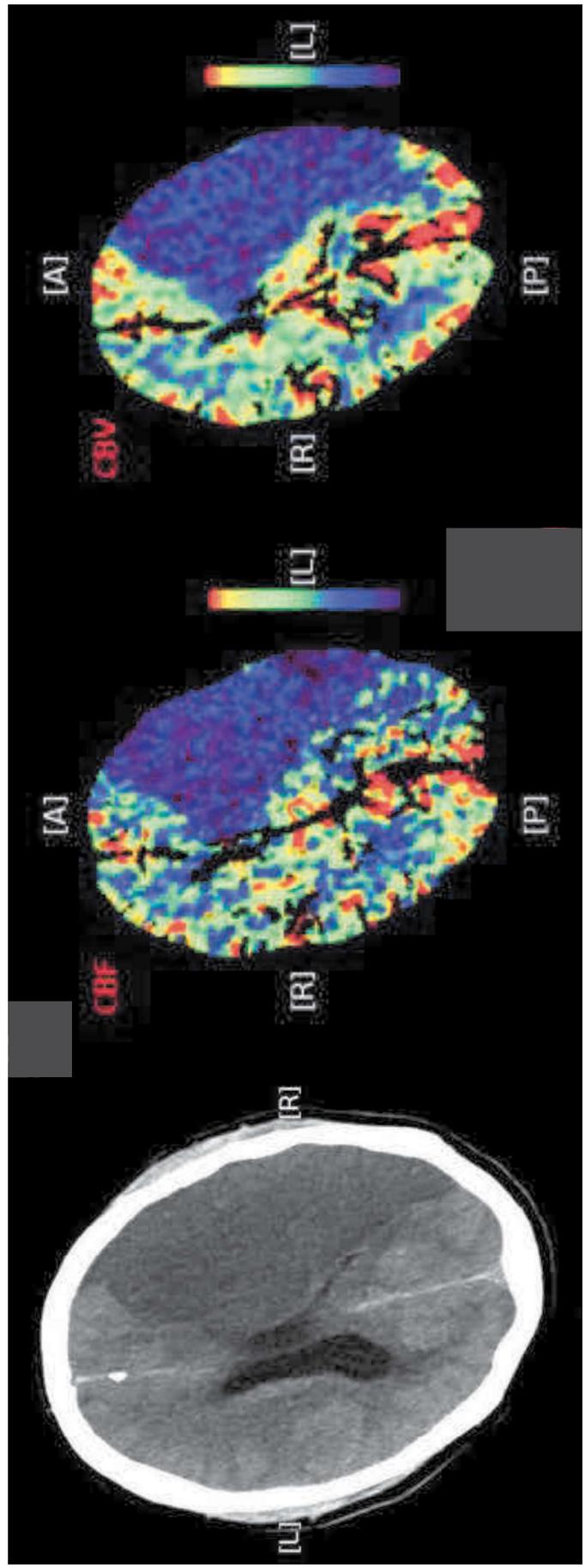
Thrombectomie – en résumé

- Situation d'urgence, hors bloc, peu de temps pour évaluer/optimiser
- Modalité anesthésique selon le contexte
- Intubation si indiqué par état clinique
- Normoventilation (pas d'hyper ou hypocapnie)
- Éviter hypoxémie (SpO2 > 94%), considérer hyperoxygénation en EVT
- Gestion de pression artérielle – éviter hypoTA, TAsyst 140-180 mmHg
- Normothermie
- Normoglycémie

Endovascular thrombectomy is a time-limited emergency procedure. Getting the procedure started (femoral arterial puncture for angiographic access) as soon as possible is a cardinal management goal. What is unknowable is when rapid workflow becomes unsafe. (Hindman et al. Anesth Analg 2016)

« Malignant MCA infarction »

AVC malin



Craniectomie décompressive en AVC « malin »

- **Identification précoce des patients à risque**
- Détérioration habituellement dans les 72-96 hrs post-AVC
(ad 10 jours!)
- Seuil pour intervention (craniectomie décompressive) incertain
 - AEC
 - Imagerie: zone infarctie
 - Précoce ≤ 6 h
 - Extensive > 50-67% territoire ACM, > 150 mL
 - Effet de masse = DLM
- **Approche proactive vs réactive?**

AHA/ASA Scientific Statement

Recommendations for the Management of Cerebral and Cerebellar Infarction With Swelling A Statement for Healthcare Professionals From the American Heart Association/American Stroke Association

*The American Academy of Neurology affirms the value of this statement as an educational tool for neurologists.
Endorsed by the American Association of Neurological Surgeons and Congress of Neurological Surgeons
Endorsed by the Neurocritical Care Society*

Stroke. 2014;45:1222–1238.

Evidence-Based Guidelines for the Management of Large Hemispheric Infarction

A Statement for Health Care Professionals from the Neurocritical Care Society and the German Society for Neuro-Intensive Care and Emergency Medicine

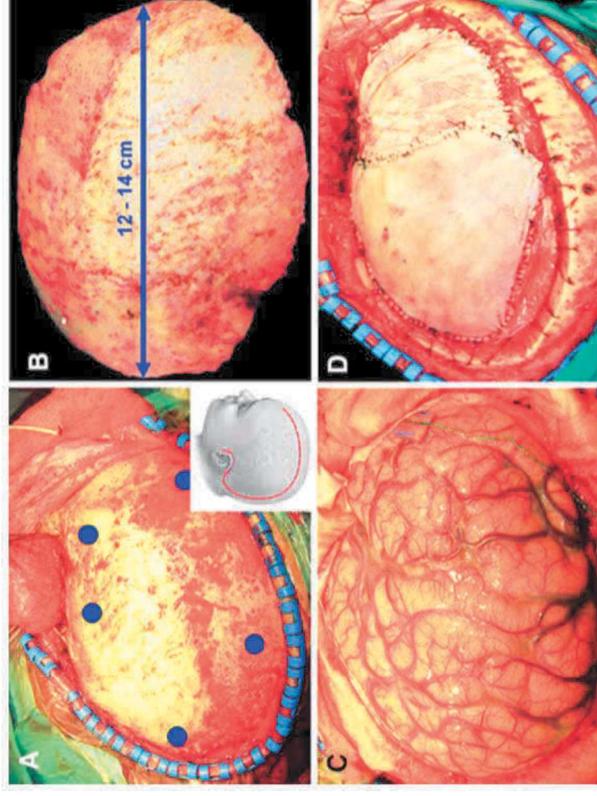
Neurocrit Care (2015) 22:146–164

Craniectomie décompressive en AVC malin

- Enjeux **éthiques**: degré d'handicap acceptable pour le patient
 - Aucun impact sur l'AVC établi
 - Diminution de mortalité
 - Outcome probable mRS 2-5

• 5 principales RCT ~2007-2014

- Decimal
- Hamlet
- Destiny
- Analyse groupée
- Destiny II
- **Méta-analyse de données individuelles**



Méta-analyses: impact de la craniectomie

Vahedi et al. Lancet Neurol 2007

Reinink et al. JAMA Neurol 2020

MRS À 1 AN



Survie NNT = 2.4
mRS ≤ 4 NNT = 2.6
mRS ≤ 3 NNT = 4.5

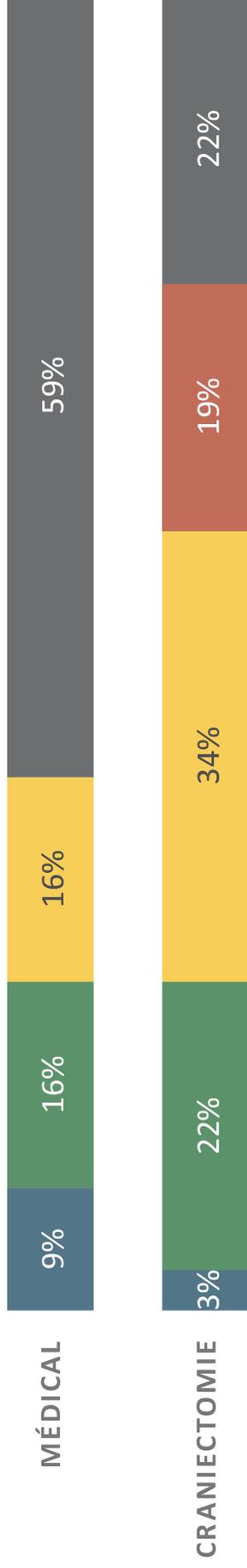
Outcome	No./total No. (%) of patients		RD	Crude OR (95% CI)	P value	Adjusted OR (95% CI)	P value
	Surgery population	Medical population					
Primary outcome							
mRS score ≤3 at 1 y	87/234 (37)	37/254 (15)	(21)	3.23 (1.75-5.94)	<.001	2.95 (1.55-5.60)	.001
Secondary outcomes							
mRS score ≤2 at 1 y	39/234 (17)	12/254 (5)	(10)	2.91 (1.06-7.99)	.04	2.77 (0.97-7.88)	.06
mRS score ≤4 at 1 y	143/234 (61)	59/254 (23)	(38)	5.55 (3.42-9.00)	<.001	5.34 (3.26-8.74)	<.001
Death at 1 y	68/234 (29)	180/254 (71)	(-41)	0.16 (0.10-0.24)	<.001	0.16 (0.10-0.24)	<.001

HAMLET: impact du délai de traitement

Hofmeijer et al. Lancet Neurol 2009

MRS À 1 AN

■ 2 ■ 3 ■ 4 ■ 5 ■ 6



Randomisés ≤ 48hrs post AVC: mRS ≤ 4 = 52% vs 22%†

Randomisés > 48hrs post AVC: aucun effet de la chirurgie

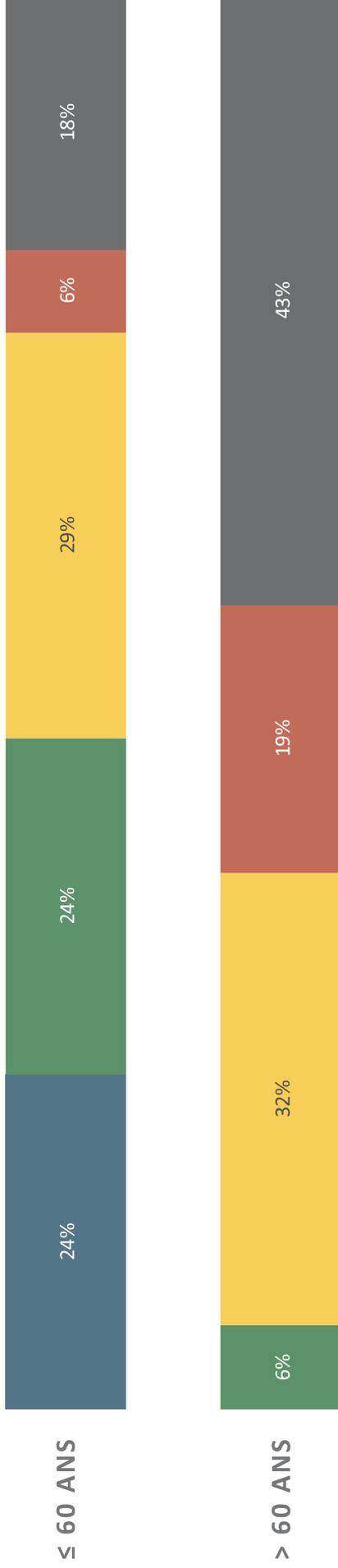
DESTINY vs DESTINY-II: impact de l'âge

Jüttler et al. NEJM 2014

Jüttler et al. Stroke 2007

MRS À 1 AN

■ 2 ■ 3 ■ 4 ■ 5 ■ 6



CD - modification des lignes directrices

2019 → 2026

1. Although the optimal trigger for decompressive craniectomy is unknown, it is reasonable to use a decrease in level of consciousness attributed to brain swelling as selection criteria.	Ila	A
2. In patients ≤60 years of age who deteriorate neurologically within 48 hours from brain swelling associated with unilateral MCA infarctions despite medical therapy, decompressive craniectomy with dural expansion is reasonable.	Ila	A
3. In patients >60 years of age who deteriorate neurologically within 48 hours from brain swelling associated with unilateral MCA infarctions despite medical therapy, decompressive craniectomy with dural expansion may be considered.	IIb	B-R

COR	LOE	Recommendations
2a	B-NR	1. In patients with large territorial cerebral infarctions at high risk for developing brain swelling and hemiation, decreased level of consciousness attributed to brain swelling is a reasonable trigger for decompressive hemicraniectomy selection. ¹
1	A	2. In patients ≤60 years of age with unilateral MCA infarctions who deteriorate neurologically within 48 hours from brain swelling despite medical therapy, decompressive craniectomy with dural expansion is beneficial to reduce mortality and improve functional outcome. ²⁻⁶
2b	B-R	3. In patients >60 years of age with unilateral MCA infarctions who deteriorate neurologically within 48 hours from brain swelling despite medical therapy, decompressive craniectomy with dural expansion may be considered to reduce mortality. ^{3,7-9}
2b	B-NR	4. In patients with AIS who received IV tPA thrombolysis and develop malignant cerebral edema despite medical therapy, early decompressive craniectomy within 48 hours may still be considered without additional safety concerns. ^{10,11}

CA "Surgery should be performed < 48h from stroke onset, ideally before clinical deterioration"
 "The discussion with the patient and decision-makers should state more clearly that there is a survival benefit, but an uncertain impact on quality of life and disability. Furthermore that even with treatment, a good outcome (MRS 0-2) is rare."

Infarctus cérébelleux – recommandations

COR	LOE	Recommendations
1	C-LD	1. In patients with cerebellar infarction and obstructive hydrocephalus, ventriculostomy is recommended to improve neurological function and decrease mortality. Concomitant or subsequent decompressive craniectomy may or may not be necessary on the basis of factors such as the size of the infarction, neurological condition, degree of brainstem compression, and effectiveness of medical management. ¹⁻³
1	B-NR	2. In patients with cerebellar infarction causing neurological deterioration from brainstem compression or volumes ≥ 35 mL, decompressive suboccipital craniectomy with dural expansion should be performed to improve outcomes and decrease mortality. ¹⁻⁷

A-B-C en AVC malin & thérapies complémentaires

- **Airway**
 - Intubation si insuffisance respiratoire ou détérioration neurologique³
- **Breathing**
 - Pas d'hyperventilation prophylactique!
 - Hyperventilation brève, modérée (PaCO₂ 30-34) «bridge to definitive therapy»¹
- **Circulation**
 - Pas de cible spécifique recommandable...²
 - PAM > 85, Pasyst < 220 (strong recommendation, [low quality evidence](#))³
 - Hypotension inhabituelle, cause secondaire à éliminer
- **Thérapies complémentaires – considérer HTIC!**
 - Osmothérapie raisonnable si détérioration clinique¹
 - Hypothermie, barbituriques, cortico non recommandés¹
 - Monitoring de PIC de routine non-recommandé³

1- Powers et al. Stroke 2019

2- Wijidicks et al. Stroke 2014

3- Torbey et al. Neurocrit Care 2015

Hémorragie intracérébrale

AHA/ASA GUIDELINE

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

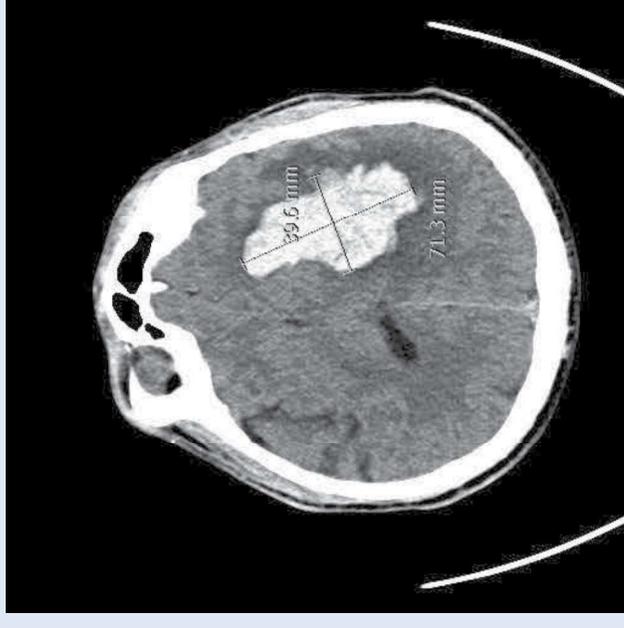
Reviewed for evidence-based integrity and endorsed by the American Association of Neurological Surgeons and Congress of Neurological Surgeons.

Endorsed by the Society of Vascular and Interventional Neurology/

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

Endorsed by the Neurocritical Care Society

Stroke. 2022;53:e282–e361.



Canadian stroke best practice recommendations: Management of Spontaneous Intracerebral Hemorrhage, 7th Edition Update 2020

Ashkan Shoamanesh (Co-chair)^{1,2}, M Patrice Lindsay³, Lana A Castellucci^{4,5}, Anne Cayley⁶, Mark Crowther⁷, Kerstin de Wit^{8,9}, Shane W English^{10,11}, Sharon Hoosein¹², Thien Huynh^{13,14}, Michael Kelly¹⁵, Cian J O’Kelly^{1,6}, Jeanne Teitelbaum^{17,18}, Samuel Yip¹⁹, Dar Dowlats Shahi²⁰, Eric E Smith²¹, Norine Foley²², Aleksandra Pikula⁶, Anita Mountain^{23,24}, Gord Gubitz²⁵ and Laura C Gioia^{17,26} (Co-chair), on behalf of the Canadian Stroke Best Practices Advisory Committee in collaboration with the Canadian Stroke Consortium and the Canadian Hemorrhagic Stroke Trials Initiative Network (CoHESIVE)

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2021, Vol. 16(8) 321–341
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Hémorragie intracérébrale

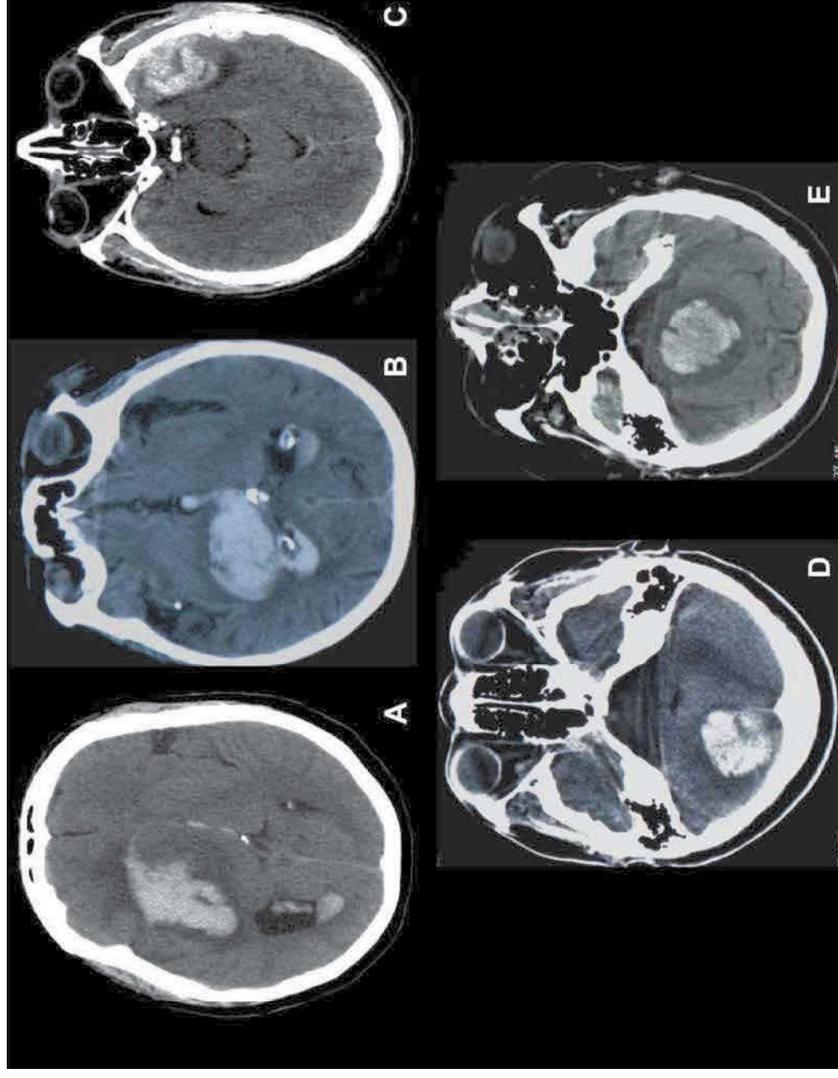
- Hémorragie intracérébrale "primaire" non-traumatique
 - 2^e cause d'accident vasculaire cérébral (10-25% des AVC)
 - Pire morbidité/mortalité
 - Taux de mortalité 40%
 - Évolution neurologique défavorable 60-70%

• Étiologies multiples

- Causes primaires (atteinte microvasculaire)
 - Artériosclérose artérioles perforantes (NGC, thalamus, tronc cérébrale, cervelet) – plus fréquente (HTA, Db, âge)
 - Angiopathie amyloïde (hémorragie lobaire) – patients plus âgés (âge > 60 ans, apolipoprotéine E allèle ε2 ou ε4)
- Causes secondaires
 - Malformation vasculaires (anévrisme, MAV, fistule durale)
 - Transformation hémorragique secondaire (AVC ischémique, thrombose veineuse cérébrale – infarctus veineux)
 - Néoplasie (primaire, métastase)
 - Infectieuses (embolie septique, anévrisme mycotique, encéphalite HSV, etc)
 - Coagulopathie (primaire, médicamenteuse, insuffisance hépatique)
 - Hyperperfusion cérébrale
 - Autres: Moyamoya, RCVS, drogues (cocaïne, amphétamines), vasculites

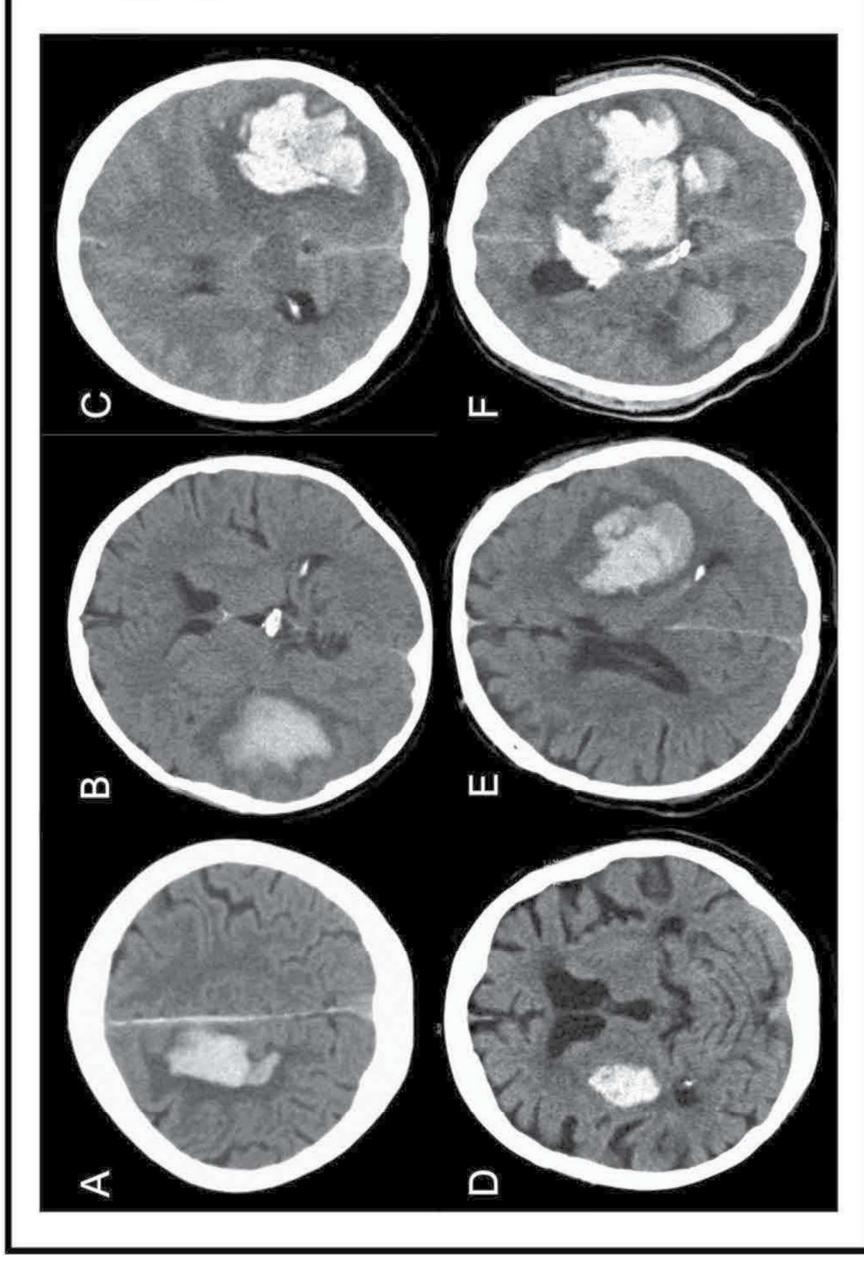
STITCH	Early surgery (n=503)	Initial conservative treatment (n=530)
Men	285 (57%)	306 (58%)
Age (years)	62 (52-70)	62 (53-71)
Any anticoagulation or thrombolytic treatment contributing to ICH	39 (8%)	55 (10%)
Past medical history*		
Hypertension	341 (69%)	378 (72%)
On antihypertensives	225 (46%)	263 (50%)
Previous myocardial infarction	28 (6%)	44 (8%)
Previous stroke	30 (6%)	43 (8%)
Smoker	146 (30%)	134 (26%)
Other medical disorders	132 (27%)	143 (27%)

Hémorragie intracérébrale: localisation typique



Taille d'hématome: 10 vs 30 vs 60 mL

LOBAIRE →



PROFOND →

Hémorragie intracérébrale

- Morbidité élevée (< 40% récupèrent indépendance fonctionnelle)
- Mortalité élevée (35% à 1 semaine, 59% à 1 an)
- Conséquences selon **volume**, **localisation** et **comorbidités**
 - Déficit focal ad altération d'état de conscience
 - HTIC – effet de masse régional, ischémie globale, engagement...
 - Déversement intraventriculaire → hydrocéphalie
 - Convulsions (15%)
 - Extracrânien → aspiration, cardiopathie
 - Comorbidités

Caractéristique	Points
GCS	
3-4	2
5-12	1
13-15	0
Âge	
≥ 80 ans	1
< 80 ans	0
Volume	
≥ 30 mL	1
< 30 mL	0
Hémorragie intraventriculaire	
Oui	1
Non	0
Localisation infratentorielle	
Oui	1
Non	0
Total	0-6
ICH Score	
Mortalité à 30 Jrs	
0	0-10%
1	7-13%
2	30-44%
3	56-78%
4	70-100%
5-6	100%

Domage cérébral

- Pression directe par l'hématome et **progression d'hématome**
- Effet d'HTIC régionale et globale (PPC, engagement)
- Hydrocéphalie
- Processus physiopathologiques secondaires
 - Neuroinflammation
 - Œdème cérébral
 - Toxicité biochimique produits de dégradation de l'hématome (Hb, Fe, trombine)



CIBLES THÉRAPEUTIQUES POTENTIELLEMENT NEUROPROTECTRICES



Progression d'hématome = précoce

Progression d'hématome (> 40% ou > 10 mL)

Délai entre début des symptômes et 1^{er} scan

0-3h	27/74	36%
3-6h	7/45	16%
6-12h	5/33	15%
12-24h	2/34	6%
> 24h	0/18	0%
Total	41/204	20%

Kazui et al. Stroke 1996

Progression d'hématome (> 33% ou > 6 mL)

Délai entre début des symptômes et 1^{er} scan

0-3h	27/70	38,6%
3-6h	10/92	10,9%
> 6h	14/129	10,9%
Inconnu	20/100	20%
Total	71/391	18,2%

Brower et al. Neurocrit Care 2012

Brouwers et al. Neurocrit Care 2012
Kazui et al. Stroke 1996
Brott et al. Stroke 1997

Répercussions sur

- Soins au patient
- Efficacité d'interventions

Progression d'hématome = grave

- Progression d'HIC → détérioration, mauvaise évolution, mortalité
 - 70% des patients vont avoir progression d'HIC, 30% progression significative (> 33%)¹
 - Progression de 10% d'HIC → ↑ 16% probabilité d'augmenter mRS de 1¹
 - Progression de 1 mL d'HIC → ↑ 5% probabilité d'évoluer vers mRS 3-5²
 - 25% nécessiteront intervention chirurgicale suite à progression HIC³
- Facteurs de risque
 - Délai début sx → 1^{er} CT (typiquement dans les 1^{ers} 24hrs)
 - Spot sign, spot sign score et autres signes à l'imagerie
 - Volume initial de l'HIC
 - Warfarine
- Imagerie sériée? (p.ex. 6-24-48h après scan initial)
 - Identifier risque de progression d'hématome et triage, pronostic?
- Évaluation neurologique sériée

1- Davis et al. Neurology 2006

2- Delcourt et al. INTERACT1 Neurology 2012

3- Maas et al. Neurology 2013

4- Mayer et al. FAST NEJM 2008

5- Gioia et al. Curr Opin Crit Care 2015

6- Greenberg et al. Stroke 2022

Interventions thérapeutiques

- Traitements de support – ABC
- Identifier et prévenir progression d'hématome: ↓ PA, renverser coagulopathie
- Interventions neurochirurgicales
- Prévenir dommage secondaire par HTIC, convulsions, insultes secondaires...

HYPERACUTE PHASE

ACUTE PHASE

<p>MEDICAL STABILIZATION & PREVENTION OF HEMATOMA EXPANSION</p> <p>A. ABC</p> <ul style="list-style-type: none"> - Airway protection (especially if GCS ≤ 8) - Oxygenation (SpO₂ > 94%) - Normoventilation (PaCO₂ 35-45 mmHg) - Hemodynamic resuscitation <p>B. Monitoring</p> <ol style="list-style-type: none"> Vital signs Standardized neurological assessment (baseline) – NIHSS or CNS; GCS or Four Score if patient obtunded/comatose <p>Frequent assessments for timely detection of clinical deterioration and signs of increased ICP</p> <p>C. Key History & Laboratory Investigations</p> <ul style="list-style-type: none"> - Time of symptoms onset (last normal) - Drugs (sympathomimetics, anticoagulants, antiplatelets) - History of coagulopathy - Recent surgery - Advanced health directives - Allergy - CBC, electrolytes/creatinine, PTT and INR, serum glucose, troponin, tox screen <p>D. Prevention of hematomas expansion</p> <p>1. Blood Pressure Management Target: SBP < 140 mmHg (within less than 1 hour of presentation to the ED)</p> <p>2. Correction of Coagulopathy (if present)</p> <ul style="list-style-type: none"> - FAS - MO - Heparin and LMWH - Antiplatelet agents - Thrombocytopenia - Fibrinolytic agents <p>E. If signs/evidence of increased ICP: Treat underlying causes of increased ICP - Initiate or intensify ICP-directed treatment</p>	<p>IMAGING</p> <p>A. NECT/CTA</p> <ul style="list-style-type: none"> - Location (supratentorial/infratentorial) - Volume (ABC2) - Presence of ICH - Signs of mass effect/herniation - "Sign" sign - Etiology <p>B. IF ETIOLOGY NOT IDENTIFIED ON NECT/CTA</p> <ul style="list-style-type: none"> - Subcortical/ Cortical - Primary ICH <p>No further imaging required</p>	<p>CONSIDER SURGICAL INTERVENTION</p> <ul style="list-style-type: none"> - Early neurological consultation for potential evacuation and/or decompression - Consider EVD insertion if hydrocephalus <p>- ICH LOCATION</p> <p>INFRA-TENTORIAL ICH ICH evacuation with cerebellar decompression in patients with: a) ICH > 3 cm in diameter who are deteriorating, and/or b) compression of the brainstem, and/or c) hydrocephalus</p> <p>SUPRATENTORIAL ICH Consider evacuation if: - Describes two large ICHs - Consider early in patients with ongoing clinical deterioration, recent onset of haemorrhage, and relatively accessible hematomas if: a. lesions with marked mass effect, edema, or midline shift (high risk of herniation) b. lesions where the symptoms appear due to increased ICP or to mass effect (vs symptoms attributable directly to brain injury from hemorrhage, unlikely to be reversed by surgical evacuation) c. persistent elevated ICP refractory to medical management</p>	<p>PREVENTION OF SECONDARY BRAIN INJURY</p> <p>A. If signs/evidence of increased ICP: Check for treat systemic causes of increased ICP - Initiate or intensify ICP-directed treatment</p> <p>B. Seizures</p> <ul style="list-style-type: none"> - Treat clinical seizures - No role for prophylaxis <p>B. General ICU management</p> <ul style="list-style-type: none"> - Early enteral nutrition - Avoid hypotatremia - Avoid hyperglycemia (> 10 mmol/L, and hypoglycemia < 3.9 mmol/L) - Maintain normothermia - Prevention of VTE (IFCs; consider pharmacological prophylaxis on day 2-3) - Anemia
			<p>RISK STRATIFICATION</p> <p>A. COMPLETE HISTORY CHECKLIST</p> <ul style="list-style-type: none"> - Hypertension - Previous stroke - History of coagulopathy - Known vascular abnormalities - History of cancer/brain tumor - Recent surgery / trauma - Recent childbirth - Major comorbidities - Prior functional status - Drugs - Substance abuse - Allergy <p>B. ICH SCORING SCALES CALCULATION Prognostic scores should only be used as a tool to communicate a particular patient's condition, for risk stratification, or for research purposes. They should not be used to determine prognosis in the initial management of patients with ICH, where, in general, aggressive therapy is recommended.</p>

Conclusion sur la réduction de PA en HIC

	INTERACT2 (NEJM 2013)	ATACH-2 (NEJM 2016)
Patients	2839	1000 (/1280 - futilité)
PAs initiale	179 mmHg	200 mmHg
Délai HIC- traitement	HIC-antiHTA ~ 4-4.5h	HIC-antiHTA ~ 2.5-2.75h
Traitement	PA syst < 180 mmHg PA syst < 140 mmHg Délai ≤ 1 heure Durée 7 jours Urapidil...	PA syst 140-170 mmHg PA syst 110-139 mmHg Délai ≤ 2 heures Durée 24 heures Nicardipine
Taille HIC	11 mL	10 mL
Échec tx	66% à 1h	12% à 2h
Bénéfice mRS	OUI	NON
↓ progression	POST-HOC	TENDANCE
Effet adverse	NON	OUI

• Signaux divergents?

- Trop vite
- Trop intense
- Trop bref

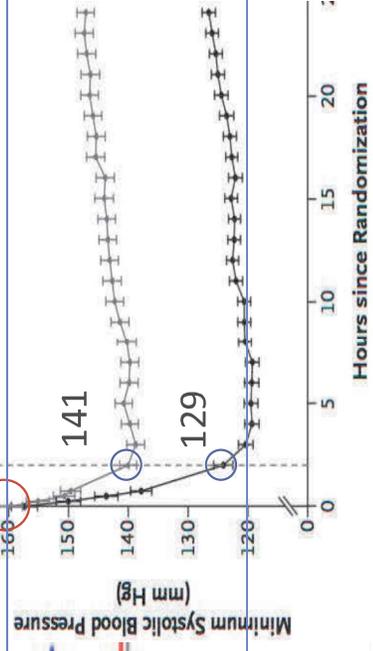
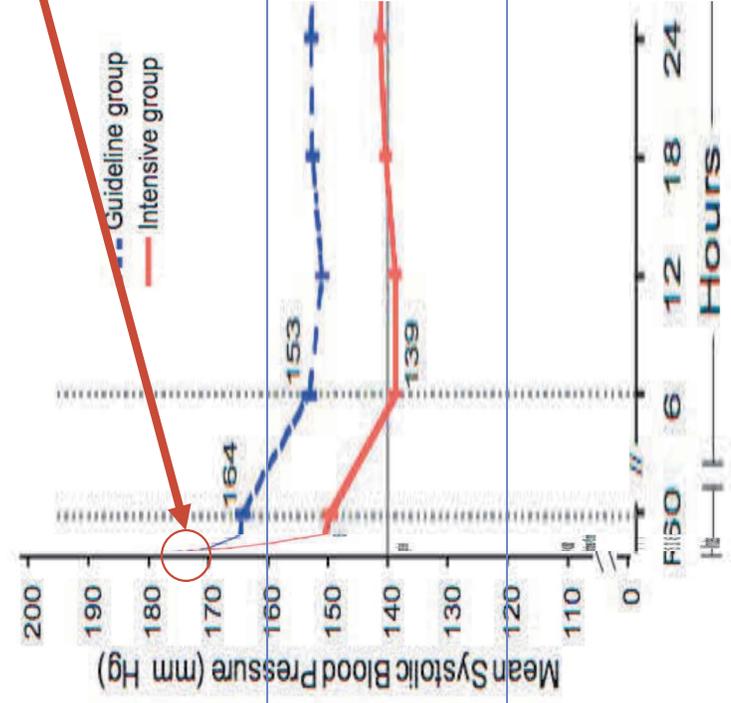
• Réduction ad 140 mmHg semble néanmoins sécuritaire

• Validité externe pour population SI questionnable

- Taille d'hématome
- Présence d'HTIC?
- Intervention neurochirurgicale?

INTERACT2 vs ATACH-2

Anti-HTA pré-randomisation
INTERACT2 → critère inclusion = $PA_{syst} \leq 220$ mmHg
ATACH-2 → tx IV permis pré-rando pour $PA_{syst} < 180$
(TA_{syst} moyenne à l'urgence ~ 200 mmHg)



Early lowering of blood pressure after acute intracerebral haemorrhage: a systematic review and meta-analysis of individual patient data

Moullaali TJ, et al. *J Neurol Neurosurg Psychiatry* 2022

Table 2 Effect of active/intensive versus placebo/guideline blood pressure lowering interventions on outcomes after acute intracerebral haemorrhage (ICH)

Outcome	Randomly allocated treatment group		Adjusted OR* (95% CI)	P value
	Active/intensive	Placebo/guideline		
Primary: unfavourable shift in modified Rankin Scale (mRS) scores	/3062	/3039	0.97 (0.88 to 1.06)	0.50
Secondary	/3062	/3039		
Dependency or death (mRS 3–6)	1735 (56.7)	1742 (57.3)	0.95 (0.84 to 1.08)	0.42
Severe dependency or death (mRS 4–6)	1214 (39.7)	1228 (40.4)	0.95 (0.84 to 1.08)	0.41
Death	411/3111 (13.2)	405/3087 (13.1)	1.01 (0.85 to 1.20)	0.91
Haematoma growth at 24 hours†				
Mean growth (95% CI), mL	3.2 (2.5 to 3.9)	4.3 (3.4 to 5.2)	Absolute difference: –1.10 (–2.22 to 0.01)	0.05
Absolute growth ≥6 mL	212/1280 (16.6)	252/1230 (20.5)	0.75 (0.60 to 0.92)§	0.007
Relative growth ≥33%	296/1280 (23.1)	326/1230 (26.5)	0.82 (0.68 to 0.99)§	0.03

Lignes directrices: réduction PA



COR	LOE	Recommendation
2a	B-NR	1. In patients with spontaneous ICH requiring acute BP lowering, careful titration to ensure continuous smooth and sustained control of BP, avoiding peaks and large variability in SBP, can be beneficial for improving functional outcomes. ¹³⁸
2a	C-LD	2. In patients with spontaneous ICH in whom acute BP lowering is considered, initiating treatment within 2 hours of ICH onset and reaching target within 1 hour can be beneficial to reduce the risk of HE and improve functional outcome. ^{139,140}
2b	B-R	3. In patients with spontaneous ICH of mild to moderate severity presenting with SBP between 150 and 220 mm Hg, acute lowering of SBP to a target of 140 mm Hg with the goal of maintaining in the range of 130 to 150 mmHg is safe and may be reasonable for improving functional outcomes. ^{138,141-147}
2b	C-LD	4. In patients with spontaneous ICH presenting with large or severe ICH or those requiring surgical decompression, the safety and efficacy of intensive BP lowering are not well established. ¹⁴⁸
3: Harm	B-R	5. In patients with spontaneous ICH of mild to moderate severity presenting with SBP >150 mm Hg, acute lowering of SBP to <130 mm Hg is potentially harmful. ^{146,149,150}

Réduction PA en HIC modérée-sévère

Characteristics	Moderate to severe grade (n = 682)
Age, mean (SD)	61.9 (13.1)
Male	426 (62.5%)
Race	
American Indian or Alaska Native	1 (0.1%)
Asian	382 (56.0%)
African American	86 (12.6%)
White	199 (29.2%)
Others or unknown	14 (2.1%)
Ethnicity	
Hispanic ethnic group	53 (7.8%)
Non-Hispanic ethnic group	610 (89.4%)
Unknown/not reported	19 (2.8%)
Baseline GCS score, mean (SD)	13.2 (2.3)
3–11	147 (21.6%)
12–14	247 (36.2%)
15	288 (42.2%)
Baseline NIHSS score, median (SD)	14.6 (6.2)
SBP mm Hg, mean (SD) ^a	174.7 (24.8)
Intracerebral hematoma volume mL, mean (SD)	17.2 (12.8)
Location of hemorrhage	
Basal ganglia	397/681 (58.3%)
Thalamus	217/681 (31.9%)
Cerebral lobe	67/681 (9.8%)



- Réduction PA intensive en HIC modérée-sévère
- Définition HIC modérée-sévère (1 critère)
 - GCS < 13
 - NIHSS ≥ 10
 - HIC ≥ 30 mL
 - Présence d'HIV*
- Issue principale = mRS 4-6 à 3 mois

**UN PEU PLUS REPRÉSENTATIF
 DE NOS PATIENTS?**

Qureshi et al. Cerebrovasc Dis 2020 (post-hoc ATACH-2)
 Greenberg et al. Stroke 2022

Nouvelles recommandations

Évolution moins favorable en général



Table 2. Primary, secondary, and safety outcomes according to ICH severity^a

Outcome	Moderate to severe (n = 682)	Mild (n = 318)	Relative risk or beta estimate (95% CI)	p value
Primary outcome: death or disability ^b	333/657 (50.7%)	34/304 (11.2%)	4.5 (3.3–6.3)	<0.0001
Hematoma expansion	160/662 (24.2%)	61/314 (19.4%)	1.2 (1.0–1.6)	0.1
Neurologic deterioration within 24 h	79 (11.6%)	16 (5.0%)	2.3 (1.4–3.9)	0.001
Treatment-related serious adverse event within 72 h ^c	10 (1.5%)	4 (1.3%)	1.2 (0.4–3.7)	1
Any serious adverse event within 3 mo	191 (28.0%)	36 (11.3%)	2.5 (1.8–3.4)	<0.0001
Hypotension within 72 h	9 (1.3%)	0 (0%)	–	–
EQ-5D utility index score, median (range) ^{d, e}	0.7 (–0.1 to 1)	0.8 (0.1–1)	0.4477 (0.39–0.52)	<0.0001
EQ-5D visual-analog scale score, median (range) ^{d, f}	65 (0–100)	70 (8–100)	0.9942 (0.99–0.99)	<0.0001

Qureshi et al. Cerebrovasc Dis 2020 (post-hoc ATACH-2)
 Greenberg et al. Stroke 2022

Nouvelles recommandations

Effet du traitement intensif...

- **Évolution neuro défavorable idem**
- **↓ progression d’HIC**
- **↑ détérioration neuro précoce?**



Table 3. Primary, secondary, and safety outcomes of the participants (moderate-to-severe ICH group) according to treatment group^a

Outcome	Intensive SBP reduction (n = 336)	Standard SBP reduction (n = 346)	Relative risk or beta estimate (95% CI)	p value	Adjusted odds ratio ^g (95% CI)	p value after adjusted
Primary outcome: death or disability ^b	170/324 (52.5%)	163/333 (48.9%)	1.1 (0.9–1.2)	0.37	1.1 (0.8–1.5)	0.64
Hematoma expansion	67/329 (20.4%)	93/333 (27.9%)	0.7 (0.55–0.96)	0.02	0.6 (0.4–0.9)	0.02
Neurologic deterioration within 24 h	46 (13.7%)	33 (9.5%)	1.4 (0.9–2.2)	0.09	1.6 (0.9–2.6)	0.08
Treatment-related serious adverse event within 72 h ^c	6 (1.8%)	4 (1.2%)	1.5 (0.4–5.4)	0.54	1.4 (0.4–5.4)	0.63
Any serious adverse event within 3 mo	110 (32.7%)	81 (23.4%)	1.4 (1.1–1.8)	0.01	1.7 (1.2–2.5)	0.003
Hypotension within 72 h	6 (1.8%)	3 (0.9%)	2.1 (0.5–8.2)	0.33	3.0 (0.6–14.8)	0.19
EQ-5D utility index score, median (range) ^{d,e}	0.7 (–0.1–1)	0.7 (–0.04–1)	1.09 (0.99–1.21)	0.08	–	–
EQ-5D visual-analog scale score, median (range) ^{d,f}	60 (5–100)	70 (0–100)	1.0006 (0.9992–1.0021)	0.4	–	–

Qureshi et al. Cerebrovasc Dis 2020 (post-hoc ATACH-2)
Greenberg et al. Stroke 2022



Intracranial Pressure and Cerebral Perfusion Pressure in Large Spontaneous Intracranial Hemorrhage and Impact of Minimally Invasive Surgery

- Étude post-hoc exploratoire
 - Cohorte MISTIE III – 72 pts, lectures PIC/PPC ad 6 jrs
 - HIC moyen 44 mL
- HTIC ≥ 20 fréquente ($> 40\%$ pts)
- PPC < 70 fréquente ($> 70\%$ pts)
- Proportion PPC < 70 et 60 associée à mortalité à court terme
- Proportion PIC ≥ 20 , ≥ 30 et PPC < 70 associée mortalité à long terme

Incertitudes...

- HIC avec HTA extrême (TAsyst > 220 mmHg)
- HIC sévères (>20-30 mL? HIV important? Décompression?)
- HIC lobaire (peu représentés dans INTRACT2 et ATACH-2)
- Agents de choix
- Réduction de PAsyst tardive (> 4-6h)
- Modalité de mesure de PA (PNI vs invasive)
 - Variabilité avec canule artérielle...
- Seuils thérapeutiques (PIC/PPC) en HTIC

CONSIDÉRATIONS SURREPRÉSENTÉES AUX SOINS INTENSIFS

Renverser la coagulopathie

AHA/ASA GUIDELINE

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

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

Endorsed by the Society of Vascular and Interventional Neurology

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

Endorsed by the Neurocritical Care Society

Stroke. 2022;53:e282–e361.

Guideline for Reversal of Antithrombotics in Intracranial Hemorrhage: Executive Summary. A Statement for Healthcare Professionals From the Neurocritical Care Society and the Society of Critical Care Medicine

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(Crit Care Med 2016; 44:2251–2257)

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SAGE

Canadian stroke best practice recommendations: Management of Spontaneous Intracerebral Hemorrhage, 7th Edition Update 2020

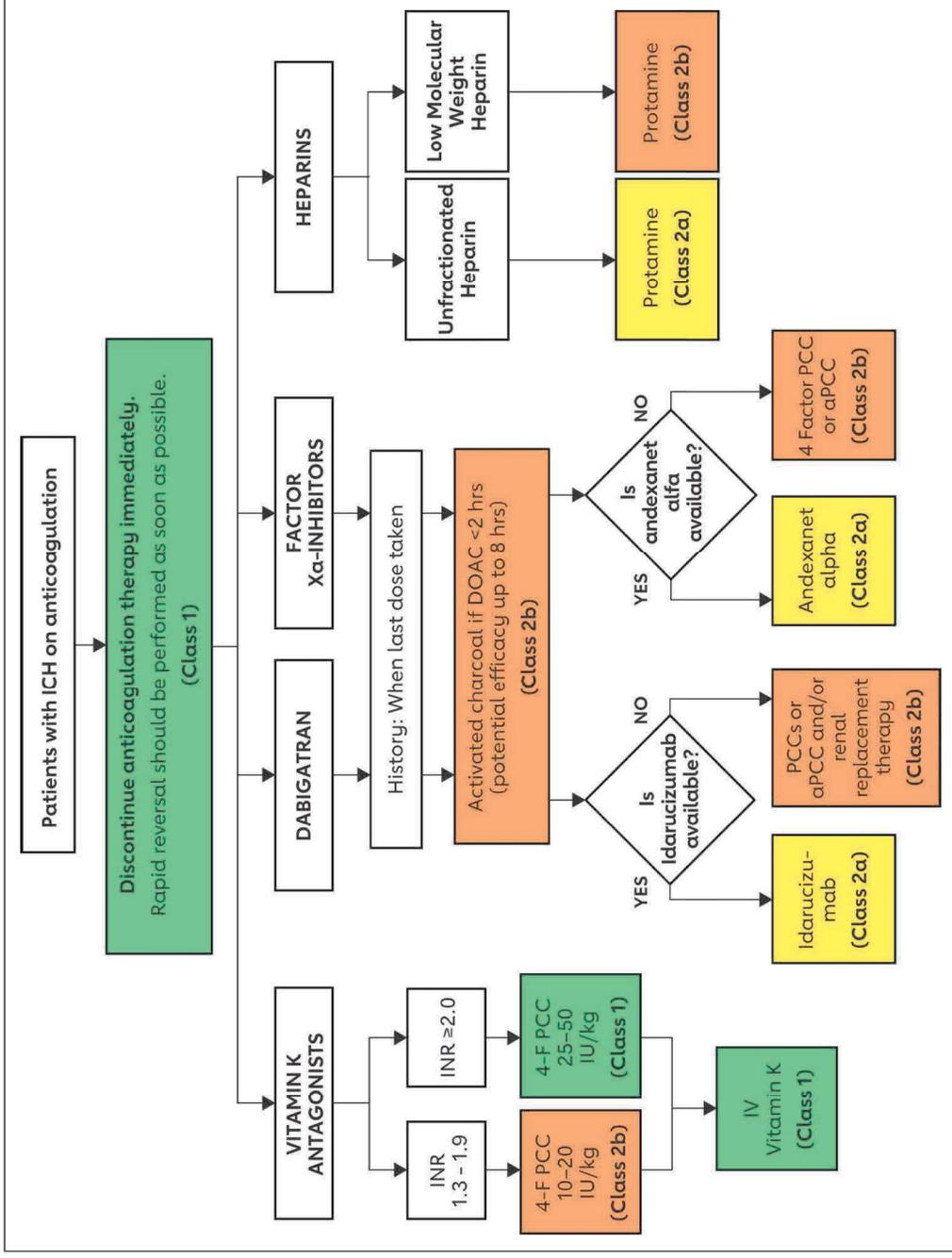
Ashkan Shoamanesh (Co-chair)^{1,2}, M Patrice Lindsay³, Lana A Castellucci^{4,5}, Anne Cayley⁶, Mark Crowther⁷, Kerstin de Wit^{8,9}, Shane W English^{10,11}, Sharon Hoosein¹², Thien Huynh^{13,14}, Michael Kelly⁵, Cian J O’Kelly¹⁶, Jeanne Teitelbaum^{17,18}, Samuel Yip¹⁹, Dar Dowlatshahi²⁰, Eric E Smith²¹, Norine Foley²², Aleksandra Pjkula⁶, Anita Mountain^{23,24}, Gord Gubitz²⁵ and Laura C Gioia^{17,26} (Co-chair), on behalf of the Canadian Stroke Best Practices Advisory Committee in collaboration with the Canadian Stroke Consortium and the Canadian Hemorrhagic Stroke Trials Initiative Network (CoHESIVE)

Lignes directrices: renverser coagulopathie

- HIC sous anticoagulants ou antithrombotiques
 - ↑ progression d'hématome
 - ↑ mortalité et de morbidité
- Ne pas attendre labo...
 - Délai depuis dernière dose
 - PK de l'agent
 - Fonction rénale

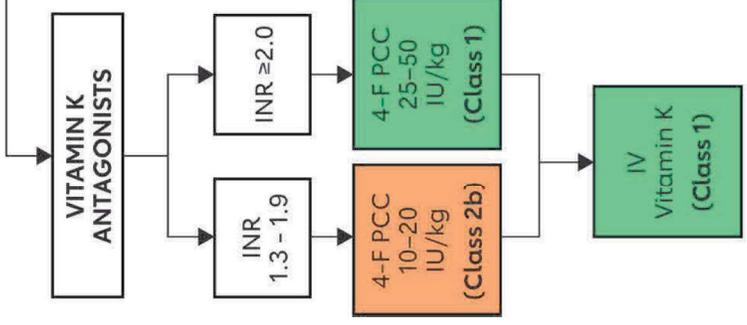


COR	LOE	Recommendations	COR	LOE	Recommendations
1	C-LD	1. In patients with anticoagulant-associated spontaneous ICH, anticoagulation should be discontinued immediately and rapid reversal of anticoagulation should be performed as soon as possible after diagnosis of spontaneous ICH to improve survival. ¹⁶²	2b	B-NR	7. In patients with direct factor Xa inhibitor-associated spontaneous ICH, a 4-F PCC or activated PCC (aPCC) may be considered to improve hemostasis. ¹⁶⁹⁻¹⁷¹
VKAs					
1	B-R	2. In patients with VKA-associated spontaneous ICH and INR ≥2.0, 4-factor (4-F) prothrombin complex concentrate (PCC) is recommended in preference to fresh-frozen plasma (FFP) to achieve rapid correction of INR and limit HE. ¹⁶³ INCH Lancet Neurol 2016	2b	C-LD	8. In patients with dabigatran- or factor Xa inhibitor-associated spontaneous ICH, when the DOAC agent was taken within the previous few hours, activated charcoal may be reasonable to prevent absorption of the DOAC. ¹⁷²⁻¹⁷⁴
1	C-LD	3. In patients with VKA-associated spontaneous ICH, intravenous vitamin K should be administered directly after coagulation factor replacement (PCC or other) to prevent later increase in INR and subsequent HE. ^{164,165}	2b	C-LD	9. In patients with dabigatran-associated spontaneous ICH, when idarucizumab is not available, aPCC or PCCs may be considered to improve hemostasis. ^{175,176}
2b	C-LD	4. In patients with VKA-associated spontaneous ICH with INR of 1.3 to 1.9, it may be reasonable to use PCC to achieve rapid correction of INR and limit HE. ^{162,164}	2b	C-LD	10. In patients with dabigatran-associated spontaneous ICH, when idarucizumab is not available, renal replacement therapy (RRT) may be considered to reduce dabigatran concentration. ¹⁷⁷
DOACs					
2a	B-NR	5. In patients with direct factor Xa inhibitor-associated spontaneous ICH, andexanet alfa is reasonable to reverse the anticoagulant effect of factor Xa inhibitors. ^{166,167}	2a	C-LD	11. In patients with unfractionated heparin (UFH)-associated spontaneous ICH, intravenous protamine is reasonable to reverse the anticoagulant effect of heparin. ¹⁷⁸
2a	B-NR	6. In patients with dabigatran-associated spontaneous ICH, idarucizumab is reasonable to reverse the anticoagulant effect of dabigatran. ¹⁶⁸	2b	C-LD	12. In patients with low-molecular-weight heparin (LMWH)-associated spontaneous ICH, intravenous protamine may be considered to partially reverse the anticoagulant effect of heparin. ¹⁷⁸



Patients with ICH on anticoagulation

Discontinue anticoagulation therapy immediately.
 Rapid reversal should be performed as soon as possible.
 (Class 1)



CORRECTION RAPIDE (ASAP < 4h)
CORRECTION ADÉQUATE (PCC >>> PFC, INR < 1.3, dose)
CORRECTION DURABLE (VITAMINE K q24h)



National Advisory Committee on Blood and Blood Products | Comité consultatif national sur le sang et les produits sanguins

Dose	PCC dose if INR 2 to < 4	PCC dose if INR 4 to 6	PCC dose if INR > 6
	25 IU/kg	35 IU/kg	50 IU/kg

INR	4-factor PCC dose (units/kg)	Max dose (units)
2-3.9	25	2500
4-6	35	3500
>6	50	5000

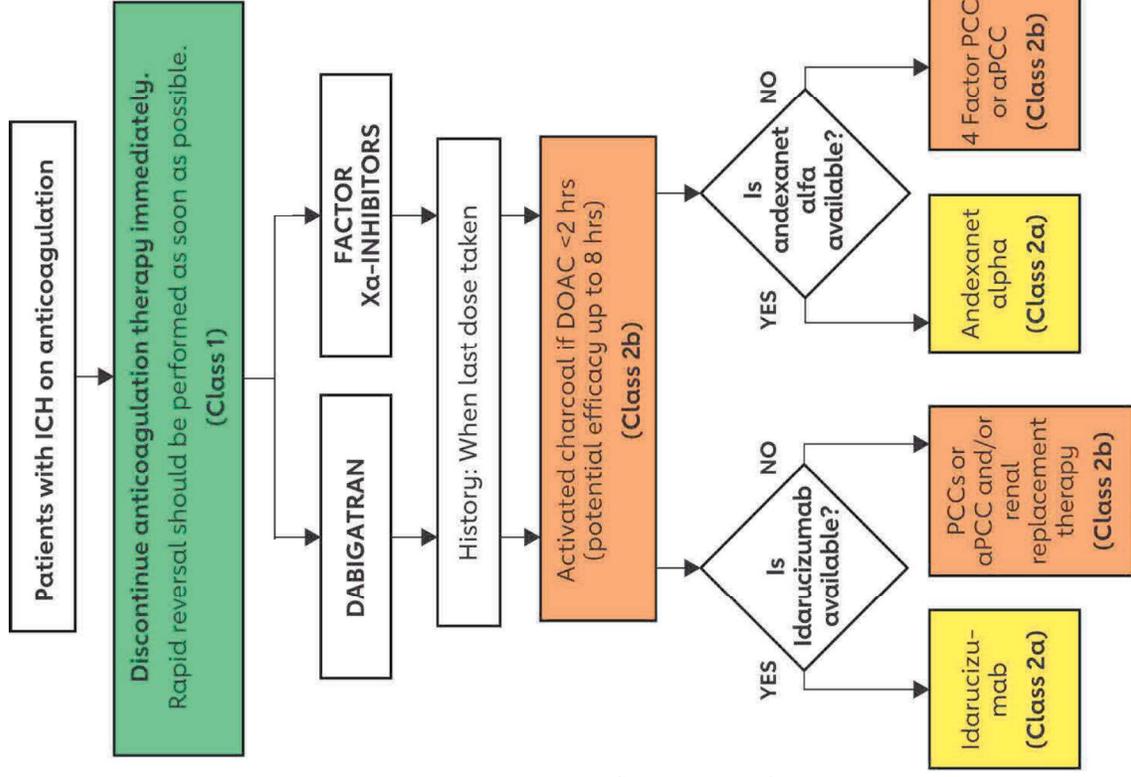
Brophy et al. Neurocrit Care 2017
 Frontera et al. Crit Care Med 2016
 Greenberg et al. Stroke 2022

PFC MIEUX QUE RIEN (15 mL/kg)

Anticoagulants oraux (DOAC, NACO, etc)

Table 2 Pharmacokinetic parameters for selected anticoagulants and antiplatelet agents [161, 423]

Medication	Mechanism of action	Elimination	Half-life	Impairment affects excretion		Dialyzable
				Renal	Hepatic	
Direct factor Xa inhibitors						
Rivaroxaban	Prevents factor Xa-mediated conversion of prothrombin to thrombin	66 % renal; 28 % fecal	5 h	Yes	Yes	No
Apixaban	Prevents factor Xa-mediated conversion of prothrombin to thrombin	majority fecal; 27 % renal	12 h	Yes	Yes	Minimal, area under the curve decreased by 14 % over 4 h
Edoxaban	Prevents factor Xa-mediated conversion of prothrombin to thrombin	50 % renal	10–14 h	Yes	Yes	No
Direct thrombin inhibitors						
Dabigatran	Competitive direct inhibition of thrombin (factor IIa) including thrombin-mediated platelet activation and aggregation	> 80 % renal	12–17 h 16.6 h in mild, 18.7 h in moderate, 27.5 h in severe renal failure, 34.1 h in patients on hemodialysis	Yes	No	Yes ~57 % over 4 h



- Interrompre l’agent!

- Connaître PK de l’agent et dernière dose

- CBA 50 g STAT si ingestion < 2-8h

- Xaban : apixaban, rivaroxaban, edoxaban

- Andexanet alpha ???

- PCC 25-50 UI/kg (ad 3000 UI)

- aPCC 20-50 UI/kg (ad 2000 UI)

- Dabigatran

- Idarucizumab 5g IV en 10 min

- PCC 50 UI/kg (ad 3000 UI)

- aPCC 50 UI/kg (ad 2000 UI)

Andexanet for Factor Xa Inhibitor–Associated Acute Intracerebral Hemorrhage

S.J. Connolly, M. Sharma, A.T. Cohen, A.M. Demchuk, A. Czlonkowska, A.G. Lindgren, C.A. Molina, D. Berezcki, D. Toni, D.J. Seiffge, D. Tanne, E.C. Sandset, G. Tsiogoulis, H. Christensen, J. Beyer-Westendorf, J.M. Coutinho, M. Crowther, P. Verhamme, P. Amarenco, R.O. Roine, R. Lemmens, R. Mikulik, R. Verhaegh, S. Middeldorp, T.G. Robinson, T.J. Milling, Jr., V. Tedim-Cruz, W. Lang, A. Himmelmann, P. Ladenvall, M. Knutsson, E. Ekholm, A. Law, A. Taylor, T. Karyakina, L. Xu, K. Tsiplova, S. Poli, B. Kallmünzer, C. Gumbinger, and A. Shoamanesh, for the ANNEXA-1 Investigators*

- RCT multicentrique internationale (23 pays) → 550/900 pts
- HIC* (0,5-60 mL*, NIHSS < 36*), début sx < 6h* + Xaban ≤ 15h*
 - HIC multicausale, HIV, HSA, HSD
- Andexanet alpha (faible dose ou haute dose) vs tx standard
- Excl: GCS < 7, chx prévue dans < 12h, thrombose < 2 sem
- Traitement administré relativement rapidement
 - Délai exact stroke-traitement? Médiane probablement ≥ 4h
 - Grande majorité ont reçu des PCC (3&4 facteurs 96%, FEIBA 4%)
- (interrompue pour efficacité)

* plusieurs modifications suite à amendements

ANNEXA-1

Table 1. Baseline Characteristics of the Patients.*

Characteristic	Andexanet (N=224)	Usual Care (N=228)
Age — yr	78.9±8.5	78.9±8.5
Female sex — no. (%)	94 (42.0)	113 (49.6)
Body-mass index†	26.9±5.3	26.3±4.6
Medical history — no. (%)		
Myocardial infarction	24 (10.7)	33 (14.5)
Stroke	48 (21.4)	48 (21.1)
Deep-vein thrombosis	18 (8.0)	22 (9.6)
Pulmonary embolism	16 (7.1)	21 (9.2)
Atrial fibrillation	202 (90.2)	192 (84.2)
Congestive heart failure	34 (15.2)	44 (19.3)
Diabetes	82 (36.6)	59 (25.9)
Creatinine clearance <30 mL/min — no. (%)	10 (4.5)	9 (3.9)
Factor Xa inhibitor used — no. (%)		
Apixaban	140 (62.5)	135 (59.2)
Rivaroxaban	64 (28.6)	65 (28.5)
Edoxaban	20 (8.9)	25 (11.0)
Hemorrhage location — no. (%)		
Intracerebral	198 (88.4)	214 (93.9)
Intraventricular	3 (1.3)	1 (0.4)
Subarachnoid	9 (4.0)	8 (3.5)
Subdural	13 (5.8)	4 (1.8)
Hemorrhage preceded by trauma — no. (%)	26 (11.6)	33 (14.5)
Systolic blood pressure in patients with intracerebral hemorrhage — mm Hg	161.2±27.0	159.8±27.7
Median hematoma volume (IQR) — mL	10.5 (4.1–24.9)	9.0 (3.1–22.8)
Median Glasgow Coma Scale score (IQR)‡	15.0 (13.0–15.0)	15.0 (13.0–15.0)
Median NIHSS score (IQR)§	9.0 (5.0–16.0)	9.0 (4.0–14.0)
Median time from symptom onset to baseline scan (IQR) — hr	2.3 (1.5–4.0)	2.4 (1.4–3.8)
Median time from baseline scan to randomization (IQR) — hr	1.1 (0.7–1.5)	1.2 (0.7–1.7)
Median time from hospital presentation to receipt of treatment (IQR) — hr¶	2.1 (1.5–2.9)	2.3 (1.7–3.1)
Patients receiving high-dose andexanet — no. (%)	45 (20.1)	—
Patients receiving low-dose andexanet — no. (%)	175 (78.1)	—
Patients receiving PCC within 3 hr — no. (%)	—	195 (85.5)

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ANNEXA-1

- Effet hémostatique démontré...
- Radiologiquement ↓ progression HIC
- Biochimiquement ↓ activité anti-Xa
- Cliniquement ↑ événements thrombotiques (2o AA vs retrait Xaban)

Table 2. Efficacy End Points.

End Point	Andexanet (N = 224)	Usual Care (N = 228)	Adjusted Difference per 100 Patients (95% CI)*	P Value**
Hemostatic efficacy	150/224 (67.0)	121/228 (53.1)	13.4 (4.6 to 22.2)	0.003
→ Hematoma volume change ≤35%†	165/215 (76.7)	137/212 (64.6)	12.1 (3.6 to 20.5)	
NIHSS score change <7 points	188/214 (87.9)	181/218 (83.0)	4.6 (-2.0 to 11.2)	
No receipt of rescue therapy between 3 hr and 12 hr	218/224 (97.3)	213/228 (93.4)	3.8 (-7.6 to 0.0)	
Hematoma volume increase ≥12.5 ml‡	24/216 (11.1)	36/214 (16.8)	-5.6 (-12.0 to 0.8)	
Hemostatic efficacy, excluding patients nonevaluable for administrative reasons	150/218 (68.8)	121/225 (53.8)	14.5 (5.7 to 23.4)	

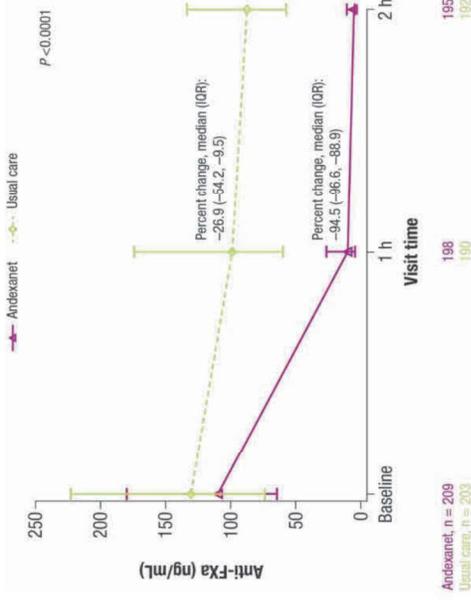


Figure S1. Reduction in Median Anti-FXa Activity From Baseline to Nadir at 2 Hours

ANNEXA-1

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- Changement absolu/relatif de progression HIC?
- mRS ≤ 3 à 30 jrs idem (28 vs 31%)

Table 3. Thrombotic Events and Deaths at 30 Days.*

Event	Andexanet (N = 263)	Usual Care (N = 267)	Increase per 100 Patients (95% CI)†	P Value‡
	<i>no. of patients (%)</i>		<i>percentage points</i>	
≥1 Thrombotic event	27 (10.3)	15 (5.6)	4.6 (0.1 to 9.2)	0.048
Transient ischemic attack	0	0	—	
Ischemic stroke	17 (6.5)	4 (1.5)	5.0 (1.5 to 8.8)	
Myocardial infarction	11 (4.2)	4 (1.5)	2.7 (–0.2 to 6.1)	
Deep-vein thrombosis	1 (0.4)	2 (0.7)	–0.4 (–2.4 to 1.5)	
Pulmonary embolism	1 (0.4)	6 (2.2)	–1.9 (–4.5 to 0.2)	
Arterial systemic embolism	3 (1.1)	2 (0.7)	0.4 (–1.7 to 2.7)	
Death	73 (27.8)	68 (25.5)	2.5 (–5.0 to 10.0)	0.51

Andexanet for Factor Xa Inhibitor–Associated Acute Intracerebral Hemorrhage

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- Financé par Portola (AZ) et entente de confidentialité avec auteurs
- Devis par MD statisticien + employés du commanditaire, medical writer...
- Refus de partage de données
- Multiples amendements après 11 mo
- Critères d’inclusions initiaux questionnables
- Point d’aboutissement primaire inhabituel
- 78 patients enrôlés après analyse intérimaire – données d’efficacité?

ONDEXXYA^{MC}

Neutralisation de l'effet anticoagulant des inhibiteurs du FXa

Avis transmis au ministre en juillet 2024

Marque de commerce : Ondexxya

Dénomination commune : Andexanet alfa

Fabricant : AZC

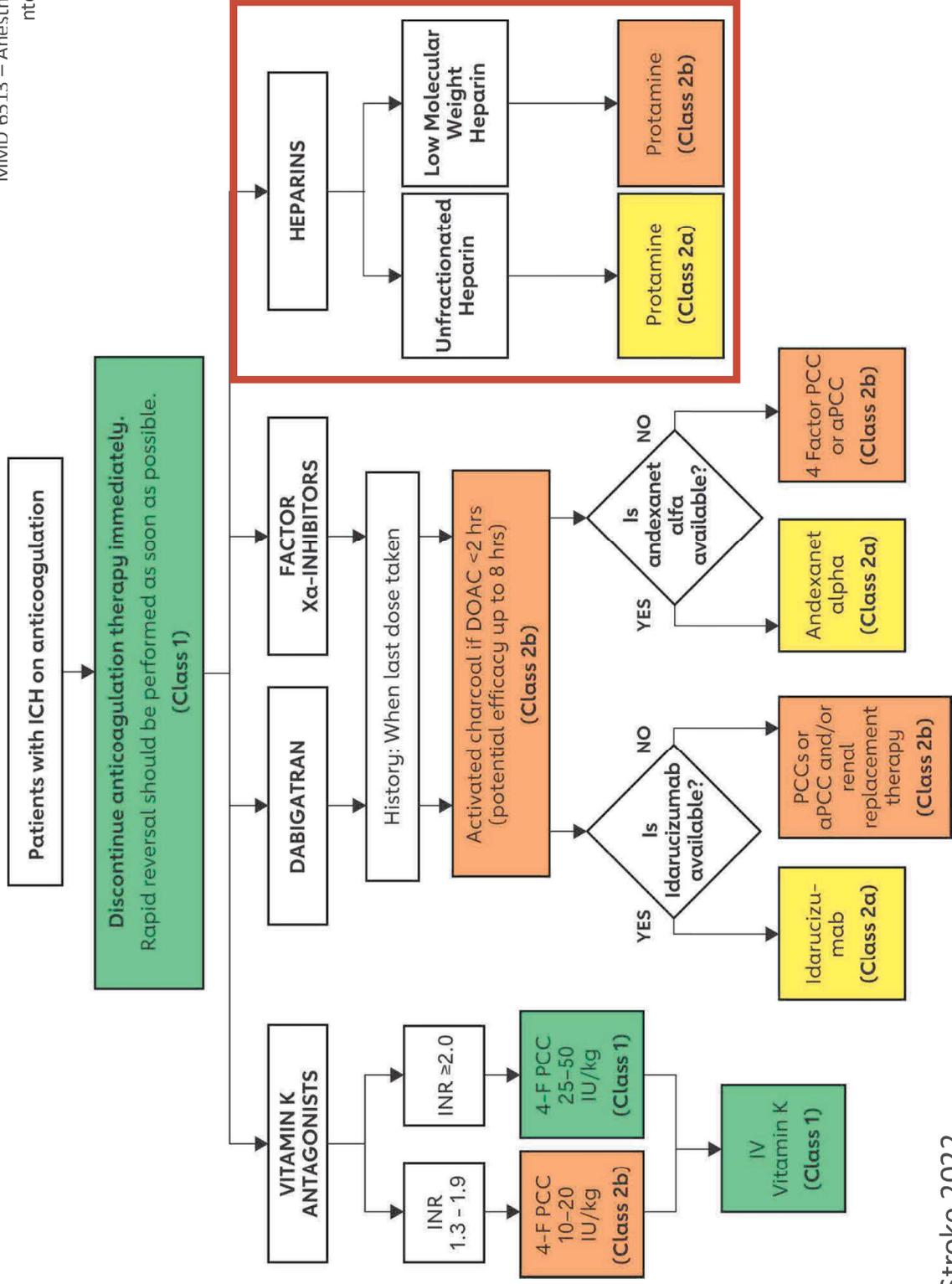
Forme : Poudre pour perfusion intraveineuse

Teneur : 200 mg

Refus d'inscription

RECOMMANDATION

L'Institut national d'excellence en santé et en services sociaux (INESSS) recommande au ministre de ne pas inscrire Ondexxya^{MC} sur la *Liste des médicaments – Établissements*, pour les patients adultes traités par des inhibiteurs du FXa (rivaroxaban ou apixaban) lorsque la neutralisation rapide de l'anticoagulation est nécessaire, car la valeur thérapeutique n'est pas reconnue.



Antiplaquettaires

- Impact incertain sur évolution neuro
- Augmentation de mortalité
- Transfusions de plaquettes
 - Pas de craniotomie → NON
 - Craniotomie → considérer?
 - Autre procédures (DVE/MIS) → ???
- Impact différent selon agents?
 - Inhibiteurs ADP P2Y12
 - Antidote ticagrelor?
- DDAVP 0,3-0,4 mcg/kg => équivoque
 - Études à venir

5.2.2. Antiplatelet-Related Hemorrhage

Recommendations for Antiplatelet-Related Hemorrhage Referenced studies that support recommendations are summarized in Data Supplements 29 through 25.		
COR	LOE	Recommendations
2b	C-LD	1. For patients with spontaneous ICH being treated with aspirin and who require emergency neurosurgery, platelet transfusion might be considered to reduce postoperative bleeding and mortality. ²⁰⁶
2b	C-LD	2. For patients with spontaneous ICH being treated with antiplatelet agents, the effectiveness of desmopressin with or without platelet transfusions to reduce the expansion of the hematoma is uncertain. ^{207–209}
3: Harm	B-R	3. For patients with spontaneous ICH being treated with aspirin and not scheduled for emergency surgery, platelet transfusions are potentially harmful and should not be administered. ²¹⁰

Traitements hémostatiques

- FVIIa → modeste HE
 - ↓ modeste progression hémato
 - ↑ thromboses artérielles?
 - Sous-groupe d'intérêt?
- Acide tranexamique
 - Sécuritaire
 - Pas d'effet sur l'évolution neuro
 - ↓ progression hémato

5.2.3. General Hemostatic Treatments

COR	LOE	Recommendations
2b	B-R	1. In patients with spontaneous ICH (with or without the spot sign), the effectiveness of recombinant factor VIIa to improve functional outcome is unclear. ^{218,219}
2b	B-R	2. In patients with spontaneous ICH (with or without the spot sign, black hole sign, or blend sign), the effectiveness of TXA to improve functional outcome is not well established. ^{220–222}

PLC, monitoring et traitements

- Incidence d'HTIC mal documentée
 - Jeunes patients, HIC supratentorial
- HTIC, durée, variabilité, PPC réduite
 ➔ mauvais! (évolution et mortalité)
- Osmothérapie prophylactique
 - Mannitol ➔ pas de bénéfices
 - Salin 3% ➔ pas de données
- Principes de tx calqués sur TCC...
 - Monitoring si GCS 3-8
 - Tx si PIC ≥ 22
 - PPC 50-70 selon autorégulation

COR	LOE	Recommendations
1	B-NR	1. In patients with spontaneous ICH or IVH and hydrocephalus that is contributing to decreased level of consciousness, ventricular drainage should be performed to reduce mortality. ³⁴⁷⁻³⁵⁰
2b	B-NR	2. In patients with moderate to severe spontaneous ICH or IVH with a reduced level of consciousness, ICP monitoring and treatment might be considered to reduce mortality and improve outcomes. ^{159,351-356}
2b	B-NR	3. In patients with spontaneous ICH, the efficacy of early prophylactic hyperosmolar therapy for improving outcomes is not well established. ³⁶⁷⁻³⁶¹
2b	C-LD	4. In patients with spontaneous ICH, bolus hyperosmolar therapy may be considered for transiently reducing ICP. ³⁶²⁻³⁶⁴
3: No Benefit	B-R	5. In patients with spontaneous ICH, corticosteroids should not be administered for treatment of elevated ICP. ³⁶⁵⁻³⁶⁹

Convulsions et anticonvulsivants

- Incidence de convulsions 2,8-28%
- Risque plus élevé – premières 24h
- Monitoring EEG 24h probablement raisonnable
 - Plus long si comateux?
 - 28% détectées dans 1ères 24h
 - 94% détectées avec monitoring 48h
- Incertitude d'implication pronostique d'un tracé anormal
 - Dx difficile, contexte clinique essentiel

COR	LOE	Recommendations
1	C-LD	1. In patients with spontaneous ICH, impaired consciousness, and confirmed electrographic seizures, antiseizure drugs should be administered to reduce morbidity. ^{325,326}
1	C-EO	2. In patients with spontaneous ICH and clinical seizures, antiseizure drugs are recommended to improve functional outcomes and prevent brain injury from prolonged recurrent seizures.
2a	C-LD	3. In patients with spontaneous ICH and unexplained abnormal or fluctuating mental status or suspicion of seizures, continuous electroencephalography (≥ 24 hours) is reasonable to diagnose electrographic seizures and epileptiform discharges. ³²⁷
3: No Benefit	B-NR	4. In patients with spontaneous ICH without evidence of seizures, prophylactic antiseizure medication is not beneficial to improve functional outcomes, long-term seizure control, or mortality. ³²⁸⁻³³¹

Safety and efficacy of prophylactic levetiracetam for prevention of epileptic seizures in the acute phase of intracerebral haemorrhage (PEACH): a randomised, double-blind, placebo-controlled, phase 3 trial

Laure Peter-Derex, Frédéric Philippeau, Pierre Garnier, Nathalie André-Obadia, Sébastien Boulogne, Hélène Catenaix, Philippe Convers, Laure Mazzola, Michel Gouttard, Maud Esieban, Julia Fontaine, Laura Mechouff, Elodie Ong, Tae-Hee Cho, Norbert Nighoghossian, Nathalie Perretton, Anne Termoz, Julie Haesebaert, Anne-Marie Schott, Muriel Rabilloud, Christine Pivot, Carole Dhelens, Andrea Filip, Yves Berthézyne, Sylvain Rheims, Florent Boutillie, Laurent Derex

Lancet Neurol 2022; 21: 789-91

- 50/104 pts (interrompue – arrêt financement, faible recrutement COVID)
- HIC non-traumatique < 24h → LEV 500 BID vs placebo x 6 sem
- Groupe placebo désavantagé (HIC 9 vs 18 mL, lobaire vs profond, NIHSS 8 vs 13)
- EEG continu précoce (24-72h post HIC)
- Installé ~ 25h post HIC, 1^{ère} crise documentée ~ 47h
- Toutes les crises = électriques

	Levetiracetam group (n=24)	Placebo group (n=26)	Effect size (95% CI)	p value
Primary efficacy outcome				
Clinical or electrographic seizure in the first 72 h after inclusion	3/19 (16%)	10/23 (43%)	0.16 (0.03 to 0.94)*	0.043
Secondary efficacy outcomes				
Number of seizures on cEEG	6	158	0.07 (0.01 to 0.38)†	0.0021
Median duration of seizures on cEEG in all patients, s	0 (0-0)‡; n=19	0 (0-540)‡; n=23	..	0.023
Median duration of seizures on cEEG in patients with seizures, s	67 (46-300); n=3	780 (380-1980); n=10	..	0.028

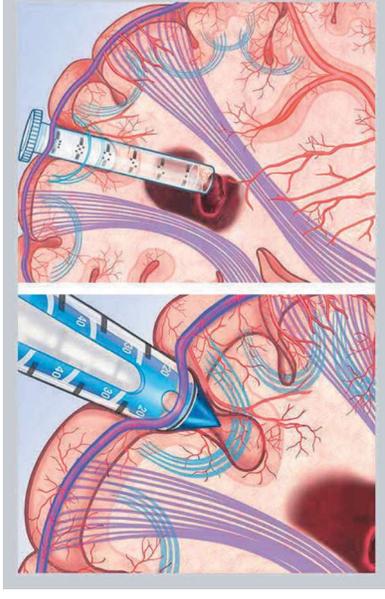
Traitements chirurgicaux

Recommendations for MIS Evacuation of ICH Referenced studies that support recommendations are summarized in Data Supplements 55 and 56.		
COR	LOE	Recommendations
2a	B-R	1. For patients with supratentorial ICH of >20- to 30-mL volume with GCS scores in the moderate range (5–12), minimally invasive hematoma evacuation with endoscopic or stereotactic aspiration with or without thrombolytic use can be useful to reduce mortality compared with medical management alone. ^{379–388}
2b	B-R	2. For patients with supratentorial ICH of >20- to 30-mL volume with GCS scores in the moderate range (5–12) being considered for hematoma evacuation, it may be reasonable to select minimally invasive hematoma evacuation over conventional craniotomy to improve functional outcomes. ^{382,383,385–387,389,390}
2b	B-R	3. For patients with supratentorial ICH of >20- to 30-mL volume with GCS scores in the moderate range (5–12), the effectiveness of minimally invasive hematoma evacuation with endoscopic or stereotactic aspiration with or without thrombolytic use to improve functional outcomes is uncertain. ^{379–385,387,388}

- MISTIE III (Lancet 2019) Cathéter CMI + tPA, HIC supratentorielle ≥ 30 mL
 - Aucun bénéfice mRS (primaire)
 - Réduction de mortalité (outcome secondaire)
 - Sous-groupe HIC < 15 mL post-procédure → amélioration mRS

• **ENRICH (NEJM 2024) MIPS ≤ 24h en HIC 30-80 mL NGC + lobaire**

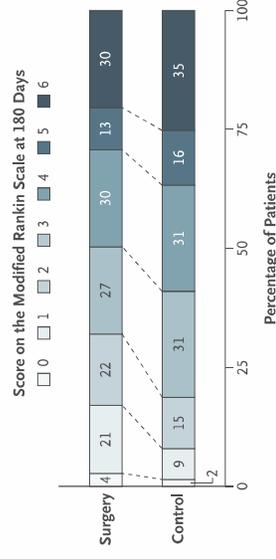
- CMI en centre d'expertise pour appliquer ces recommandations...
- Meilleure sélection de patients?



Trial of Early Minimally Invasive Removal of Intracerebral Hemorrhage

G. Pradilla, J.J. Ratcliff, A.J. Hall, B.R. Saville, J.W. Allen, G. Paulon, A. McGlothlin, R.J. Lewis, M. Fitzgerald, A.F. Caveney, X.T. Li, M. Bain, J. Gomes, B. Jankowitz, G. Zenonos, B.J. Molyneaux, J. Davies, A. Siddiqui, M.R. Chicoine, S.G. Keyrouz, J.A. Grossberg, M.V. Shah, R. Singh, B.N. Bohnstedt, M. Frankel, D.W. Wright, and D.L. Barrow, for the ENRICH trial investigators*

- **MIPS = minimally invasive trans-sulcal para-fascicular surgery (tractography)**
- **HIC supratentorielle spontanée 30-80 mL (55), lobaire ou NGC antérieurs, LKW-tx < 24h**
- **Bénéfice évolution fonctionnelle UW-mRS (lobaire) et mRS ≤ 3 à 180 jrs**
- **Diminution mortalité et durée séjour USI + hospitalisation ~ 3 jours**
- **Diminution convulsions et craniectomie décompressive (3 vs 20%)**



Traitements chirurgicaux

Recommendations for Craniotomy for Supratentorial Hemorrhage Referenced studies that support recommendations are summarized in Data Supplements E3 and E4.	
COR	LOE Recommendations
2b	A 1. For most patients with spontaneous supratentorial ICH of moderate or greater severity, the usefulness of craniotomy for hemorrhage evacuation to improve functional outcomes or mortality is uncertain. ^{380,382,384,393,420–431}
2b	C-LD 2. In patients with supratentorial ICH who are deteriorating, craniotomy for hematoma evacuation might be considered as a lifesaving measure. ^{382,384,429,432}
Recommendations for Craniotomy for ICH Referenced studies that support recommendations are summarized in Data Supplements E5 through E7.	
COR	LOE Recommendations
2b	C-LD 1. In patients with supratentorial ICH who are in a coma, have large hematomas with significant midline shift, or have elevated ICP refractory to medical management, decompressive craniectomy with or without hematoma evacuation may be considered to reduce mortality. ^{453–460}
2b	C-LD 2. In patients with supratentorial ICH who are in a coma, have large hematomas with significant midline shift, or have elevated ICP refractory to medical management, effectiveness of decompressive craniotomy with or without hematoma evacuation to improve functional outcomes is uncertain. ^{459–462}

- STITCH (Lancet 2005) HIC supratentorielle: chx < 24h vs conservateur
- STITCH II (Lancet 2013) HIC superficielle lobaire: chx < 12h rando vs conservateur
 - Aucun bénéfice sur GOS-E
 - Bénéfice pour patients jugés à mauvais Px (GCS/âge/taille HIP)?
 - Taux élevé de croisement ~ 20%
 - Bénéfice d'une chirurgie plus précoce (< 12 hrs du saignement)?

• SWITCH (Lancet 2024) CD pour HIC profonds 30-100 mL

- Fosse postérieure: aucune ERC, manque d'équipose?

Recommendations for Craniotomy for Posterior Fossa Hemorrhage Referenced studies that support recommendations are summarized in Data Supplement E8.		
COR	LOE	Recommendation
1	B-NR	1. For patients with cerebellar ICH who are deteriorating neurologically, have brainstem compression and/or hydrocephalus from ventricular obstruction, or have cerebellar ICH volume ≥ 15 mL, immediate surgical removal of the hemorrhage with or without EVD is recommended in preference to medical management alone to reduce mortality. ^{442–444}

**Decompressive craniectomy plus best medical treatment
versus best medical treatment alone for spontaneous
severe deep supratentorial intracerebral haemorrhage:
a randomised controlled clinical trial**

Lancet 2024; 403: 2395–404

Jürgen Beck, Christian Fung, Daniel Strbian, Lukas Bütikofer, Werner J Z'Graggen, Matthias F Lang, Seraina Beyeler, Jan Gralla, Florian Ringel, Karl Schaller, Nikolaus Plesnila, Marcel Arnold, Werner Hacke, Peter Juni, Alexander David Mendelow, Christian Stapf, Rustam Al-Shahi Saliman, Jenny Bressan, Stefanie Lerch, Arsany Hakim, Nicolas Martinez-Magander, Anna Pippo-Karjalainen, Peter Vajkoczy, Stefan Wolf, Gerrit A Schubert, Anke Höllig, Michael Veideman, Roland Roelz, Andreas Gruber, Philip Rauch, Dorothee Mielke, Veit Rohde, Thomas Kerz, Eberhard Uhl, Enea Thanasi, Hagen B Huttner, Bernd Kallmünzer, L Jaap Kappelle, Wolfgang Densberger, Christian Roth, Robin Lemmens, Jan Leppert, Jose L Sammillan, Jonathan M Coutinho, Katharina A M Hackenberg, Gernot Reimann, Mikael Mazighi, Claudio L A Bassetti, Heinrich P Mattle, Andreas Raabe, Urs Fischer, on behalf of the SWITCH study investigators*

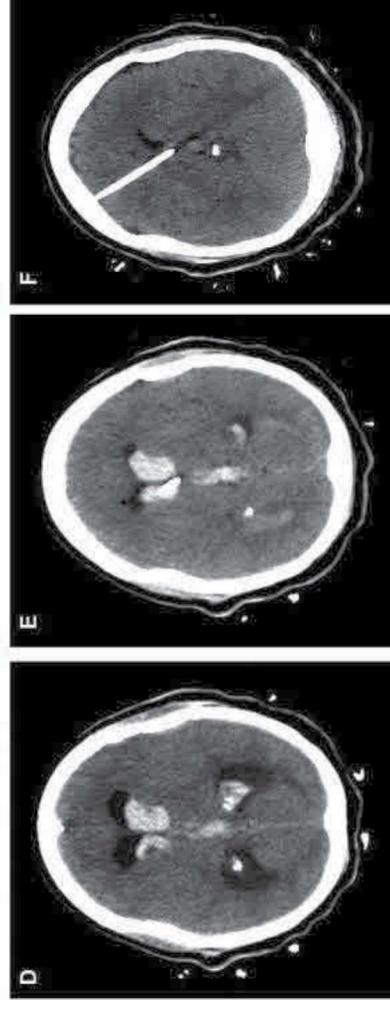
- **HIC profond (NGC/thalamus) sévère (NIHSS \geq 10, volume \geq 30 mL, GCS $<$ 14), CHX $<$ 72 h**
- **197/300 pts (2014-2023)**
- **NIHSS ~ 18, volume 55-59 mL, IVH 30%,**
- **\downarrow mRS 5-6 à 180 jrs 44 vs 58% aRR 0.77 (0.59-1.01)**

- **(Interrompue manque de financement)**

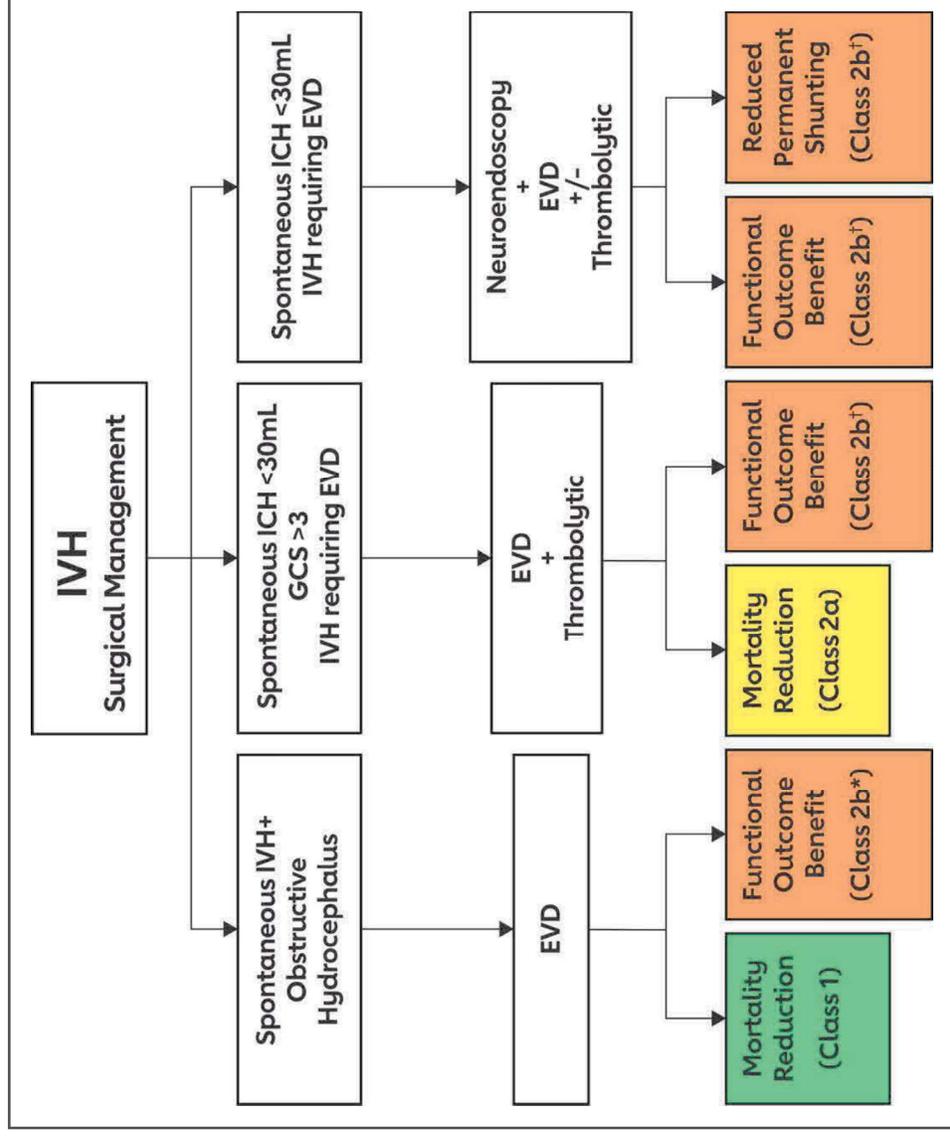
Traitements chirurgicaux

Recommendations for MIS Evacuation of IVH Referenced studies that support recommendations are summarized in Data Supplements B7 through G.		
COR	LOE	Recommendations
1	B-NR	1. For patients with spontaneous ICH, large IVH, and impaired level of consciousness, EVD is recommended in preference to medical management alone to reduce mortality. ³⁴⁷⁻³⁴⁹
2a	B-R	2. For patients with a GCS score >3 and primary IVH or IVH extension from spontaneous supratentorial ICH of <30-mL volume requiring EVD, minimally invasive IVH evacuation with EVD plus thrombolytic is safe and is reasonable compared with EVD alone to reduce mortality. ⁴¹⁵⁻⁴¹⁶
2b	B-R	3. For patients with a GCS score >3 and primary IVH or IVH extension from spontaneous supratentorial ICH of <30-mL volume requiring EVD, the effectiveness of minimally invasive IVH evacuation with EVD plus thrombolytic use to improve functional outcomes is uncertain. ^{392,407,415-419}
2b	B-NR	4. For patients with severe spontaneous ICH, large IVH, and impaired level of consciousness, the efficacy of EVD for improving functional outcomes is not well established. ³⁴⁷⁻³⁴⁹
2b	C-LD	5. For patients with spontaneous supratentorial ICH of <30-mL volume and IVH requiring EVD, the usefulness of minimally invasive IVH evacuation with neuroendoscopy plus EVD, with or without thrombolytic, to improve functional outcomes and reduce permanent shunt dependence is uncertain. ^{419,420}

- Déversement intraventriculaire chez 30-50% HIC
- CLEAR III (Lancet 2017) tPA via DVE en HIC ≤ 30 mL stable avec HIV
 - Aucun bénéfice mRS
 - Diminution mortalité (mais plus de mRS 5)
 - Moins de ventriculites, DVP idem
 - Saignement symptomatique idem (~2%)
 - ~ 60% HIC thalamique → moins bon pronostic
- Meilleure sélection de patients?



Traitements chirurgicaux



Conclusion

- Peu d'interventions démontrant bénéfiques sur évolution neurologique
 - Validité externe douteuse chez population chirurgicale (HTIC, taille d'hématome)
 - Réduction agressive de pression artérielle
 - Renversement de coagulopathie
 - Thérapies hémostatiques
- Utilité d'approches chirurgicales demeurent mal définies
 - Détérioration neurologique
 - HTIC réfractaire, HIP volumineux, patient comateux
 - Identification de sous-groupe de patients ayant bénéfice
- Considérations anesthésiques demeurent relativement non-spécifiques
 - Urgence, patient instable – ABC (considérer estomac plein)
 - Anticiper et traiter HTIC, convulsions, insultes secondaires...
 - Maintenir PPC adéquate (considérer altération d'autorégulation cérébrale)
 - Éviter hypertension extrême (validité de cible INTERACT2, ATACH-2 douteuse)
 - Réduire PIC – algorithme HTIC/relaxation cérébrale (TIVA, osmothérapie, +/- hyperventilation si engagement imminent) (cours HTIC)
 - Renverser coagulopathie
 - Considérer atteintes extracrâniennes (CMP, IM, œdème pulmonaire)
 - Considérer co-morbidités associées

Hémorragie sous-arachnoïdienne

AHA/ASA GUIDELINE

2023 Guideline for the Management of Patients With Aneurysmal Subarachnoid Hemorrhage: A Guideline From the American Heart Association/American Stroke Association

Stroke. 2023;54:e314–e370.

NCS GUIDELINES

Guidelines for the Neurocritical Care Management of Aneurysmal Subarachnoid Hemorrhage

Miriam M. Treggiari^{1*}, Alejandro A. Rabinstein², Katharina M. Busi³, Meghan M. Caylor¹¹, Giuseppe Citerio^{12,13}, Steven Deem⁴, Michael Diringier⁵, Elizabeth Fox⁶, Sarah Livesay⁷, Kevin N. Sheth⁸, Jose I. Suarez⁹ and Stavropoula Tjoumakaris¹⁰

Neurocrit Care (2023) 39:1–28

“there is insufficient evidence”

Picetti et al. *Journal of Anesthesia, Analgesia and Critical Care*
<https://doi.org/10.1186/s44158-022-00042-x>

(2022) 2:13



ORIGINAL ARTICLE

Open Access

Early management of patients with aneurysmal subarachnoid hemorrhage in a hospital with neurosurgical/neuroendovascular facilities: a consensus and clinical recommendations of the Italian Society of Anesthesia and Intensive Care (SIAARTI)–Part 1



Delphi – recommandations d’experts

Hémorragie sous-arachnoïdienne

- HSA non-traumatiques (~ 5-10% AVC)
 - Incidence mondiale variable 9-23/100,000 JPFI
 - Population typiquement plus jeune (40-60 ans)
 - Origine **anévrismale** chez 80%, autres causes non-anévrysmales (MAV, idiopathique, etc)
 - Morbidité/mortalité élevée
 - Mortalité 15% avant admission, 30-40% à 30 jours
 - Morbidité/mortalité augmente selon sévérité de l'atteinte initiale, re-saignement, DCI, âge et comorbidités
- Anévrysme intracrânien 1-2%
 - Habituellement asymptomatiques
 - Circulation antérieure > 80%
 - Sacculaire 90%, fusiforme 10%
 - Multiples anévrysmes 35%
- FR anévrysme: femmes, HTA, tabagisme, ROH, reins polykystiques, syndrome Ehlers-Danlos, syndrome Marfan, NF type 1, dysplasie fibromusculaire
- FR rupture: femmes, HTA, tabagisme, âge avancé, ROH, drogues sympathomimétiques, origines (afroaméricains, hispanoaméricains, JP, FI), caractéristiques d'anévrysme (circulation postérieure, > 7-10 mm, anévrysmes géants > 20-25 mm)

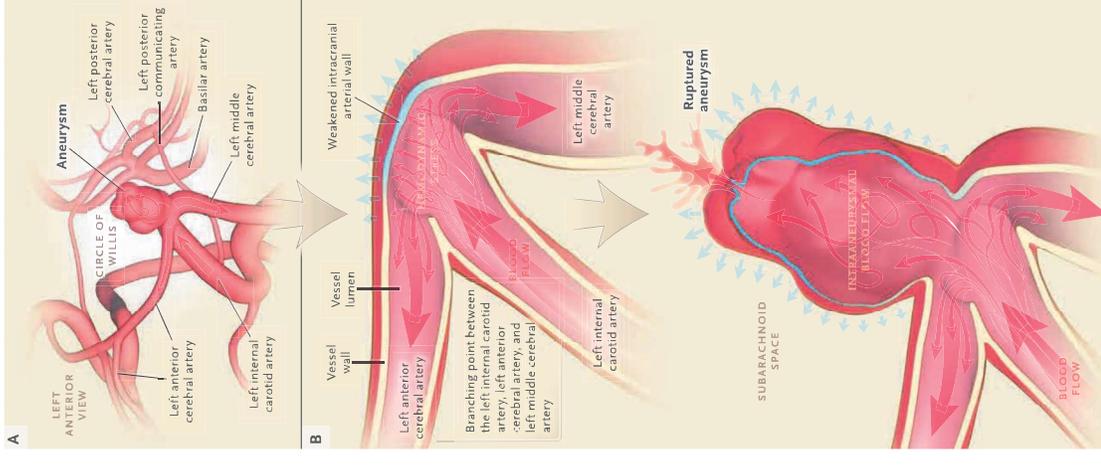


TABLE 2. Clinical Features of Intracranial Aneurysms Based on Location

Location of the Aneurysm	Clinical Features
Anterior communicating artery	Bilateral temporal hemianopsia Bilateral lower extremity weakness
Posterior communicating artery	Third nerve palsy
Intercavernous internal carotid artery	Facial or orbital pain Epistaxis Progressive vision loss and/or ophthalmoplegia
Posterior circulation aneurysms	Brainstem dysfunction

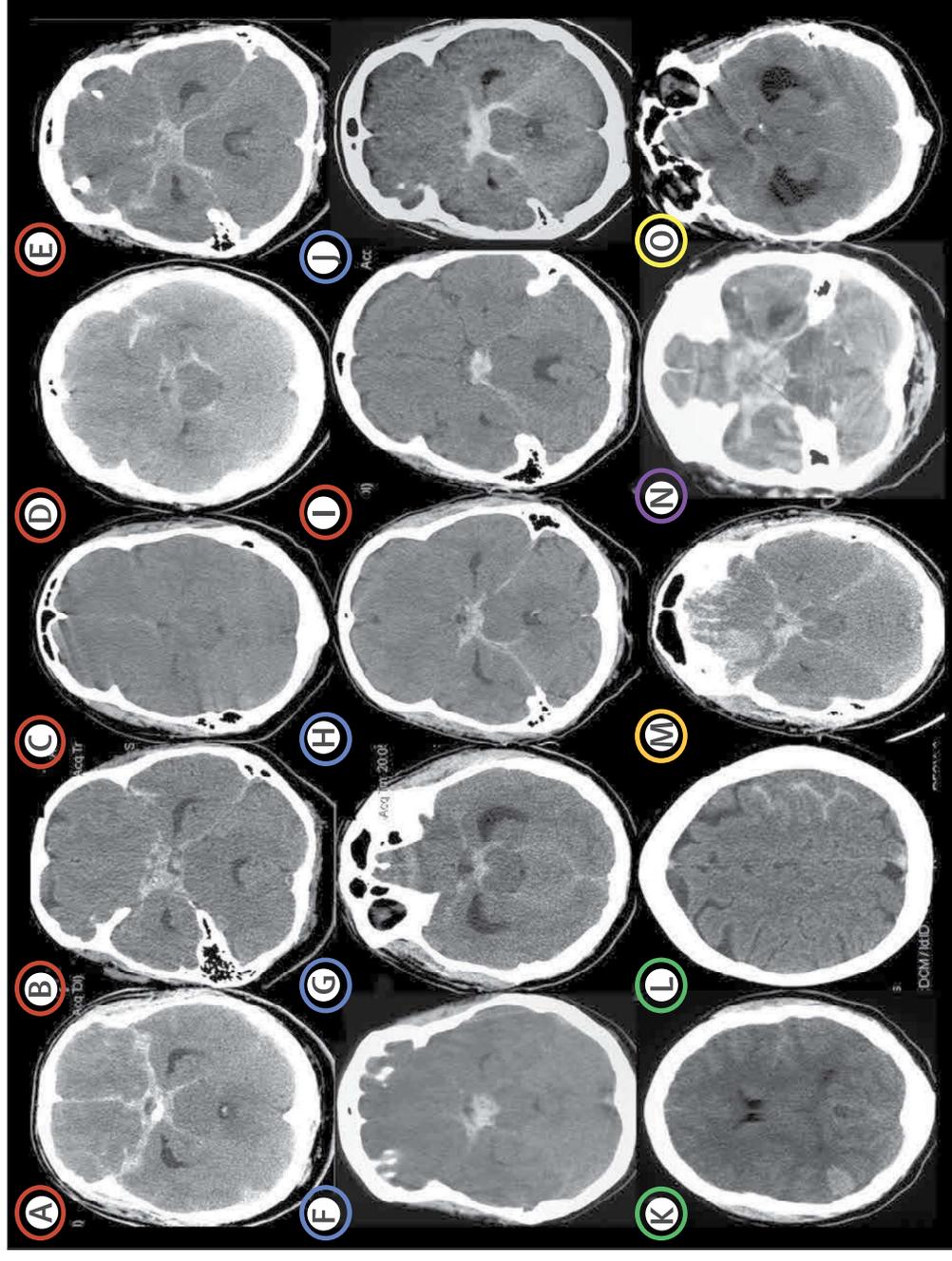
HSA: trajectoire intra-hospitalière typique

- **Présentation immédiate**
 - Diagnostic HSA anévrysmale
 - Stabilisation initiale
- **Pré-sécurisation**
 - Prévenir re-saignement
 - Sécurisation précoce, contrôle pression artérielle
 - Prévenir dommage cérébral secondaire
 - DVE, anticonvulsivants, complications intra/extracrânienne
- **Post-sécurisation**
 - Anticiper et prévenir dommage cérébral secondaire
 - Anticiper et traiter ischémie cérébrale retardée
- **Plus tardivement**
 - DVP, remise volet
 - Sevrage trachéostomie
 - Réadaptation et orientation

Étiologie

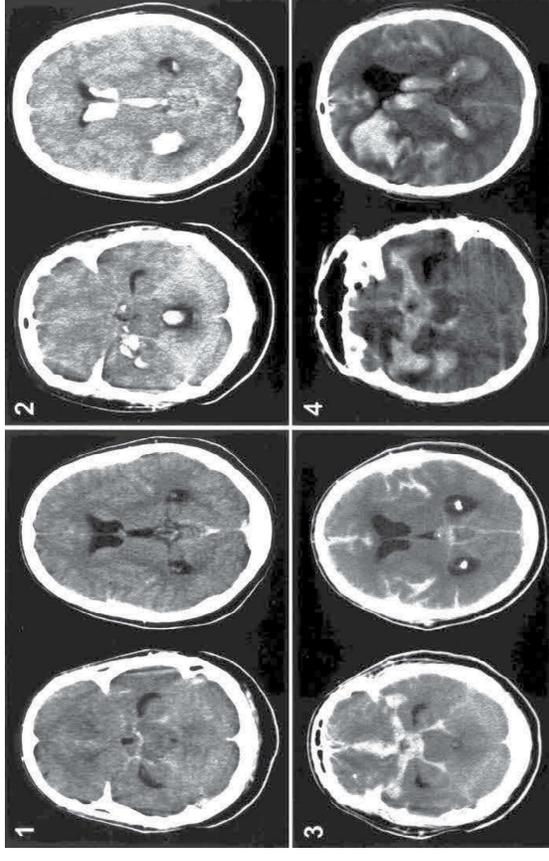
- HSA anévrysmale
- Périmesencéphalique
- Thrombose veineuse
- Trauma
- Apoplexie hypophysaire
- Pseudo-HSA

- Distribution du sang**
 - Citerne de la base
 - Vallée sylvienne
 - Citerne interpédonculaire
 - Sillon interhémisphérique
- OUI, mais...**
CTA, DSA nécessaire!



Présentation clinique et pronostication

- Céphalée brutale (thunderclap) ad décès
 - 10-40% céphalée « sentinelle » 2-8 sem pré-HSA
 - Altération de l'état de conscience
 - Déficit neurologique focal
 - Convulsions ad 20%
- Échelles pronostiques
 - Cliniques (Hunt and Hess, WFNS)
 - Radiologique (Fisher modifié, Hijdra score)
 - Combinés (VASOGRADE)



VASOGRADE	WFNS	mFisher	OR DCI
Green	1-2	1-2	
Yellow	1-3	3-4	1.31 (0,77-2,23)
Red	4-5	any	3.19 (2,07-4,50)

Letters

Claassen et al. Stroke 2001
 Hijdra et al. Stroke 1990
 Teasdale et al. J Neurol Neurosurg Psychiatr 1988
 de Oliveira Manoel et al. Stroke 2015

Présentation clinique et pronostic

Grade #	Hunt and Hess ⁴¹	WFNS ⁴²	mRS 4-6
Grade #1	Asymptomatic or mild headache and slight nuchal rigidity	GCS 15	15%
Grade #2	Moderate to severe headache, nuchal rigidity, no focal neurological deficit other than cranial nerve palsy	GCS 14–13 without major focal deficit (aphasia or hemiparesis/hemiplegia)	30%
Grade #3	Confusion, lethargy, or mild focal neurological deficit other than cranial nerve palsy	GCS 14–13 with major focal deficit	50%
Grade #4	Stupor or moderate to severe hemiparesis	GCS 12–7 with or without major focal deficit	60%
Grade #5	Coma, extensor posturing, moribund appearance	GCS 6–3 with or without major focal deficit	90%

Autres caractéristiques défavorables

- Resaignement
- Âge
- Comorbidités
- Œdème cérébral
- HIV/HIP
- DCI
- Complications extra-cérébrales

Présentation clinique et pronostication

Fisher scale ⁴⁴	Claassen scale ⁴⁶	Modified Fisher scale ⁴⁷	Ischémie cérébrale retardée
Grade #0	No SAH or intraventricular haemorrhage	No SAH or intraventricular haemorrhage	
Grade #1	No SAH or intraventricular haemorrhage	Minimum or thin SAH, no intraventricular haemorrhage in either lateral ventricle	12%
Grade #2	Diffuse deposition of thin layer with all vertical layers of blood (interhemispheric fissure, insular cistern, ambient cistern) <1 mm thick	Minimal or thin SAH, with intraventricular haemorrhage in both lateral ventricles	21%
Grade #3	Vertical layers of blood ≥ 1 mm thick or localised clots (clots defined as $>3 \times 5$ mm)	Thick SAH completely filling two or more cisterns or fissures, no intraventricular haemorrhage in both lateral ventricles	19%
Grade #4	Diffuse or no subarachnoid blood, but with intracerebral or intraventricular clots	Thick SAH completely filling two or more cisterns or fissures, with intraventricular haemorrhage in both lateral ventricles	40%

The severity of subarachnoid and intraventricular haemorrhage on the initial CT scan is the most important factor predicting delayed cerebral ischaemia and cerebral infarction and is a prognostic factor for outcome.⁴⁷ Qualitative, semiquantitative, and quantitative scales have been proposed to measure the extent of damage.^{49,50} SAH=subarachnoid haemorrhage.

Table 2: Scales used to determine severity of SAH and intraventricular haemorrhage using the Fisher, Claassen, Modified Fisher, and Hijdra scales

Pronostic: HSA « bas grades »

- **Données prospectives (ISAT) – 88% « bas grades »**
 - mRS 0-2 → 73%
 - mRS 3 → 12%
 - mRS 4-5 → 6.5%
 - mRS 6 → 9%
- Mais, limites des échelles d'évaluation pronostique...
 - Dysfonction cognitive ~ 20%
 - Troubles de l'humeur
 - Trouble du sommeil
 - Troubles de comportement
 - Répercussions sociales
 - Qualité de vie

Pronostic: HSA « hauts grades »

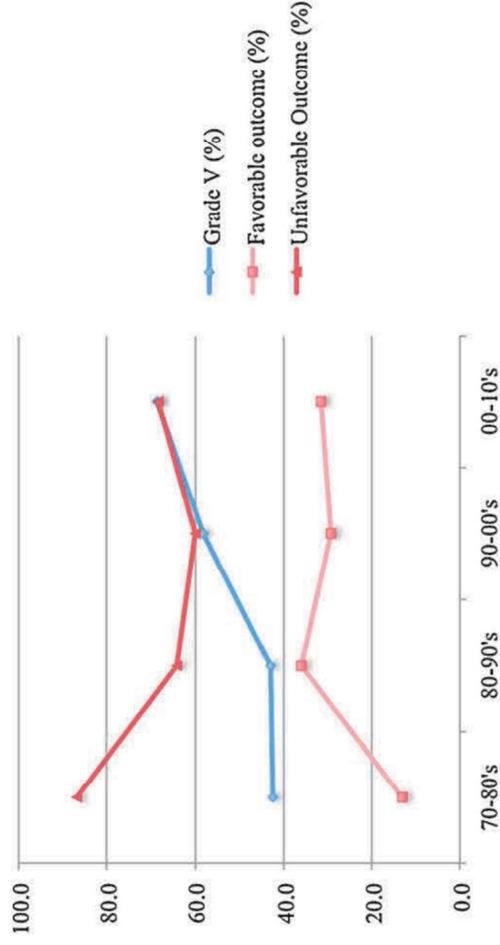


Fig. 3 Chart of favorable versus unfavorable functional outcome. This chart shows the functional outcome trends according to different decades of patients' enrollment in the included articles. Also, the percentage of Grade 5 patients included was plotted, showing a trend to increase inclusion of Grade 5 patients. The definition of favorable outcome varies according to study, including GOS 4-5, mRS 0-2, ability to live independently (for full detail, please refer to Table 2)

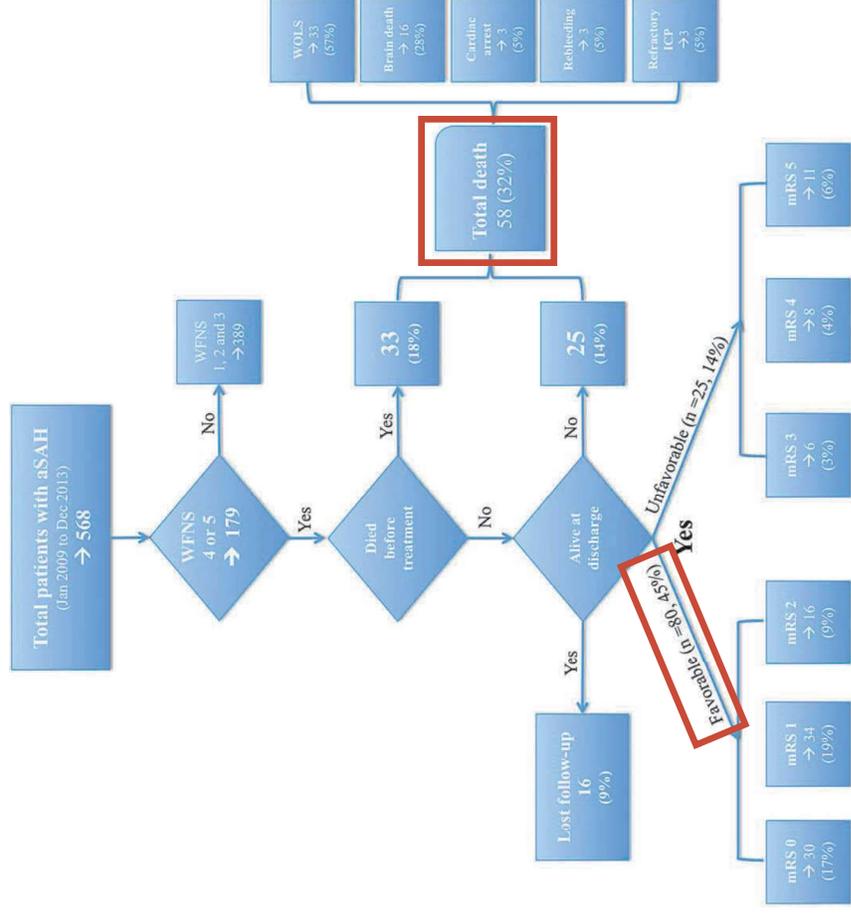
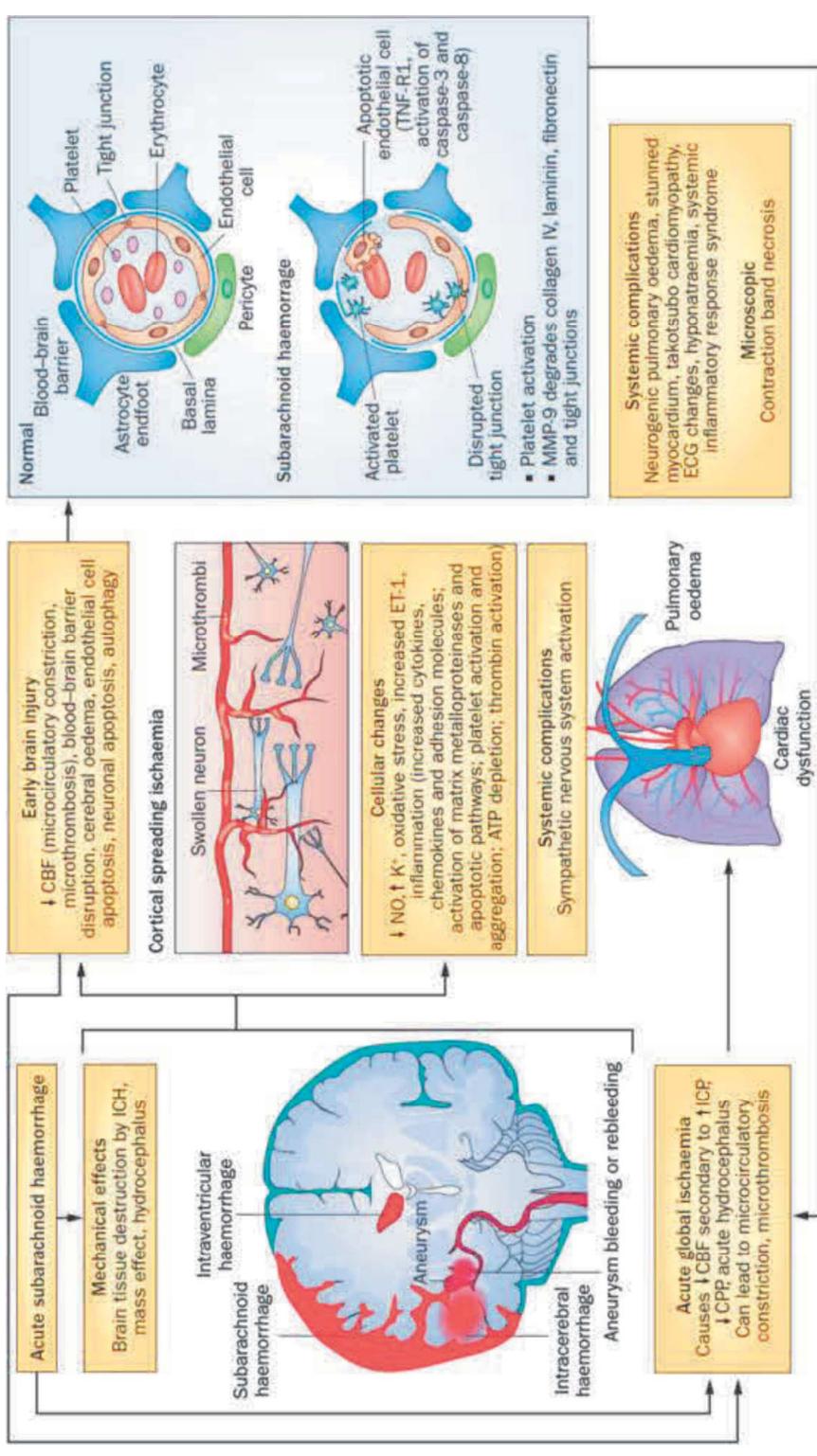


Fig. 1 Flowchart of the entire cohort admitted from January 2009 to December 2013. aSAH: aneurysmal subarachnoid hemorrhage, WOLS: withdrawal of life support, WFNS: World Federation of Neurosurgical Societies, ICP: intracranial pressure, mRS: modified Rankin Scale

Domage cérébral précoce (early brain injury)

- **EBW = J0-J3**
- **↑↑ brutale de PIC → hypoperfusion globale transitoire**
- Sang sous-arachnoïdien
- Re-saignement
- Contribution de complications extracrâniennes
 - Activation adrénergique
 - Vasoconstriction microcirculation
 - Microthromboses
 - Altération BHE
 - Œdème cytotoxique
 - Œdème vasogénique
 - **Altération autorégulation cérébrale**
- **↑ secondaire de la PIC**
 - HCP (communicante, non-communicante)
 - Effet de masse (HIP, HSD)
 - Œdème cérébral



Prévenir resaignement

- Resaignement jusqu'à 20% dans 1^{ers} 72 hrs → mortalité 20-70%
 - 50-90% dans les 1ers 6 hrs
 - FR: grade élevé, inconscience ou convulsions à l'ictus, gros anévrismes, saignement sentinél, HIP, HIV, possiblement HTA et antiplaquettaires
- **Sécurisation le plus tôt possible (< 72 hrs)**
 - Ultra rapide (< 24 hrs) vs rapide (24-72 hrs) – bénéfiques incertains, stabiliser pt avant
 - **LIGNES DIRECTRICES ≤ 24 hrs**
 - « As early as feasible » « as early as logistically and technically possible »
 - Modalité thérapeutique: « clip vs coil? »
 - Transfert dans un centre d'expertise (high-volume > 35 aSAH/an)

AHA/ASA Hoh et al. Stroke 2023
Oudshoorn et al. Neurocrit Care 2014

Naidech et al. Arch Neurol 2005
Lawton et al. NEJM 2017

Muehlschlegel et al. Continuum 2018

Macdonald RL et al. Lancet 2017

D'Souza S. J Neurosurg Anesthesiol 2015

Prévenir resaignement: pression artérielle

- Limiter poussées hypertensives
 - ASA/AHA 2012 → PAsyst < 160 mmHg (Classe IIa, Niveau C)
 - NCS 2011 → PAsyst < 160 mmHg, PAM < 110 mmHg
 - ESO 2013 → PAsyst < 180 mmHg, PAM > 90 mmHg
 - SIAARTI 2022 → PAsyst entre 120-160 mmHg

NCS 2023
There is insufficient evidence to recommend a blood pressure reduction goal for the treatment of hypertension before aneurysm treatment in aSAH. Lack of evidence to recommend a specific blood pressure reduction goal does not necessarily imply that blood pressure reduction is not helpful before aneurysm treatment*



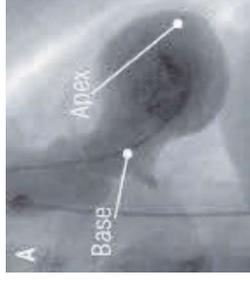
Prévenir resaignement: pression artérielle

COR	LOE	Recommendations
1	C-EO	1. In patients with aSAH and unsecured aneurysm, frequent blood pressure (BP) monitoring and BP control with short-acting medication(s) is recommended to avoid severe hypotension, hypertension, and BP variability.
1	C-EO	2. In patients with aSAH who are receiving anticoagulants, emergency anticoagulation reversal with appropriate reversal agents should be performed to prevent rebleeding.
3: No benefit	A	3. In patients with aSAH, routine use of anti-fibrinolytic therapy is not useful to improve functional outcome. ⁸⁵⁻⁸⁷

- Manque de données pour recommander cible précise
- Variabilité PA associée à évolution défavorable
- ↓ PA excessive pourrait compromettre PPC
- Réduction graduelle d’HTA excessive (> 180-200 mmHg)
- Éviter hypoTA (PAM < 65 mmHg)
- Surveillance neurologique
- À considérer: PA à la présentation, oedème cérébral, HCP, HTA chronique, insuffisance rénale

Complications extra-crâniennes

- Effets cardiaques
 - Changements ECG (Δ ST-T, prolongation QT, ondes Q, ondes U)
 - Élévation de biomarqueurs (troponines, NT-proBNP)
 - Dysrythmies (supraventriculaires, ventriculaires)
 - Dysfonction myocardique – dysfonction globale, ARC
 - STEMI / NSTEMI type I surajouté
 - NSTEMI type II 2^o stress adrénergique
 - Changement ECG sans infarctus
 - Cardiopathie de stress (“axe neuro-cardiaque”)
 - Poussée adrénergique (haut grade)
 - Tachycardie sinusale, changements ST-T
 - Élévation importante des biomarqueurs
 - Hypotension inhabituelle... 🙄



Complications extra-crâniennes

- Complications respiratoires
 - Œdème pulmonaire (neurogénique, cardiogénique, ARDS)
 - Aspiration / surinfection
- Volémie et électrolytes
 - Hypovolémie – polyurie (CSW, HTA, osmothérapie)
 - Dysnatrémie (CSW, SIADH >> DI; osmothérapie)
 - HypoMg*
 - HypoK, HypoCa
- Autres
 - Hyperthermie – viser normothermie, éviter frissons
 - Hyperglycémie – viser normoglycémie
 - Infections, sepsis
 - **Anémie – SaHARA, TRAIN**
 - TVP et thromboembolies

COR	LOE	Recommendations
Pulmonary management		
1	B-NR	1. In patients with aSAH who require mechanical ventilation for >24 hours, implementation of a standardized ICU care bundle is recommended to reduce the duration of mechanical ventilation and hospital-acquired pneumonia. ^{172,178}
2b	B-NR	2. In patients with aSAH who develop severe acute respiratory distress syndrome (ARDS) and life-threatening hypoxemia, rescue maneuvers such as prone positioning and alveolar recruitment maneuvers with ICP monitoring may be reasonable to improve oxygenation. ¹⁷⁹⁻¹⁸²
Intravascular volume and electrolyte management		
2a	B-R	3. In patients with aSAH, close monitoring and goal-directed treatment of volume status are reasonable to maintain euvolemia. ¹⁸³⁻¹⁸⁵
2a	B-R	4. In patients with aSAH, use of mineralocorticoids is reasonable to treat natriuresis and hyponatremia. ^{186,187}
3: Harm	B-R	5. In patients with aSAH, induction of hypervolemia is potentially harmful because of the association with excess morbidity. ¹⁸⁸⁻¹⁹⁰
Other		
1	C-LD	6. In patients with aSAH whose ruptured aneurysm has been secured, pharmacological or mechanical venous thromboembolism (VTE) prophylaxis is recommended to reduce the risk for VTE. ¹⁹¹⁻¹⁹³
2a	B-NR	7. In patients with aSAH, effective glycemic control, strict hyperglycemia management, and avoidance of hypoglycemia are reasonable to improve outcome. ^{141,142,194,195}
2b	C-LD	8. In patients with aSAH with fever refractory to antipyretic medications, the effectiveness of therapeutic temperature management (TTM) during the acute phase of aSAH is uncertain. ¹⁹⁶⁻¹⁹⁸

Seuil transfusionnel en HSA

- 3 RCT multicentrique qui remettent en cause seuil restrictif
- **TRAIN Taccone et al. JAMA 2024**
 - 850 pts (22% HSA), seuil transfusionnel < 90 vs < 70
 - **Évolution défavorable (GOSE 1-5) 63% vs 73%**
- **SAHARA English et al. NEJM 2024**
 - 742 pts (100% HSA), seuil transfusionnel < 100 vs < 80
 - Évolution défavorable (mRS > 4) 34 vs 38% NS
 - Nouvel infarctus cérébral 16 vs 18% NS
 - DCI 18 vs 20% NS
- **Vasospasme radiologique 32% vs 41%**

Taccone et al. Intensive Care Med 2025
SAHARA English et al. NEJM 2024
TRAIN Taccone et al. JAMA 2024
HEMOTION Turgeon et al. NEJM 2024

International Subarachnoid Aneurysm Trial (ISAT)

	ISAT (Lancet 2002)
Devis	RCT multicentrique pragmatique (RU, Suède, Finlande, Canada, Allemagne, France, Australie, Danemark, Suisse, ÉUA) 1994-2002
Patients	2143 / 2500
Inclusion	HSA anévrysmale Traitement envisagé Équipoise p/r modalité tx
Exclusion	HSA > 28 jrs 1 ou 2 tx inappropriés
Traitement	Clip vs coil
Issue primaire	mRS ≥ 3 à 1 an

78% exclus (validité externe?)

	Endovascular treatment (n=1073)	Neurosurgery (n=1070)
Male sex	400 (37%)	399 (37%)
Age (years) *	52 (44–60, 18–87)	52 (43–60, 18–84)
WFNS grade		
1	674 (63%)	661 (62%)
2	269 (25%)	280 (26%)
3	66 (6%)	68 (6%)
4	38 (4%)	36 (3%)
5	11 (1%)	9 (1%)
6 (not assessable)†	15 (1%)	16 (1%)
Maximum target aneurysm lumen size (mm)		
≤5	552 (51%)	572 (53%)
6–10	438 (41%)	426 (40%)
≥11	83 (8%)	72 (7%)
Number of aneurysms detected		
1	836 (78%)	850 (79%)
2	173 (16%)	170 (16%)
3	44 (4%)	35 (3%)
≥4	20 (2%)	15 (1%)
Time between SAH and randomisation (days) *	2 (1–4, 0–26)	2 (1–5, 0–28)

WFNS=World Federation of Neurological Surgeons clinical grading scale.¹⁸
*Median (IQR, range). †Patient ventilated and clinical state could not be assessed.

Table 1: **Baseline characteristics**

International Subarachnoid Aneurysm Trial (ISAT)

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Issue primaire	mRS ≥ 3 à 1 an

	Right	Midline	Left	Total
Anterior cerebral artery				
Anterior communicating	219	549	205	973
Proximal to anterior communicating	9	..	7	..
Pericallosal	46	..	49	..
Subtotal	1084 (50.5%)
Internal carotid artery				
Proximal or ophthalmic region	12	..	18	30
Posterior communicating region	313	..	223	536
Bifurcation	34	..	45	79
Other internal carotid	27	..	26	53
Subtotal	286	..	304	698 (32.5%)
Middle cerebral artery				
Proximal to bifurcation	14	..	14	..
Bifurcation	163	..	94	..
Distal to main bifurcation	7	..	11	..
Subtotal	184	..	119	303 (14.1%)
Posterior circulation				
Basilar bifurcation	..	17
Basilar trunk	..	1
Superior cerebellar	..	5
Posterior cerebral	1	..	3	..
Posterior inferior cerebellar	9	..	22	..
Subtotal	58 (2.7%)
Total	2143 (100%)

97%

Table 2: Aneurysm locations

International Subarachnoid Aneurysm Trial (ISAT)

- Traitement par voie endovasculaire...
 - ↓ dépendance et mortalité (mRS ≥ 3) 23.5 vs 30,9% (p=0.0001)
 - ↓ risque absolu de 7,4%, NNT ~ 14
 - Plus de récurrences de saignement à 1 an (2,6% vs 1%)
 - Oblitération complète de l'anévrisme 66% vs 82%
 - Moins de convulsion à 1 an (1% vs 2%)
 - Évolution fonctionnelle chez < 40 ans similaires dans les 2 groupes
- Petits anévrismes (< 1 cm)
- Circulation antérieure (97%)
- Bas grades (WFNS I-II 88%)
- Patients relativement jeunes

	Endovascular treatment (n=801)	Neurosurgery (n=793)
Modified Rankin scale		
0 No symptoms	207 (25.8%)	152 (19.2%)
1 Minor symptoms	217 (27.1%)	220 (27.7%)
2 Some restriction in lifestyle	187 (23.4%)	178 (22.4%)
3 Significant restriction in lifestyle	611 (76.3%)	550 (69.4%)
4 Partly dependent	80 (10.0%)	106 (13.4%)
5 Fully dependent	24 (3.0%)	32 (4.0%)
6 Dead	21 (2.6%)	25 (3.2%)
(3-6 inclusive)	65 (8.1%)	80 (10.1%)
	190 (23.7%)	243 (30.6%)

Data in Italics are primary outcome.

Table 6: **Outcome at 1 year in 1594 patients (primary outcome)**

Molyneux et al. Lancet 2002
 Molyneux et al. Lancet 2005
 Molyneux et al. Lancet Neurol 2009
 Molyneux et al. Lancet 2015

International Subarachnoid Aneurysm Trial (ISAT)

- Traitement par voie endovasculaire...
- À 5 ans
 - Mortalité diminuée (11% vs 14%, $p=0,03$)
 - Autonomie (mRS ≤ 2) idem (83% vs 82%)
 - Moins de convulsions
 - Plus de resaignement de l'anévrisme traité
- À 10 ans
 - Mortalité idem (17% vs 21%)
 - Autonomie (mRS ≤ 2) idem (82% vs 78%)
 - Plus de survivant et autonome OR 1,34 (1,07-1,67)

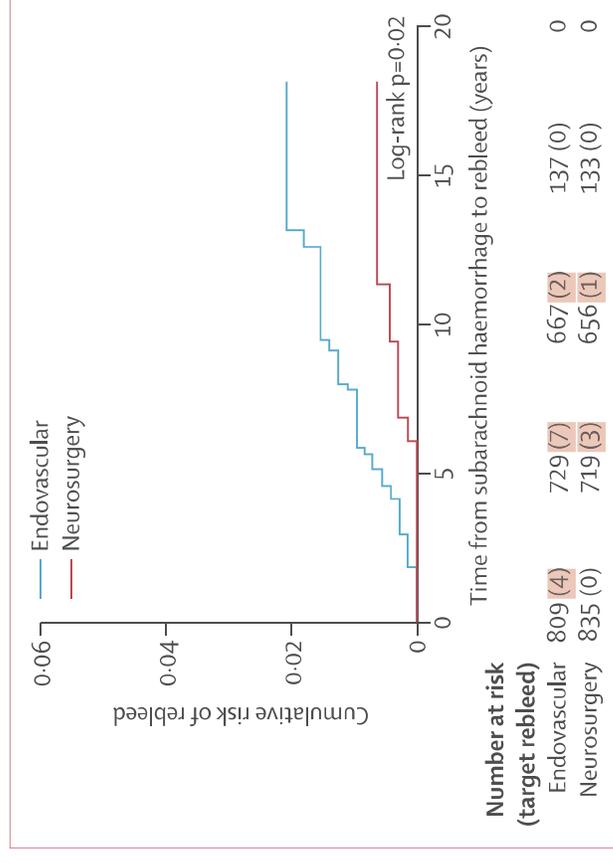


Figure 1: Kaplan-Meier plot of cumulative risk of rebleeding from target (treated) aneurysm later than 1 year after subarachnoid haemorrhage

Molyneux et al. Lancet 2002
Molyneux et al. Lancet 2005
Molyneux et al. Lancet Neurol 2009
Molyneux et al. Lancet 2015

The Barrow Ruptured Aneurysm Trial (BRAT)

	BRAT (J Neurosurg 2012)
Devis	Pseudo-randomisée monocentrique 2003-2007
Patients	472 57 – absence d’anévrysme
Inclusion	Age 18-80 ans HSA non-traumatique aux SI
Exclusion	HSA > 14 jrs HSA traumatique
Traitement	Clip vs coil
Issue primaire	mRS ≥ 3 à 1 an

- Étude pilote
- Devis ajusté à réalité de pratique
 - Politique d’alternance de traitement
 - Intent-to-treat
 - Droit de premier regard aux traitement alloué
 - 4 neurochirurgiens (2 endovasc, 2 chx)
- Absence d’anévrysme 57 patients

The Barrow Ruptured Aneurysm Trial (BRAT)

TABLE 2: Hunt and Hess grades at presentation in 471 patients in the BRAT*

Hunt & Hess Grade	No. Assigned to Clip Group (%)	No. Assigned to Coil Group (%)
I	32 (13.4)	31 (13.3)
II	92 (38.7)	93 (39.9)
III	71 (29.8)	61 (26.2)
IV	29 (12.2)	34 (14.6)
V	14 (5.9)	14 (6.0)

~ 50%

* The number originally assigned to surgical clipping was 238; the number originally assigned to coil therapy was 233.

TABLE 1: Patient and aneurysm characteristics in the BRAT*

Characteristic	No. Assigned to Clip Group (%)	No. Assigned to Coil Group (%)	p Value†
total no. of patients	238	233	
mean age in yrs	53.1 ± 12.8	54.3 ± 12.0	0.33
female	166 (69.7)	166 (71.2)	0.72
status at presentation			
mean GCS score	12.3 ± 3.6	12.5 ± 3.6	0.75
mean Hunt & Hess grade	2.6 ± 1.1	2.6 ± 1.1	0.94
mean Fisher grade	2.7 ± 0.7	2.7 ± 0.6	0.77
aneurysm feature			
size in mm‡			0.61
mean	6.8 ± 4.1	6.6 ± 4.0	
median; IQR	6.0; 4–8	6.0; 4–8	
location			0.62‡
posterior circulation	38 (16.0)	32 (13.7)	Ant 83% Post 17%
anterior circulation	174 (73.1)	169 (72.5)	
angiography negative	26 (10.9)	31 (13.3)	
other	not applicable	1 (0.4)	

* IQR = interquartile range.

† Continuous variables were analyzed using the Wilcoxon test, and nominal variables using chi-square tests, unless otherwise noted. The means are expressed ± SD throughout.

‡ Analyzed using the Fisher exact test.

§ Calculated based on 212 cases for the surgical clipping group and 201 for the coil embolization group.

The Barrow Ruptured Aneurysm Trial (BRAT)

- Traitement par voie endovasculaire...
 - ↓ dépendance et mortalité (mRS ≥ 3) 23.2 vs 33.7% (p=0.02)
 - ↓ risque absolu de 10,5%, NNT ~ 10
 - Même lorsque ajusté pour grade > 2 ou âge > 50 ans
 - Même lorsque analysé par traitement reçu et lorsque « cross-over » exclus

TABLE 4: Multivariable analysis of patients with poor outcome (mRS score > 2) at 1 year in the BRAT

Characteristic	OR (95% CI)	p Value
clipping*	1.72 (1.09–2.76)	0.020
age >50 yrs	2.03 (1.23–3.42)	0.007
Hunt & Hess grade >III†	3.51 (2.21–5.68)	<0.0001

* Includes all patients assigned to surgical clipping (intent to treat).

† The Hunt and Hess grade is entered into the regression as a binary variable.

Choix de modalité de sécurisation

- Devis différent mais signal idem favorisant traitement endovasculaire
- Si traitable par 2 approches, embolisation généralement privilégié
- Anatomie de l'anévrisme
 - Non favorable à embolisation: géant, collet large (ratio > 0,5), fusiforme
- Condition clinique + comorbidités du patient
- Évaluation multidisciplinaire

Preference for Treatment of Unsecured Aneurysms^a

TABLE 3-5

Treatment Type	Clinical or Aneurysm Factors Supporting Treatment Type
Endovascular coiling	Older age, poor clinical grade, multiple comorbidities, top of the basilar aneurysm, high surgical risk, aneurysm suitable for coiling or clipping
Surgical clipping	Aneurysm with wide neck-to-body ratio, crucial arteries arising from aneurysm dome, middle cerebral artery aneurysm, aneurysm with large parenchymal hematoma

TABLE 3. Choice of Technique for Intracranial Aneurysms

Endovascular coiling	Posterior circulation aneurysms Basilar tip aneurysms Intercavernous internal carotid artery aneurysms Elderly patients Patients with comorbid conditions Middle cerebral artery aneurysms
Surgical clipping	Fusiform aneurysms Giant aneurysms Aneurysms with wide neck Aneurysms at arterial bifurcations Ruptured aneurysms Younger patients

A randomized trial comparing endovascular and surgical management of ruptured intracranial aneurysms excluded from previous trials

MMD 6513 – Anesthésie et système nerveux
Antoine Halwagi, MD FRCPC

ISAT-2

Tim E. Darsaut, MD,¹ Nicolas Lecaros, MD,² Pierre-Olivier Comby, MD,² Roland Jabre, MD,³ Daniela Iancu, MD,² Daniel Roy, MD,² Alain Weill, MD,² Michel W. Bojanowski, MD,³ Chiraz Chaalala, MD,³ Gilles El Hage, MD,³ Alain Bilocq, MD,⁴ Eric Truffer, MD,⁴ J. Max Findlay, MD, PhD,¹ Jeremy L. Rempel, MD,⁵ Michael M. C. Chow, MD,¹ Cian J. O’Kelly, MD,¹ Robert A. Ashforth, MD,⁵ Owen Stechishin, MD,⁵ Thomas Gaberei, MD,⁶ Charlotte Barbier, MD,⁷ Fuat Arıkan, MD, PhD,⁸ Ignacio Arrese, MD,⁹ Rosario Sarabia, MD,⁹ David J. Altschul, MD,¹⁰ Miguel Chagnon, MSc,¹¹ Justine Zehr, MSc,¹¹ Jai J. S. Shankar, MD,¹² François Proust, MD,¹³ Guylaine Gevry, BSc,¹⁴ and Jean Raymond, MD^{2,14}

- RCT multicentrique pragmatique
- Inclusion: HSA < 30 jrs, WFNS 1-4, adressable par ligature et embolisation
- Exclusion: anévrisme associé à MAV, apex basilaire
- But: inclure des patients qui auraient été exclus de ISAT
- Exemples:
 - Anévrisme > 1 cm
 - Circulation postérieure
 - WFNS III-IV
 - Âge > 70 ans

A randomized trial comparing endovascular and surgical management of ruptured intracranial aneurysms excluded from previous trials

Tim E. Darsaut, MD,¹ Nicolas Lecaros, MD,² Pierre-Olivier Comby, MD,² Roland Jabre, MD,³ Daniela Iancu, MD,² Daniel Roy, MD,² Alain Weill, MD,² Michel W. Bojanowski, MD,³ Chiraz Chaalala, MD,³ Gilles El Hage, MD,³ Alain Bilocq, MD,⁴ Eric Truffer, MD,⁴ J. Max Findlay, MD, PhD,¹ Jeremy L. Rempel, MD,⁵ Michael M. C. Chow, MD,¹ Cian J. O’Kelly, MD,¹ Robert A. Ashforth, MD,⁵ Owen Stechishin, MD,⁵ Thomas Gaberei, MD,⁶ Charlotte Barbier, MD,⁷ Fuat Arikan, MD, PhD,⁸ Ignacio Arrese, MD,⁹ Rosario Sarabia, MD,⁹ David J. Altschul, MD,¹⁰ Miguel Chagnon, MSc,¹¹ Justine Zehr, MSc,¹¹ Jai J. S. Shankar, MD,¹² François Proust, MD,¹³ Guylaine Gevry, BSc,¹⁴ and Jean Raymond, MD^{2,14}

ISAT-2

- 270/1896 pts en 10 ans – cessé prématurément
- mRS >2 à 1 an 30 vs 27% (NS)
- **Ligature: trajectoire plus compliquée**
 - ↑ durée de séjour (29 vs 19 jrs)
 - ↑ DVE (42 vs 24%) et DVP (19 vs 7%)
 - ↑ craniectomie décompressive (12 vs 5%)
 - ↑ vasospasme symptomatique (29 vs 18%)
- **Embolisation: ↑ anévrysme résiduel (20 vs 8%)**

Darsaut et al. J Neurosurg 2025

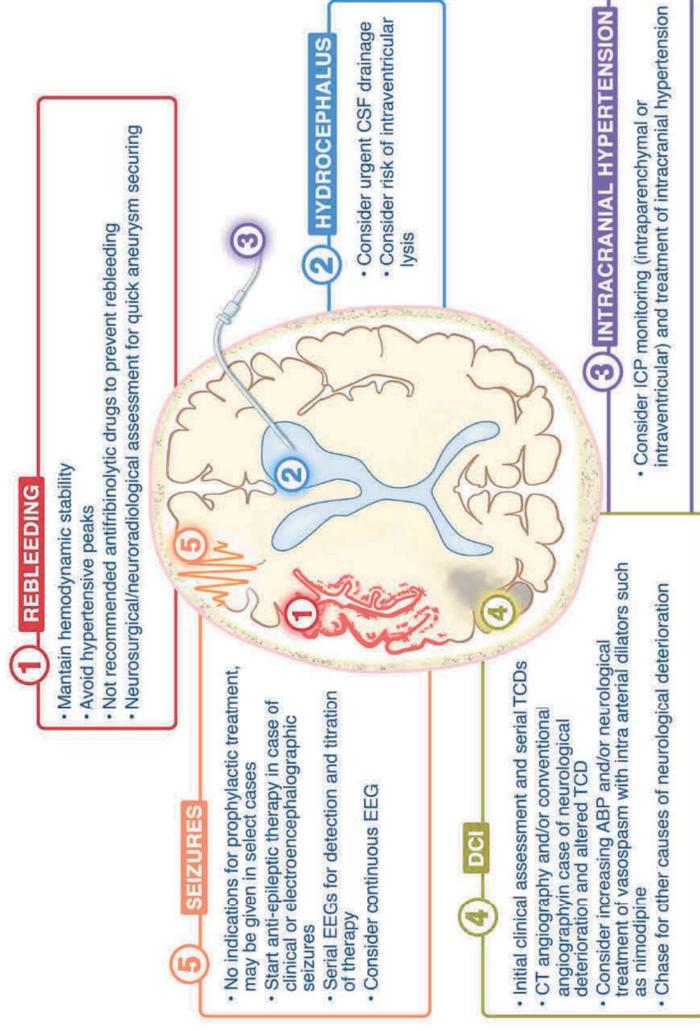
MMD 6513 – Anesthésie et système nerveux
Antoine Halwagi, MD FRCP C

TABLE 1. Patient and aneurysm characteristics

Characteristic	Surgical	EVT
No. of patients	133	130
Mean age at treatment ± SD, yrs	57.5 ± 12.5	57.1 ± 11.6
Age ≥70 yrs	19 (14)	17 (13)
Female sex	94 (71)	86 (66)
Prerupture mRS score		
0	117 (88)	116 (89)
1	15 (11)	11 (8)
2	1 (1)	3 (2)
WFNS grade		
I–III	111 (83)	107 (82)
IV	22 (17)	23 (18)
Index aneurysm location		
Anterior circulation	124 (93)	123 (95)
Ophthalmic/paraophthalmic artery	4 (3)	1 (1)
PCoA/AChA	23 (15)	19 (15)
Carotid terminus	1 (1)	3 (2)
MCA	56 (42)	50 (38)
ACoA	33 (25)	39 (30)
Pericallosal artery	10 (8)	11 (8)
Posterior circulation	9 (7)	7 (5)
Aneurysm size, mm		
Mean	7.5	6.6
Median (range)	6 (1–48)	6 (2–25)
≤3	32 (24)	29 (22)
4–9	72 (54)	79 (61)
≥10	29 (22)	22 (17)
Wide neck, ≥ 4 mm	43 (32)	40 (31)
Recurrent, previously treated index aneurysm	5 (5)	4 (3)
Multiple aneurysms	24 (18)	28 (22)
Intraparenchymal hematoma >2 cm	22 (17)	27 (21)
Pretreatment infarction	5 (5)	4 (3)
Fisher grade		
0–2	45 (34)	38 (29)
3 or 4	88 (66)	92 (71)
Treatment		
Mean (median) time from SAH to treatment, days	4 (1)	2 (1)
Adherence to allocated treatment	124 (93)	127 (98)
Aneurysm reupture after randomization, prior to treatment*	4 (3)	4 (3)
Received stent or flow diverter	1 (1)	17 (13)
Received parent vessel occlusion	0	4 (3)

Complications intracrâniennes

- Priorité de prévenir le dommage cérébral secondaire
- Hydrocéphalie
- Convulsions
- Hypertension intracrânienne
- Ischémie cérébrale retardée



Prévenir dommage cérébral secondaire

- Hydrocéphalie
 - 20% des HSA
 - Résolution spontanée en 24h - 30%
- Risques DVE
 - Infection 7.9%
 - Hémorragie (HIV, HIP) 8.4%
 - Symptomatique 0.7%
 - **Resaignement 0-43% - précipité par DVE?**
 - Dérivation permanente 7%
- **Possibilité d'amélioration grade clinique post-DVE**
- Évolution fonctionnelle corrélée avec ce « nouveau » grade clinique

Prévenir dommage cérébral secondaire

- Convulsions
 - Ictus 4-26%, durant hospitalisation 1-28%
 - Épilepsie à long terme ~ 2%
 - FR: jeunes, inconscience @ ictus, HTA, MCA, haut grade radiologique, HIP, HSD, ligature chirurgicale, DCI
 - Pas d'ERC
 - Phénytoïne associée à mauvaise évolution neurologique

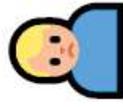
Lignes directrice NCS en cours de révision

COR	LOE	Recommendations
Patients who present without seizures		
2a	B-NR	1. In patients with aSAH and either fluctuating neurological examination, depressed mental state, ruptured MCA aneurysm, high-grade SAH, ICH, hydrocephalus, or cortical infarction, cEEG monitoring is reasonable to detect seizures. ^{291,405,406}
2b	B-NR	2. In patients with aSAH and high-seizure-risk features (ie, ruptured MCA aneurysm, high-grade SAH, ICH, hydrocephalus, and cortical infarction), use of prophylactic antiseizure medication(s) may be reasonable to prevent seizures. ⁴⁰⁷⁻⁴¹³
3: No benefit	B-R	3. In patients with aSAH without high-seizure-risk features (ie, ruptured MCA aneurysm, high-grade SAH, ICH, hydrocephalus, and cortical infarction), prophylactic treatment with antiseizure medication is not beneficial. ³⁹²
3: Harm	B-NR	4. In patients with aSAH, phenytoin for seizure prevention and/or antiseizure prophylaxis is associated with excess morbidity and mortality. ^{407-411,413-415}
Patients who present with seizures		
2a	B-NR	5. In patients with aSAH who present with seizures, treatment with antiseizure medications for ≤7 days is reasonable to reduce seizure-related complications in the perioperative period. ^{411,416,417}
3: No benefit	B-NR	6. In patients with aSAH without prior epilepsy who present with seizures, treatment with antiseizure medications beyond 7 days is not effective for reducing future SAH-associated seizure risk. ^{408,410,411}

Prévenir dommage cérébral secondaire

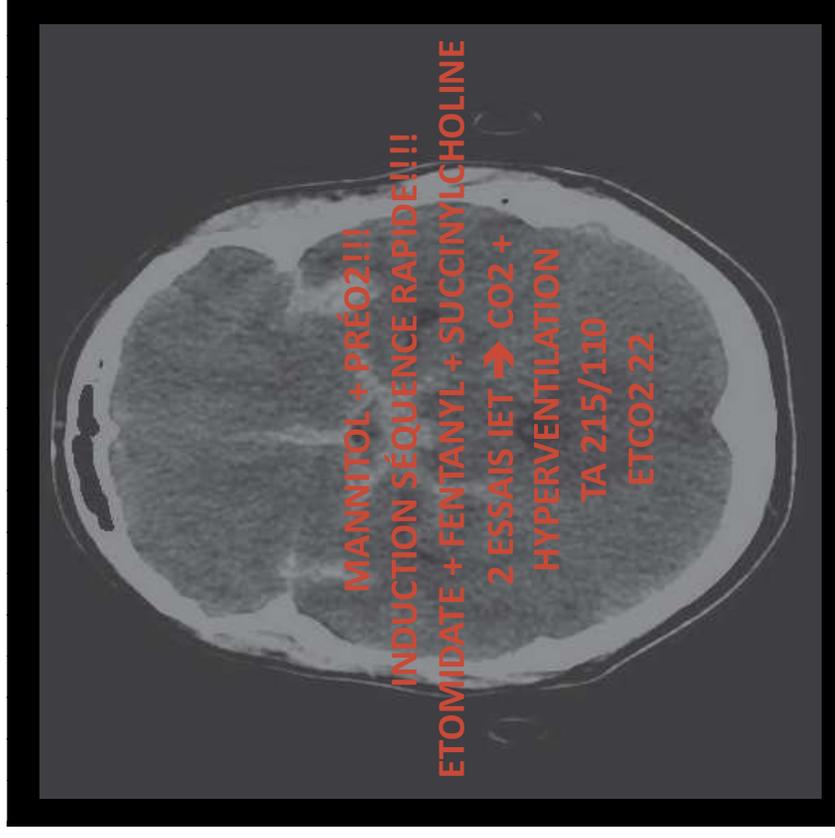
- Hypertension intracrânienne - 50% des HSA
- Mécanismes d'hypertension intracrânienne
 - Œdème cérébral (ischémie focale / globale)
 - HIP → effet de masse
 - HCP aigu (communicante / non-communicante)
 - Vasospasme → vasodilatation en aval
 - Perte d'autorégulation
 - Convulsions
 - Causes extracrâniennes (hypercapnie, hypoxémie, hypo/hypertension, hyperthermie, ...)
 - **Marqueur de dommage cérébral extensif?**
- Traitement HTIC en HSA - principes largement extrapolé de TCC
 - Viser PIC < 20 mmHg
 - Viser PPC entre 50-70 mmHg
 - **Lignes directrices restent muettes...**

HSA non-sécurisée: induction / intubation



48 ans, pas ATCD, céphalée brutale

- AEC au retour du scan
- TA 180/95 FC 75 SpO2 95%

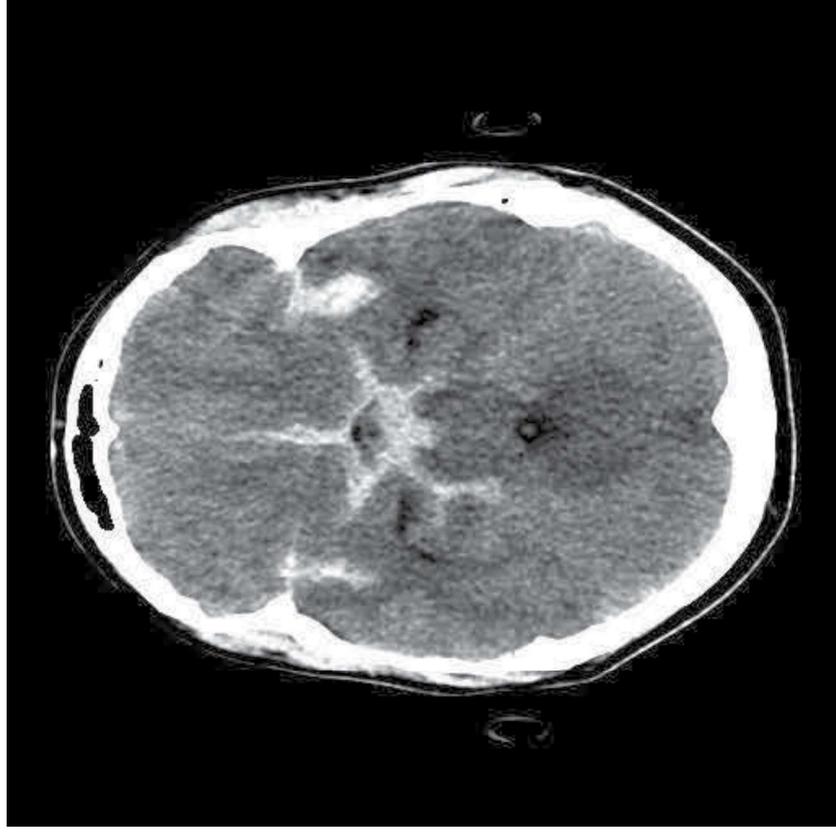


HSA non-sécurisée: induction / intubation

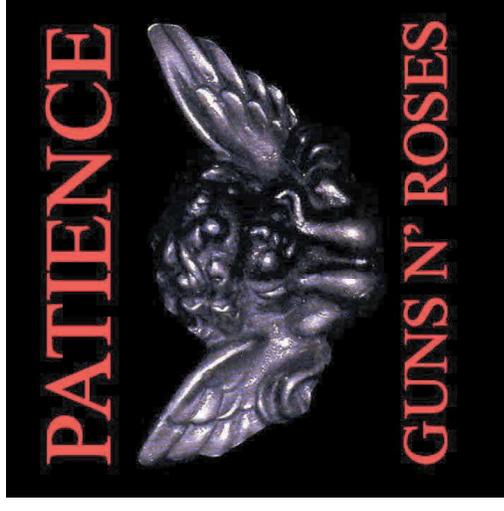
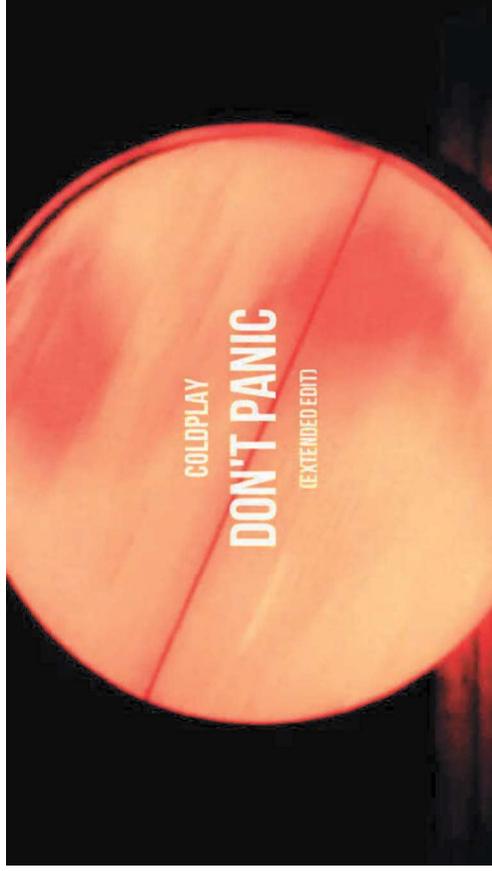


37 ans, G1 37+, céphalée brutale

- AEC au retour du scan
- TA 165/90 FC 92 SpO2 94%
- Pupilles isocores



HSA non-sécurisée: induction / intubation

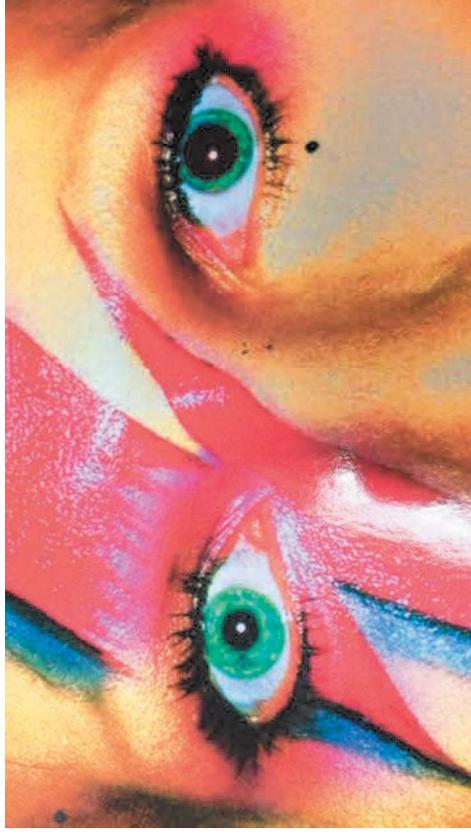


HSA non-sécurisée: induction / intubation



25 ans, pas ATCD, céphalée brutale

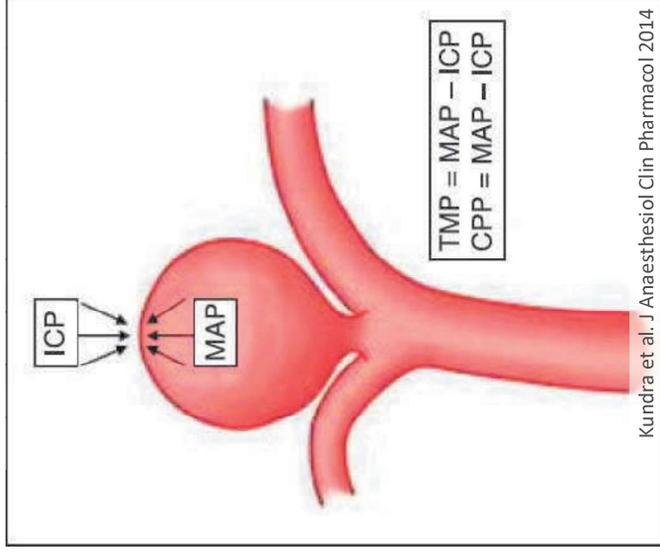
- AEC au retour du scan
- TA 200/105 FC 45 SpO2 92%



HSA non-sécurisée: induction / intubation

- Prévenir réponse adrénergique à laryngoscopie
- Titration d'agents – éviter variation HD brusques
- Malgré HTA et agents anti-HTA, anticiper hypoTA
- HSA haut grade non-HTA → ❤️
- Canule artérielle avant induction
- Si DVE in situ → lecture PIC (présence ou absence d'HTIC?)

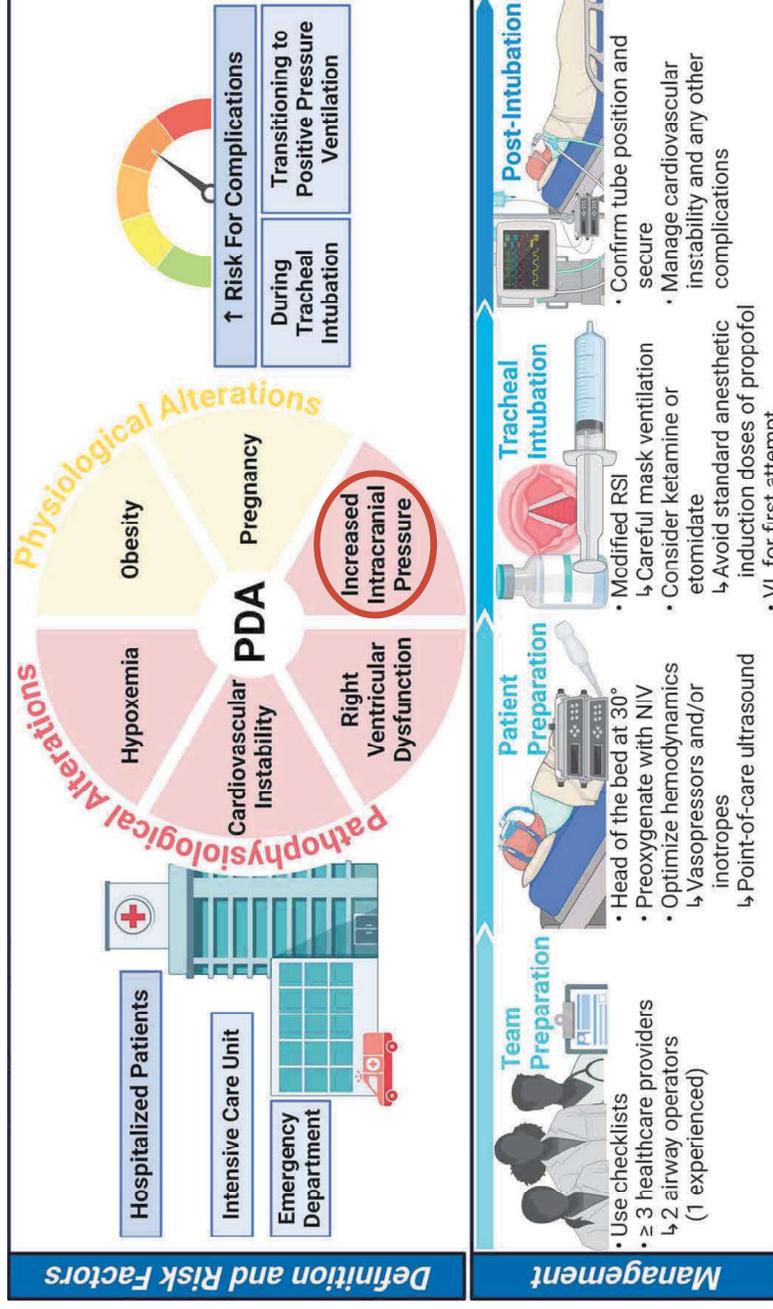
- Éviter interventions qui vont...
 - ↑↑↑ PA (sauf si hypoTA sévère)
 - ↓↓↓ PIC (sauf si « brain code »)



CONFERENCE REPORTS AND EXPERT PANEL



Tracheal intubation in critically ill adults with a physiologically difficult airway. An international Delphi study



Hemodynamic Monitoring and Optimization

The minimum mandatory monitoring in patients with a PDA undergoing tracheal intubation should include non-invasive blood pressure, continuous electrocardiogram, and pulse oximetry.

Hemodynamics should be optimized prior to tracheal intubation in patients with a PDA. Interventions such as vasopressor and/or inotrope infusion administration can help prevent and/or minimize peri-intubation cardiovascular collapse.

Use of peri-procedural point-of-care ultrasound, when feasible, can improve the safety of the tracheal intubation by aiding the assessment and management of cardio-respiratory compromise.

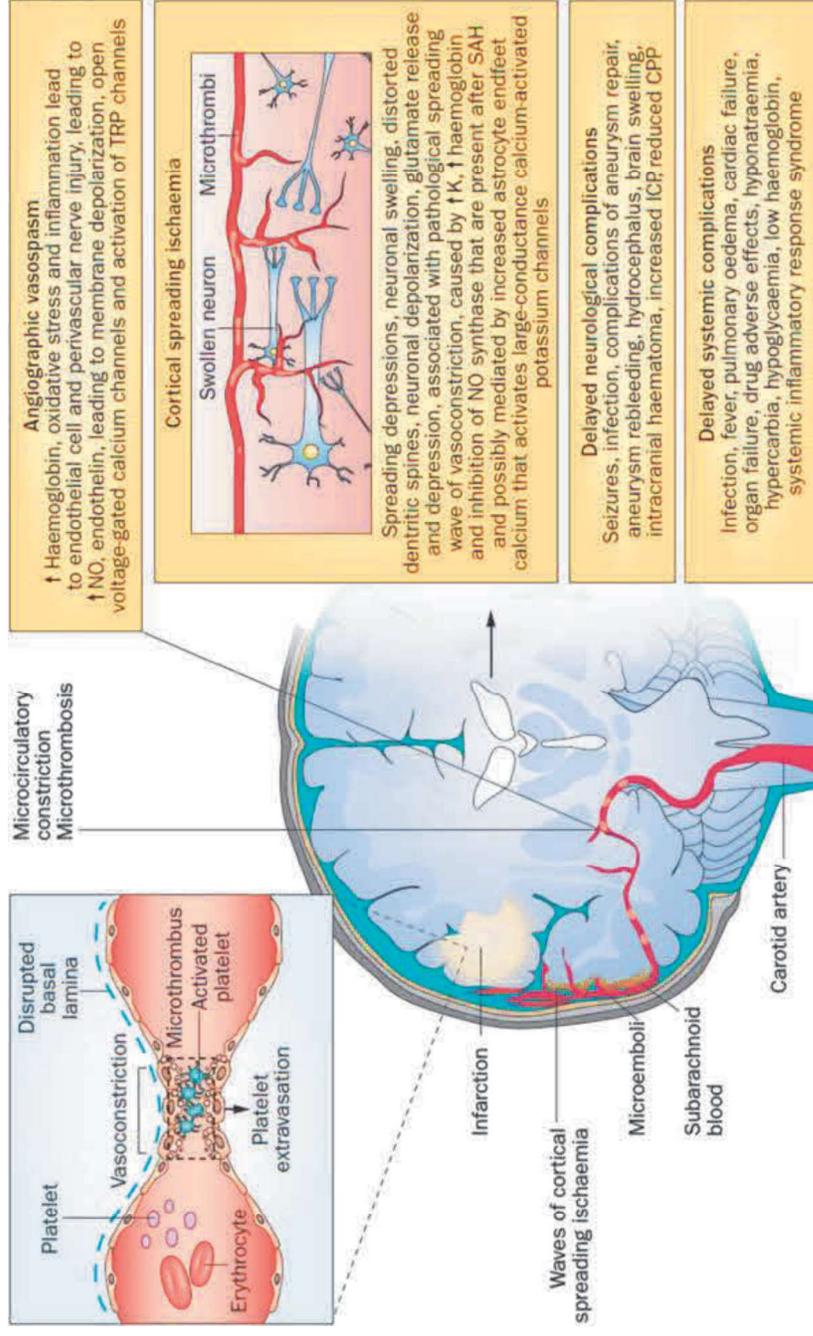
Recommandations intra-opératoires limitées...

COR	LOE	Recommendations
2a	B-R	1. In patients with aSAH, the intraoperative use of mannitol or hypertonic saline can be effective in reducing ICP and cerebral edema. ^{135,136}
2a	B-NR	2. In patients with aSAH, anesthetic goals should include minimizing postprocedural pain, nausea, and vomiting. ¹³⁷⁻¹⁴⁰
2a	B-NR	3. In patients with aSAH, prevention of intraoperative hyperglycemia and hypoglycemia during aneurysm surgery is reasonable to improve outcomes. ¹⁴¹⁻¹⁴⁷
2a	C-LD	4. In patients with aSAH and an unsecured ruptured aneurysm, frequent intraoperative BP monitoring and BP control are reasonable to prevent ischemia and rerupture. ¹⁴⁸⁻¹⁵³

COR	LOE	Recommendations
2b	B-NR	5. In patients with aSAH, intraoperative neuromonitoring may be reasonable to guide anesthetic and operative management. ¹⁵⁴⁻¹⁵⁹
2b	C-LD	6. In patients with aSAH and an uncontrolled intraoperative aneurysmal rupture, adenosine may be considered to facilitate aneurysm clip placement by inducing cardiac standstill and temporary profound pause. ^{160,161}
3: No benefit	B-R	7. In patients with good-grade aSAH, the routine use of induced mild hypothermia during aneurysm surgery is not beneficial. ^{147,162-168}

Ischémie cérébrale retardée (*delayed cerebral ischaemia*)

- Détérioration neurologique retardée
 - Progression EBI
 - Hydrocéphalie
 - Re-saignement
 - Convulsions
 - Cause extra-crânienne
 - **Ischémie cérébrale retardée (DCI)**
- Période à risque J3-J14
- **Vasospasme** angiographique
- Microthromboses
- Vasoconstriction microvasculaire
- Dysfonction endothéliale
- Altération autorégulation cérébrale
- Dépression corticale propagée
- Contribution de complications extracrâniennes



Conséquences de dommage cérébral retardé

- ♀53A WFNS 5 (4), mF 4 (HCP)
- DVE frontale D J0 (PIC 14)
- Anévrisme Pcomm G – embolisée J0
- Extubée J1 →
 - Ralentissement psychomoteur
 - Désorientée
 - Communiquer par phrases simples
 - Parésie MSD 4/5
 - IRM ~N J2



Conséquences de dommage cérébral retardé

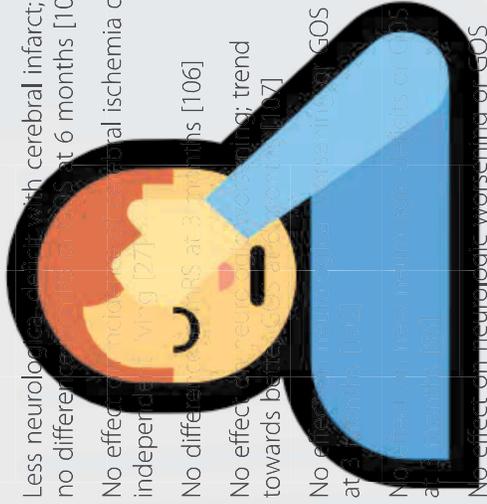
- ♀53A WFNS 5 (4), mF 4 (HCP)
- Détérioration J6
 - DCI et vasospasme sévère
 - Traitement médical
 - Traitement endovasculaire
- IRM catastrophique J14
- Soins palliatifs subséquemment



Prévention

Table 2 Selected pharmacologic interventions that have been evaluated for DCI prevention^a

Intervention	Effect
Aspirin	No effect on new lesion associated with neurological worsening [103]
Clazosentan	No effect on mortality or vasospasm-related morbidity [5]
Enoxaparin	No effect on DCI or GOS at 3 months [104]
Erythropoietin	Less neurological morbidity with cerebral infarct; no difference in GOS at 6 months [105]
Fludrocortisone	No effect on mortality or vasospasm-related morbidity; independent of cerebral ischemia or infarct [106]
Magnesium	No difference in GOS at 3 months [106]
Methylprednisolone	No effect on mortality or vasospasm-related morbidity; trend towards better GOS at 3 months [107]
Nicardipine	No effect on mortality or vasospasm-related morbidity; trend towards better GOS at 3 months [108]
Prophylactic angioplasty	No effect on mortality or vasospasm-related morbidity; trend towards better GOS at 3 months [69]
Prophylactic hypervolemia	No effect on neurologic worsening or GOS at 3 months [69]
Statins	No effect on DCI, death or mRS at 6 months [108]



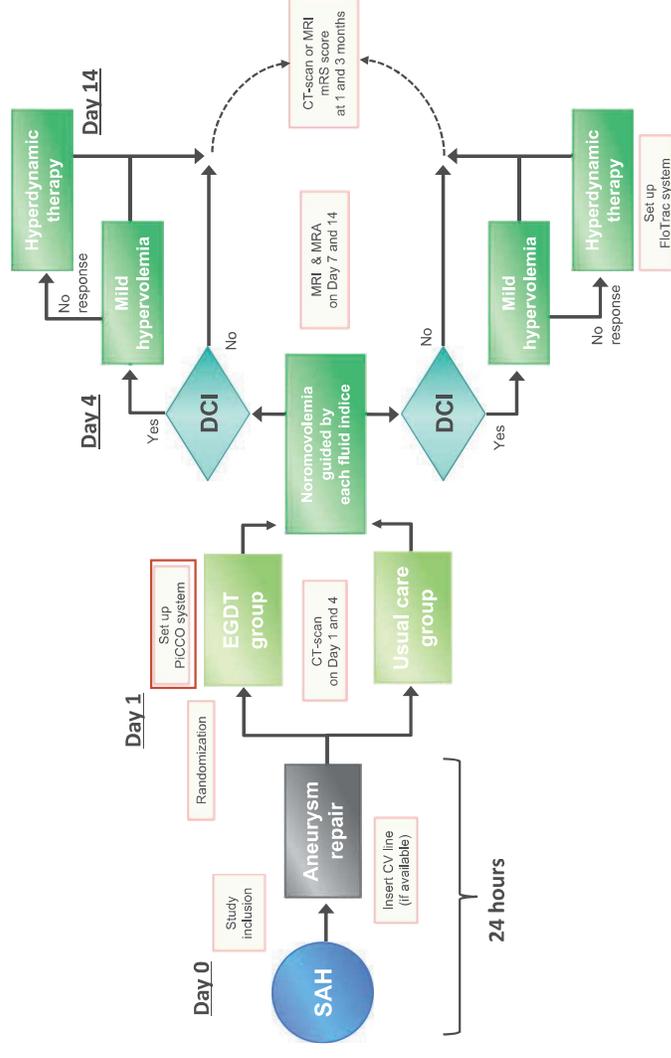
Hormis nimodipine (et euvoémie), aucun traitement prophylactique ne semble efficace...

COR	LOE	Recommendations
1	A	1. In patients with aSAH, early initiation of enteral nimodipine is beneficial in preventing DCI and improving functional outcomes. ^{316–319}
2a	B-NR	2. In patients with aSAH, maintaining euvoemia can be beneficial in preventing DCI and improving functional outcomes. ^{320,321}
3: No benefit	A	6. In patients with aSAH, routine use of statin therapy to improve outcomes is not recommended. ^{345,346}
3: No benefit	A	7. In patients with aSAH, routine use of intravenous magnesium to improve neurological outcomes is not recommended. ^{347,348}
3: Harm	B-R	8. For patients with aSAH at risk of DCI, prophylactic hemodynamic augmentation should not be performed to reduce iatrogenic patient harm. ^{189,211,349,350}

Gestion volémique en HSA

Impact favorable de thérapie dirigée (EGDT) chez patients à haut risque

- HSA haut grades (WFNS IV-V)
 - ↓ DCI (5 vs 14% p=0.036)
 - ↑ mRS 0-3 (52 vs 36% p=0.026)
 - ↓ séjour USI (14 vs 17j p=0.043)
 - ↑ réponse au tx hyperdynamique
 - ↓ volume et OAP (tendance)
- Complication cardiopulmonaires
 - ↑ mRS 0-3 (63 vs 38% p=0.045)
 - ↓ séjour USI réduite (tendance)



Gestion volémique en HSA

Impact favorable de thérapie dirigée (EGDT)

- ↓ DCI (13 vs 32% $p=0.021$)
- Vasospasme idem (50 vs 59%)
- ↓ hypona (11 vs 31% $p=0.01$)
- ↓ œdème cérébral (15 vs 30% $p=0.064$)
- ↓ infarctus non-DCI (2 vs 11% $p=0.051$)
- ↑ GOS=5 @ 3 mo (66 vs 44% $p=0.025$)
- Globalement même quantité de volume...
 - Impact de le donner au bon moment?

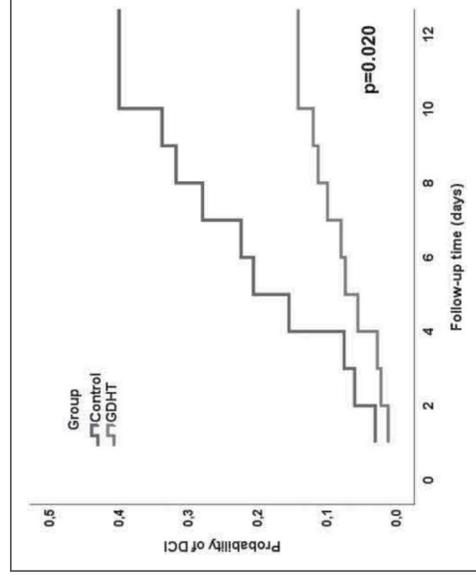
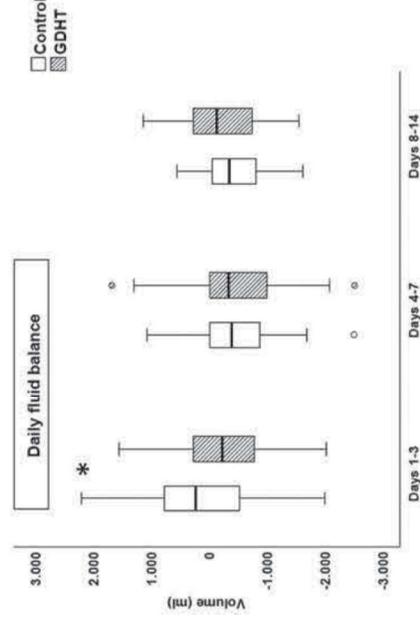


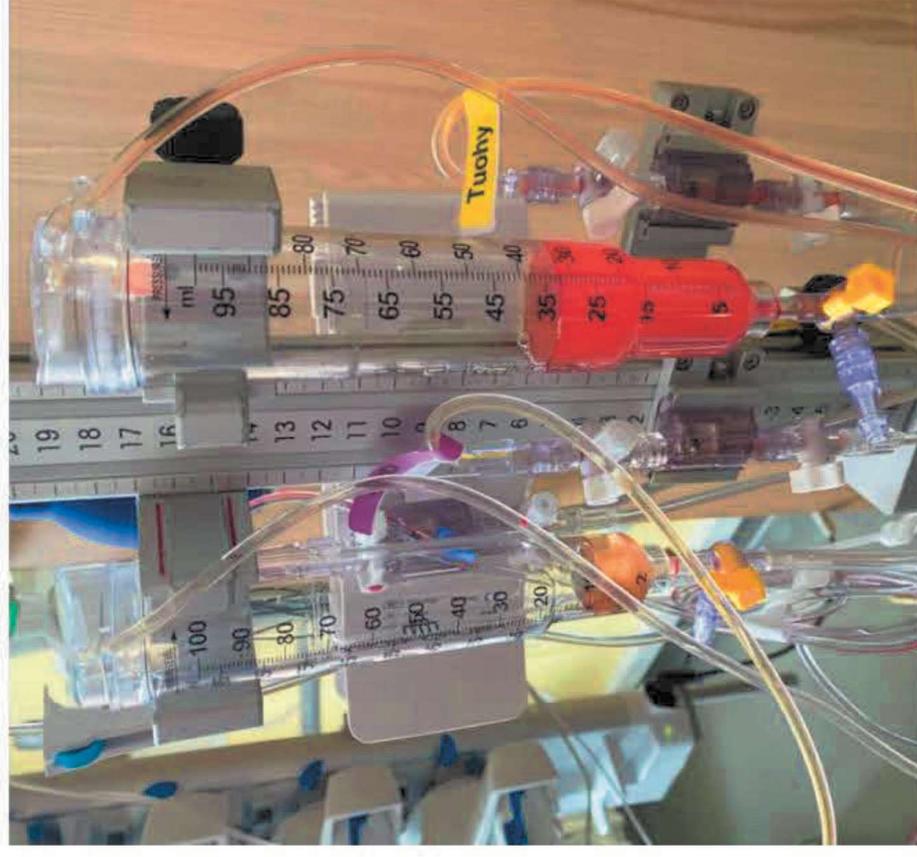
Figure 3. Cox proportional hazards model for goal-directed hemodynamic therapy (GDHT) vs control group.

Effectiveness of Lumbar Cerebrospinal Fluid Drain Among Patients With Aneurysmal Subarachnoid Hemorrhage A Randomized Clinical Trial

Stefan Wolf, MD; Dorothee Mielke, MD; Christoph Barner, MD; Yesna Malinova, MD; Thomas Kerz, MD; Maria Wostrack, MD; Patrick Czoflich, MD; Farid Salih, MD; Dooirje C. Engel, MD; Angelika Ehlerl, MD; Dimitre Staykov, MD; Abdulrahman Y. Alturki, MD; Ulrich Sure, MD; Jürgen Bardutzky, MD; Henry W. S. Schroeder, MD; Ludwig Schürer, MD; Jürgen Beck, MD; Tareq A. Juratli, MD; Michael Fritsch, MD; Johannes Lemcke, MD; Anne Pohrt, Dipl.-Math; Bernhard Meyer, MD; Stefan Schwab, MD; Veit Rohde, MD; Peter Vajkoczy, MD; for the EARLYDRAIN Study Group

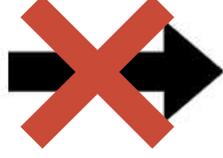
- RCT multicentrique 19 unités DECA
- 287 pts drain lombaire ≤ 72h HAS
- Drainage LCR 5 mL/h x 8 jrs
- WFNS 1 ~ 33% WFNS 3-5 ~ 50%
- **mRS 3-6 (3mo) 33 vs 45%**
- Vasospasme idem
- ↓ infarctus secondaire 29 vs 40%

Wolf et al. EARLYDRAIN JAMA Neurol 2023



Changement de paradigme

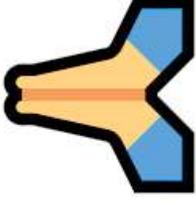
↓ VASOSPASME



ÉVOLUTION FAVORABLE

Surveillance DCI/vasospasme

COR	LOE	Recommendations
2a	B-NR	1. In patients with aSAH with suspected vasospasm or limited neurological examination, CTA or CT perfusion (CTP) can be useful to detect vasospasm and predict DCI. ²⁷⁰⁻²⁷⁵
2a	B-NR	2. In patients with aSAH, transcranial Doppler (TCD) ultrasound monitoring is reasonable to detect vasospasm and predict DCI. ^{253,276-280}
2a	B-NR	3. In patients with high-grade aSAH, continuous EEG (cEEG) monitoring can be useful to predict DCI. ^{276,280-292}
2b	B-NR	4. In patients with high-grade aSAH, invasive monitoring of brain tissue oxygenation, lactate/pyruvate ratio, and glutamate may be considered to predict DCI. ²⁹³⁻³⁰⁵

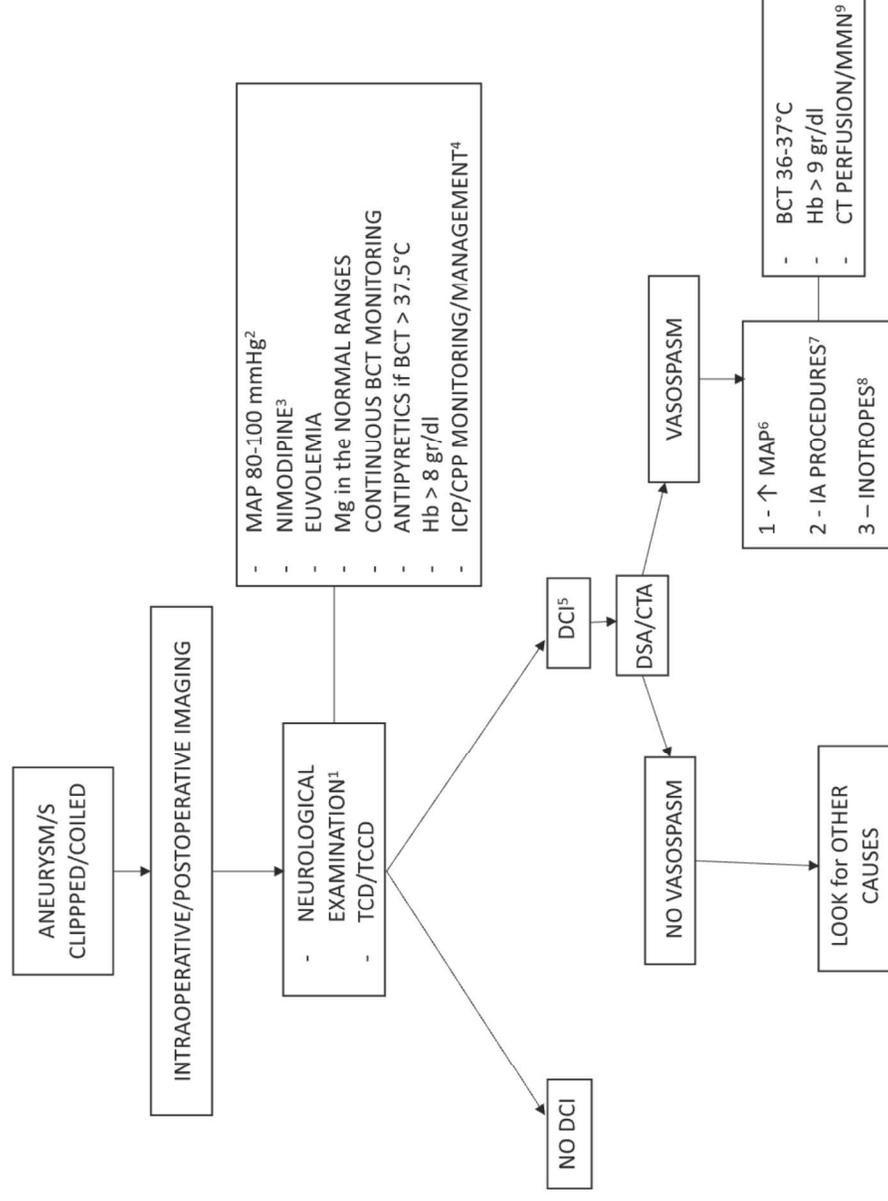


Traitements possibles

- **AUCUN TRAITEMENT DÉMONTRÉ EFFICACE...**
- Neuromonitoring multimodal pour individualiser thérapie?
- Multiples études en cours
 - Milrinone
 - Anti-inflammatoires
 - Héparine

COR	LOE	Recommendations
2b	B-NR	3. In patients with aSAH and symptomatic vasospasm, elevating systolic BP values may be reasonable to reduce the progression and severity of DCI. ^{322–325}
2b	B-NR	4. In patients with aSAH and severe vasospasm, use of intra-arterial vasodilator therapy may be reasonable to reverse cerebral vasospasm and reduce the progression and severity of DCI. ^{326–335}
2b	B-NR	5. In patients with aSAH and severe vasospasm, cerebral angioplasty may be reasonable to reverse cerebral vasospasm and reduce the progression and severity of DCI. ^{336–344}

DCI / vasospasme – exemple d’algorithme



Prévention / traitement DCI – résumé

Table 1 Prevention and management of delayed cerebral ischemia

Guideline recommendations
American Heart Association [30], Neurocritical Care Society [122] & European Stroke Organization [123]

DCI prevention

Strong recommendation Nimodipine (early initiation, enteral) [30, 122, 123]

Moderate recommendation Maintenance of euvolemia [30, 122, 123]

Insufficient evidence Calcium channel blockers (other than nifedipine), intravenous or intraventricular [122] or into surgical space [123]

Not recommended Intravenous nifedipine [122, 123], endothelin receptor antagonist [122], statins [30, 122], magnesium sulphate [30, 122, 123], hypervolemia [122, 123], prophylactic hemodynamic augmentation [30]

Management of DCI

Weak recommendations Hemodynamic augmentation if symptomatic vasospasm present [30]

Intraarterial vasodilator therapy for severe vasospasm [30]

Cerebral angioplasty for severe vasospasm [30]

Insufficient evidence Hemodynamic augmentation [122, 123]

Augmentation hémodynamique → augmentation DSC?

- 1 seule étude randomisée contrôlée – cessée prématurément
- 41/240 patients prévus, manque de puissance
- HTA (volume + NA) vs contrôle → différence PAM ~ 10 mmHg @ 24h
- Plus d'amélioration clinique dans groupe HTA (57% vs 30%)
- Évolution défavorable (mRS > 3) @ 3 mois IDEM

Table 2. Differences in CBF Values Between the Groups

	On Treatment		Intention to Treat		Crossover Case Excluded	
	No Induced Hypertension (n=13)	Induced Hypertension (n=12)	No Induced Hypertension (n=12)	Induced Hypertension (n=13)	No Induced Hypertension (n=12)	Induced Hypertension (n=12)
Change in overall CBF (median, range)	-8.5 (-42 to 30)	0.1 (-31 to 43)	-9.7 (-42 to 30)	-1.2 (-31 to 43)	-9.7 (-42 to 30)	0.1 (-31 to 43)
Change in lowest CBF (median, range)	1.0 (-23 to 41)	11.2 (-23 to 50)	1.9 (-23 to 41)	10.3 (-23 to 50)	1.9 (-23 to 41)	11.2 (-23 to 50)
			P Value*	P Value*	P Value*	P Value*
			0.25	0.25	0.25	0.24
			0.38	0.50	0.50	0.41

HIIMALAIA

Gathier et al. Stroke 2015
 Gathier et al. Stroke 2018

Augmentation hémodynamique → augmentation DSC?

- 1 seul
- 41/24
- HTA (v
- Plus d
- Évolut

Our findings do not support the use of induced hypertension to augment overall CBF in aneurysmal subarachnoid hemorrhage patients with DCI. However, a small effect cannot be definitively excluded because a trend was seen in improved CBF in areas with lowest perfusion. This might be of clinical interest, as hypoperfused areas might progress to infarction if not treated. Induced hypertension might thus be beneficial in improving CBF in regions with impaired perfusion. Based on our results, 225 to 250 patients per group would be needed to find a statistically significant difference in change in overall CBF between induced hypertension and no hypertension.

@ 24h

Table 2. Differences in

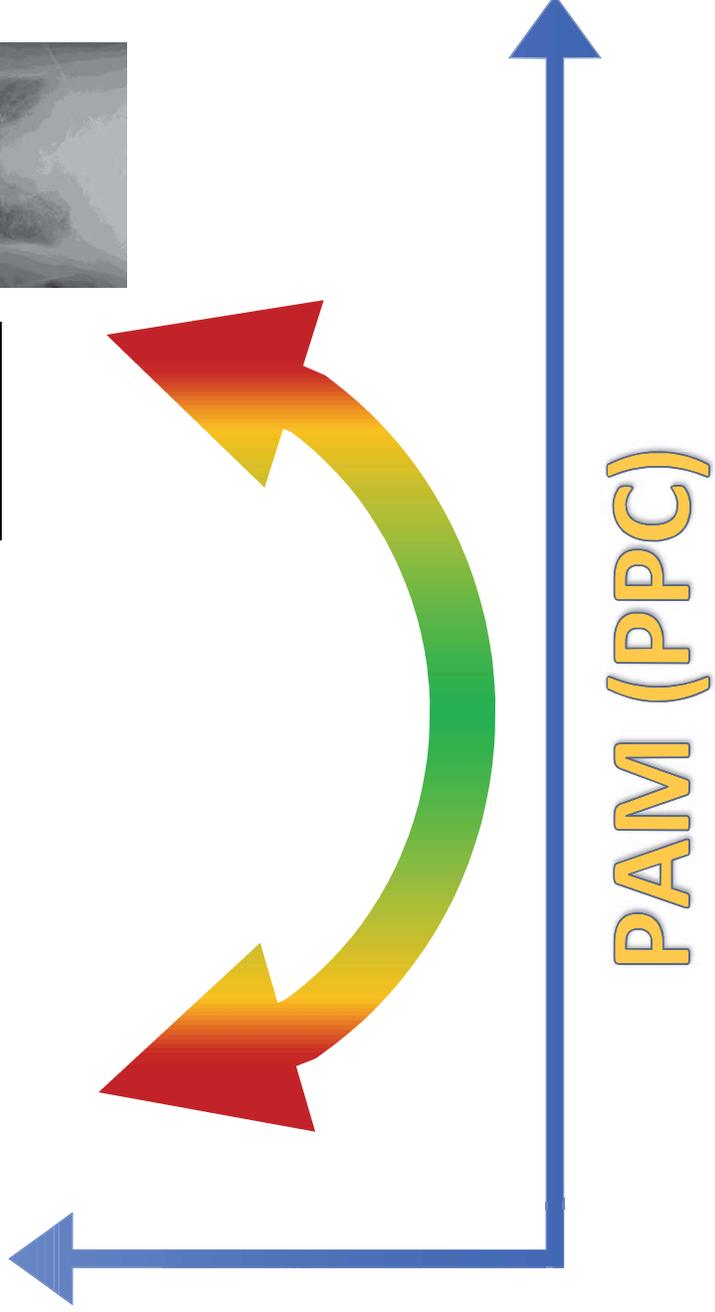
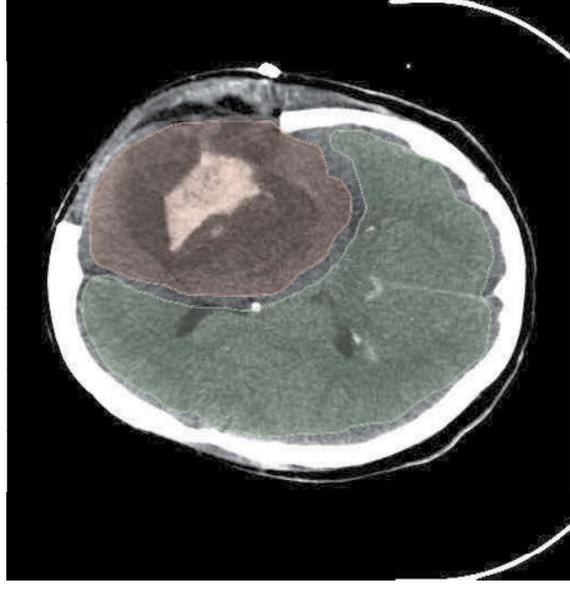
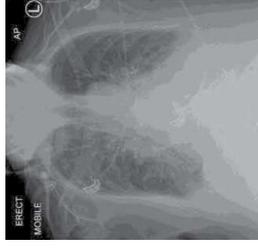
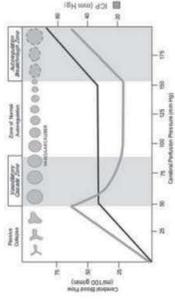
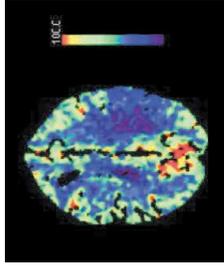
Change in overall CBF
(median, range)

Change in lowest CBF
(median, range)

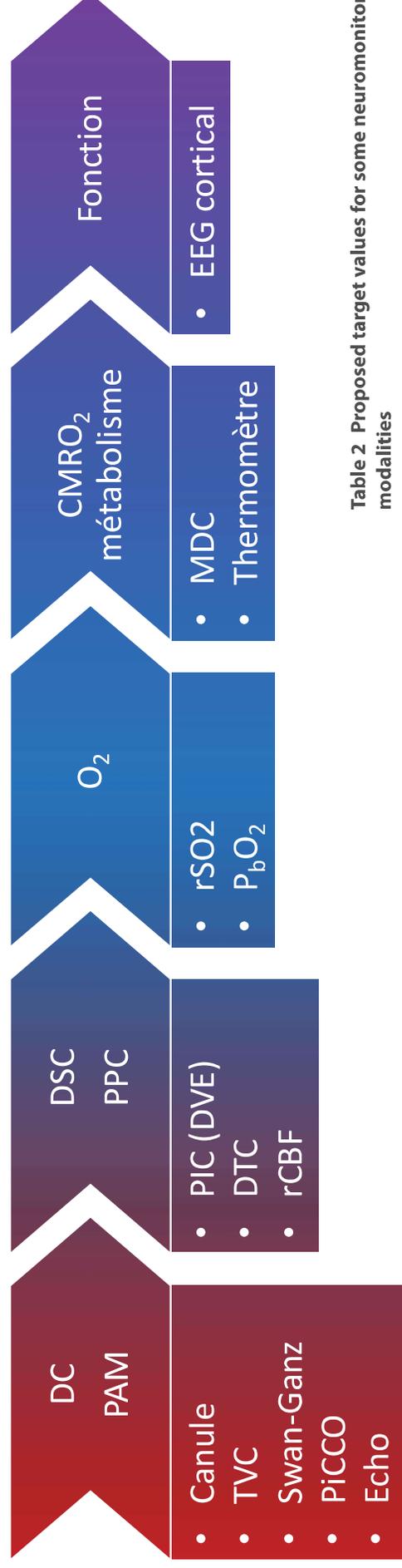


Augmentation hémodynamique → **DANGER!**

R I S Q U E S



Neuromonitoring multimodal



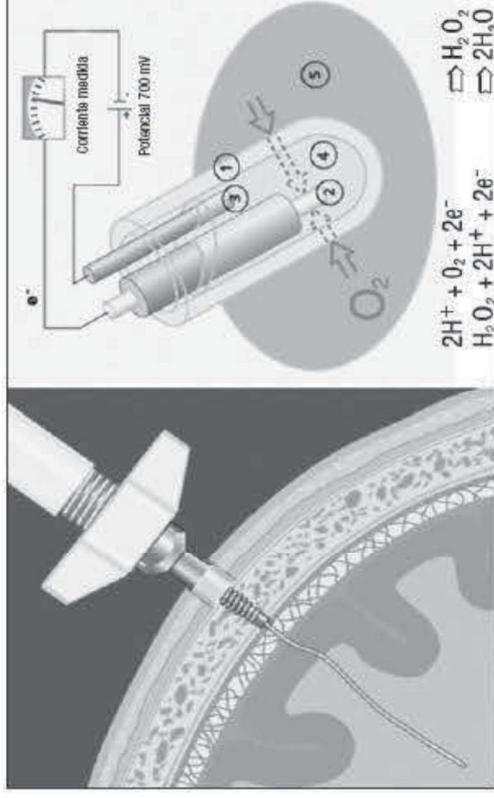
- Détection de souffrance cérébrale occulte
- Prévention de dommage cérébral secondaire
- Individualisation de thérapie

Table 2 Proposed target values for some neuromonitoring modalities

	Normal	Desirable	Critical
ICP	~ 10 mmHg	< 18–22 mmHg	> 25 mmHg
CPP	50–60 mmHg	60– (80) mmHg	< 50 mmHg
PbtO ₂	~ 30 mmHg	20–25 mmHg	< 15 mmHg
Lactate/Pyruvate Ratio	< 25	< 25	> 40
Brain Glucose	> 1 mmol/l	> 0.8 mmol/l	< 0.5 mmol/l
Brain temperature	~ 36.5 °C	36.5–37 °C	> 37.5 °C

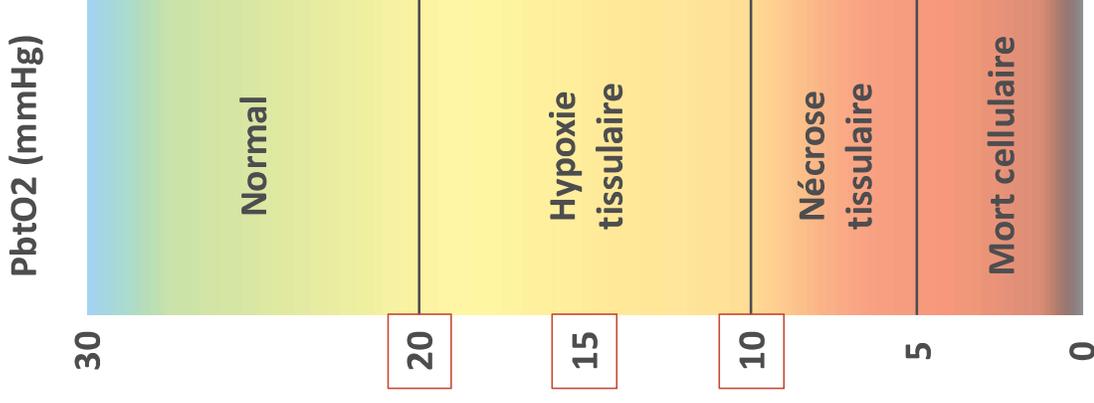
Neuromonitoring avancé: P_bO_2

- Électrode polarographique de Clark → pression tissulaire partielle O_2
 - Diffusion O_2 à travers membrane → dépolarisation → courant électrique
 - Mesure pression partielle O_2 dans volume de qq mm^3



Déterminants de $P_{bt}O_2$

- Livraison O_2 = contenu O_2 x débit
 - DSC (DC, PPC)
 - SaO₂
 - Hb
 - PaO₂
- Consommation O_2 = métabolisme
 - CMRO₂ – état d'éveil, agitation, convulsions, frissons
 - T°
- Diffusion tissulaire O_2
 - Œdème interstitiel
 - Dysfonction microvasculaire



Monitoring de l'oxygénation cérébrale

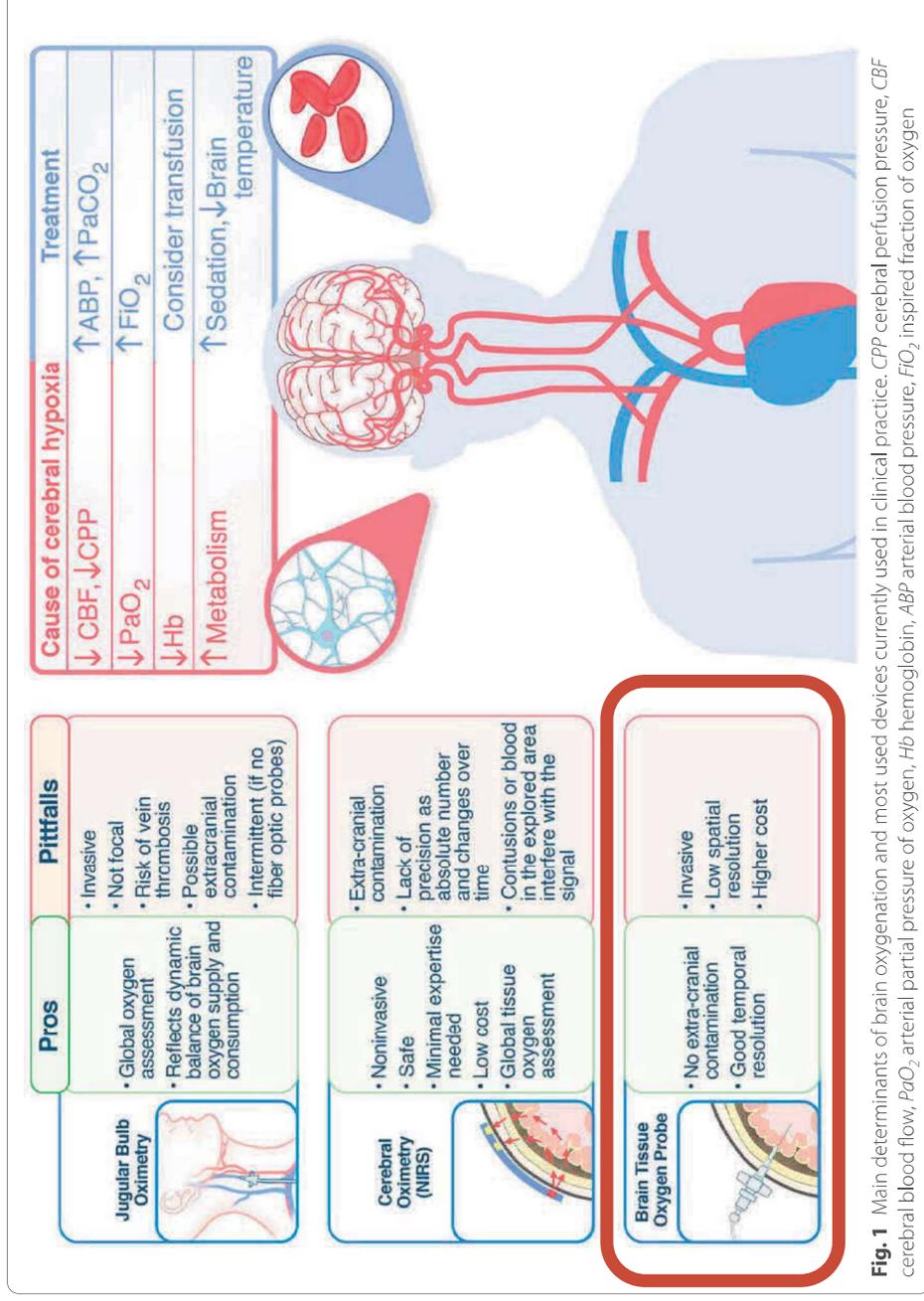
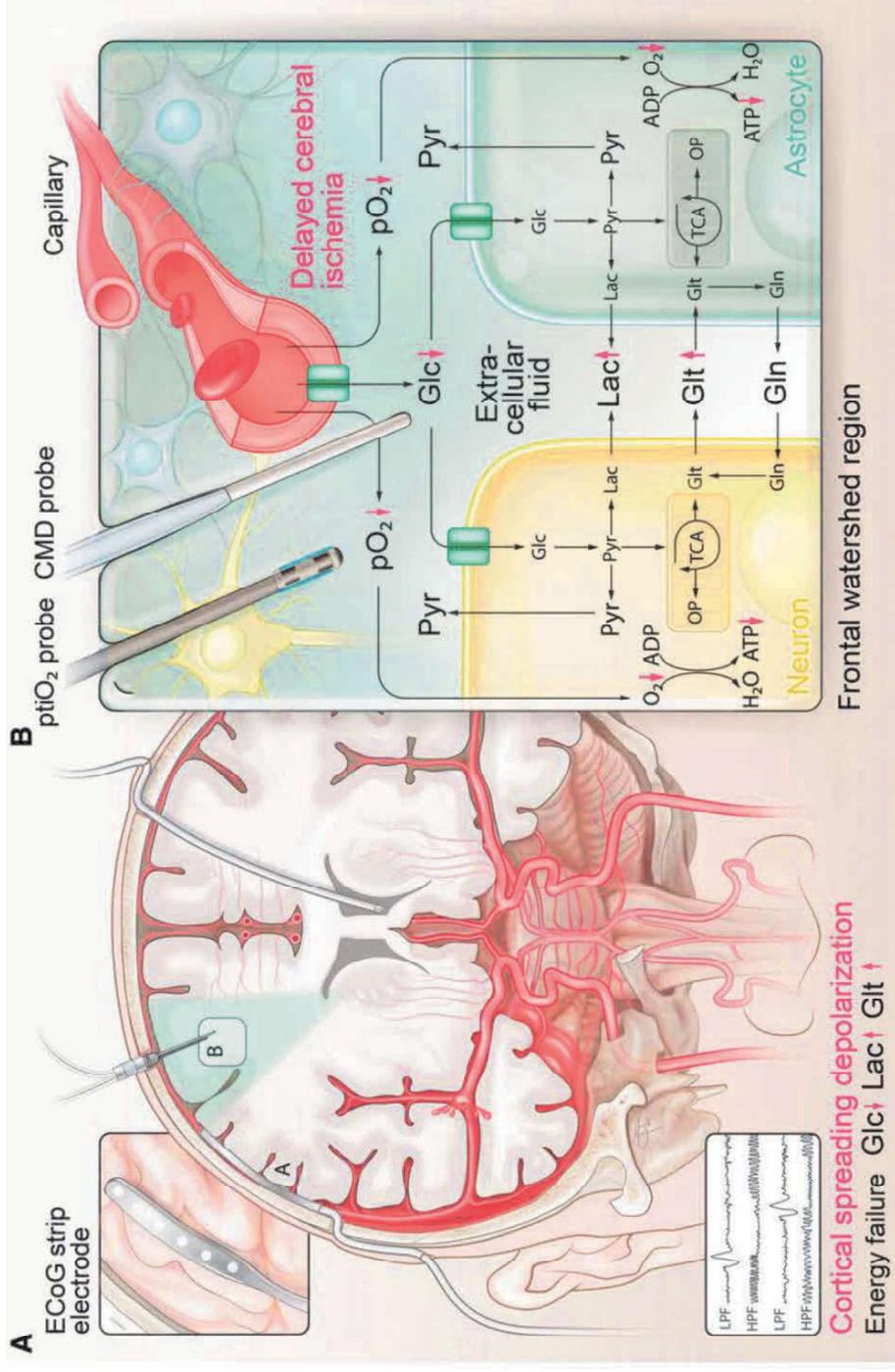


Fig. 1 Main determinants of brain oxygenation and most used devices currently used in clinical practice. CPP: cerebral perfusion pressure, CBF: cerebral blood flow, PaO₂: arterial partial pressure of oxygen, Hb: hemoglobin, ABP: arterial blood pressure, FIO₂: inspired fraction of oxygen

Neuromonitoring multimodal en HSA



Conclusion

- Considérations en HSA varient en fonction:
 - Sévérité du saignement
 - Sécurisation de l'anévrisme
 - Présence d'hydrocéphalie ou d'HTIC
 - Mécanisme d'HTIC
- Présence ou période à risque d'ischémie cérébrale retardée
- Étendue d'atteinte extra-cérébrale

En somme...

- Considérations à ajuster en fonction de pathologie et évolution
- Pas de « recette neuro »
- HTIC n'est pas invariablement présente
- Littérature en évolution
- Individualisation de thérapie
- Impact de pathologies extracrâniennes sur bien-être cérébral