FACCIA DA STROKE incontro su casi clinici di stroke

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Il ruolo dell’anestesista-rianimatore

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Alessandria

Qual è il ruolo dell’anestestista-rianimatore??

Partiamo da un caso clinico….

- Uomo di 56 anni
- APR: diabete non insulino-dipendente, ipertensione, fumatore, obesità (BMI 35)
- APP: improvvisa comparsa di afasia e deficit di forza all'emisoma dx durante l'attività lavorativa
- 118: GCS 13 (O4, V4, M5), PA 200/120 mmHg, FC 70, SpO_2 95% in aa. Somministrato ebrantil a boli refratti

- In PS:
  - GCS 13, PA 180/100 mmHg, FC 70, SpO_2 97% con 5 lt/min O_2
  - Controllo della PA: 160/80 mmHg
  - Valutazione neurologica:
    - NIHSS 14, deficit emisoma dx
    - Tac cranio: non lesioni recenti, dubbio spianamento dei solchi a sx
    - Non controindicazione a trombolisi ev (entro 3 ore)
  - Peggioramento delle condizioni cliniche: agitato, sudato, PA 200/100 mmHg
  - Ripete tac cranio+ angioTAC: non sanguinamento, occlusione ramo superiore ed inferiore di ACM sx
  - valutazione neuroradiologica: indicata angiografia e trombolisi meccanica

- Inizia procedura di trombolisi intrarteriosa
- GCS 12-13, PA 170/90 mmHg, FC 70, SpO_2 97% con O_2
- Durante la procedura GCS 9 (O2,V2, M5), desaturazione fino a SpO_2 85% con O_2
- Chiamata del rianimatore: GCS 8 (O1,V2,M5), pupille isocoriche, isocicliche
- Sedazione e tentativo di IOT
- Intubazione difficile, episodio di vomito, desaturazione fino a SpO_2 70%, parametri cardiocircolatori stabili
- Al 3° tentativo IOT, VAM con ripresa della saturazione fino a 95%

- Prosegue e termina la procedura con buon risultato
- TAC cranio post procedura: non emorragia
- Ricovero in terapia intensiva

…Tutto ciò che il rianimatore non vorrebbe.. E che per fortuna non succede..

..in realtà cosa accade…

- Valutazione in pronto soccorso
- Valutazione neurologica
- Iniziato trattamento trombolitico ev se indicato
- Valutazione del neuroradiologo
- Chiamata all'anestesista-rianimatore per l'assistenza durante la procedura di trombolisi intrarteriosa

- Cosa deve fare l'anestesista-rianimatore?

Partiamo dalle Linee Guida

Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association

(Stroke 2013;44:870-947)

Airway-Breathing-Circulation

Perché le vie aeree e il respiro?

Airway, Ventilatory Support, and Supplemental Oxygen

Stroke is a primary failure of focal tissue oxygenation and energy supply. Thus, it is intuitive that systemic hypoxemia and hypotension be avoided and, if present, corrected to limit further cellular damage. **Initial assessment of the airway,** breathing, and circulation occurs in the **prehospital setting** and again on arrival in the ED. **Constant reassessment of the airway, breathing,** and circulation is required to identify oxygen desaturation, respiratory compromise, and hypotension.

On the basis of these data, it is not apparent that routine supplemental oxygen is required acutely in nonhypoxic patients with mild or moderate strokes. **Supplemental oxygen may be beneficial in patients with severe strokes**

Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association.

recommend administration of oxygen to hypoxemic patients to maintain **oxygen saturation >94%.**[15] When oxygen therapy is indicated, it is reasonable to use the least invasive method possible to **achieve normoxia**. Available methods include nasal cannula, Venturi mask, nonrebreather mask, bilevel positive airway pressure, continuous positive airway pressure, or endotracheal intubation with mechanical ventilation.

Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association.

- No clinical trial has tested the utility of endotracheal intubation in the management of critically ill patients with stroke. It is generally agreed that **endotracheal intubation** and mechanical ventilation should be performed if the airway is threatened. Evidence suggests that prevention of early aspiration reduces the incidence of pneumonia,[383] and protection of the airway may be an important approach in certain patients. Endotracheal intubation and mechanical ventilation may also assist in the management of elevated ICP or malignant brain edema after stroke.[378,384] The need for intubation has prognostic implications. Although a small percentage of patients may have a satisfactory outcome after intubation,[385] the overall prognosis of intubated stroke patients is poor, with up to 50% mortality within 30 days after stroke.[386–388]

Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association.

Airway support and ventilatory assistance are recommended for the treatment of patients with acute stroke who have decreased consciousness or who have bulbar dysfunction that causes compromise of the airway *(Class I; Level of Evidence C)*.(Unchanged from the previous guideline[13])

- l’ipossia può essere causata da:
  - Ostruzione parziale delle vie aeree (caduta della lingua)
  - Ipoventilazione
  - Aspirazione (assenza del riflesso della tosse)
  - Atelettasie
  - Polmoniti
  - Depressione del drive respiratorio
- Conseguenza dell’ipoventilazione è l’ipercapnia con conseguente vasodilatazione ed aumento della pressione intracranica

Perché il circolo?

Cardiac Monitoring

Cardiac monitoring begins in the prehospital setting and continues throughout the initial assessment and management of acute stroke. As mentioned before, continuous cardiac monitoring is indicated for at least the first 24 hours after stroke.[136,405,406] Recent studies have suggested Holter monitoring is more effective in identifying atrial fibrillation or other serious arrhythmias after stroke.[134]

In patients with markedly elevated blood pressure who do not receive fibrinolysis, a reasonable goal is to lower blood pressure by 15% during the first 24 hours after onset of stroke. The level of blood pressure that would mandate such treatment is not known, but consensus exists that medications should be withheld unless the systolic blood pressure is >220 mm Hg or the diastolic blood pressure is >120 mm Hg *(Class I; Level of Evidence C)*. (Revised from the previous guideline[13])

Hypovolemia should be corrected with intravenous normal saline, and cardiac arrhythmias that might be reducing cardiac output should be corrected *(Class I; Level of Evidence C)*. (Revised from the previous guideline[13])

- Airway-Breathing-Circulation…sono trattati e sotto controllo

Anestesia generale? O sedazione?
Estenuante dibattito

rianimatore	neuroradiologo
Paziente sedato ed intubato	Paziente non intubato
Maggiore stabilità di parametri	Osservare miglioramenti clinici
Non rischio di inalazione	Paziente “immobile”

Cosa dice la letteratura?

- Studi osservazionali
- Studi non randomizzati
- Rewiew

- Discussione continua

Conscious Sedation Versus General Anesthesia During Endovascular Therapy for Acute Anterior Circulation Stroke

Preliminary Results From a Retrospective, Multicenter Study

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Marilyn M. Rymer, MD; Ashis H. Tayal, MD; Osama O. Zaidat, MD, MS;
Sabareesh K. Natarajan, MD, MS; Raul G. Nogueira, MD; Ashish Nanda, MD; Melissa Tian, RN;
Qing Hao, MD, PhD; Junaid S. Kalia, MD; Thanh N. Nguyen, MD; Michael Chen, MD; Rishi Gupta, MD

Conclusions—Patients placed under GA during IAT for anterior circulation stroke appear to have a higher chance of poor neurologic outcome and mortality. There do not appear to be differences in hemorrhagic complications between the 2 groups. Future clinical trials with IAT can help elucidate the etiology of the differences in outcomes. (***Stroke*. 2010; 41:1175-1179.**)

Impact of Anesthesia on Mortality During Endovascular Clot Removal for Acute Ischemic Stroke

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Vladyslav Melnyk, BS,† Reza Gorji, MD,* and Zhong-jin Yang, MD**

Mortalità > nei pazienti trattati con AG

CRITICAL CARE MEDICINE

Anesthetic Management and Outcome in Patients during Endovascular Therapy for Acute Stroke

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In this cohort of patients the main predictor of neurologic outcome, the baseline stroke severity, was worse in patients who received general anesthesia (fig. 2). This finding is not

In conclusion, our retrospective review has replicated previous reports that patients managed with general anesthesia, and its concomitant relative systolic hypotension, during endovascular therapy for acute ischemic stroke have a much lower likelihood of good neurologic outcome, compared to patients managed with local anesthesia. Since avoidance of

GA: prognosi peggiore,
causa ipotensione

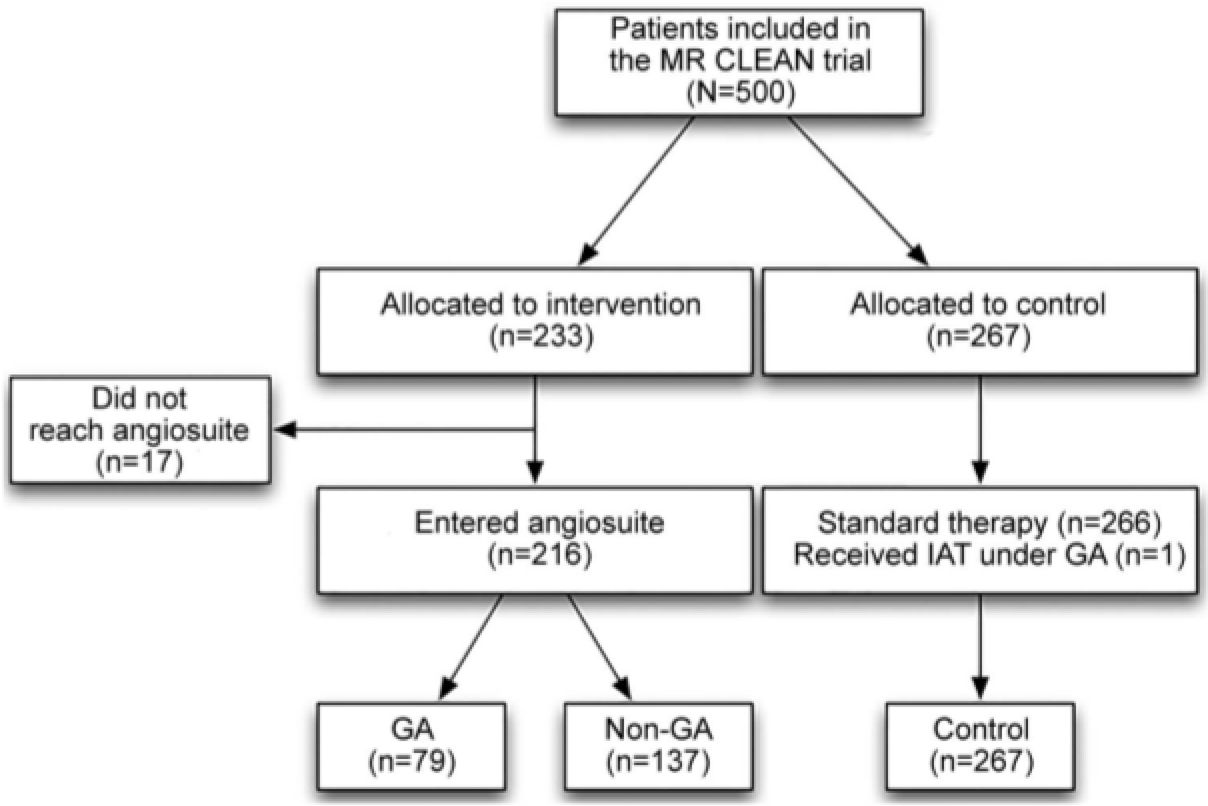
The effect of anesthetic management during intra-arterial therapy for acute stroke in MR CLEAN

Neurology® 2016;87:656-664

The Multicenter Randomized Clinical Trial of Endovascular Treatment for Acute Ischemic Stroke in the Netherlands (MR CLEAN) showed a clear benefit of intra-arterial therapy (IAT) in patients with acute ischemic stroke (AIS) caused by a proximal intracranial occlusion of the anterior circulation.[1] The intervention contrast was IAT vs no IAT against a background of best medical care including IV alteplase if indicated. The trial demonstrated a shift in the distribution of functional outcomes on the modified Rankin Scale (mRS) in favor of the intervention, which was consistent in almost all subgroup analyses. With IAT, the rate of als. Reported outcomes are better when IAT is performed without general anesthesia (non-GA), which means applying local anesthesia in the groin, with or without subsequent use of conscious sedation.[7–9] The non-GA approach may lead to faster treatment initiation and may avoid complications associated with intubation. Furthermore, it is known that most anesthetic agents used for GA induce sympathicolysis, which may lead to hypotension and decreased cerebral perfusion.[10,11] On the other hand, GA reduces patient movement during the procedure and may therefore decrease procedural times and procedure-related complications. The choice between

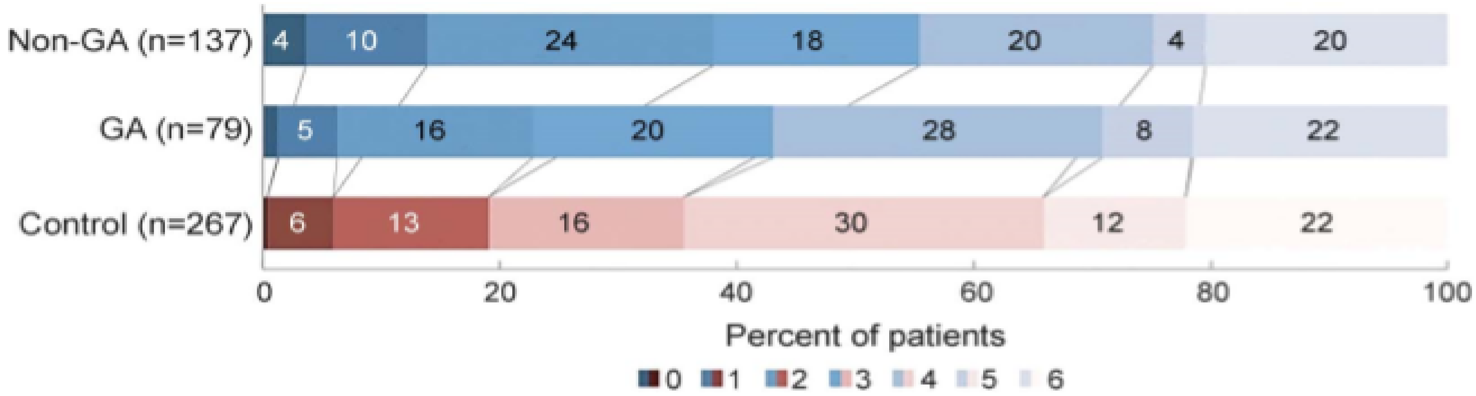
- Outcome migliore in non-GA, non complicanze legate a IOT, ipotensione
- GA: pz immobile, ridotto tempo di procedura

Figure 1 **Flow diagram of patients included in Multicenter Randomized Clinical Trial of Endovascular Treatment for Acute Ischemic Stroke in the Netherlands (MR CLEAN)**



Patients included in MR CLEAN subdivided into intra-arterial therapy (IAT) under general anesthesia (GA), intra-arterial treatment without general anesthesia (non-GA), and control arms of the trial.

Figure 2 **Modified Rankin Scale (mRS) score at 90 days by intention-to-treat groups**



The number and percentages of patients are shown in each cell according to distribution of the mRS (range 0–6, with 0 indicating no symptoms, 1 no clinically significant disability, 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 and attention], and 6 dead). We found a shift in the distribution on the mRS in favor of the non-general anesthesia (GA) group compared to the Multicenter Randomized Clinical Trial of Endovascular Treatment for Acute Ischemic Stroke in the Netherlands (MR CLEAN) control group (adjusted common odds ratio [acOR] 2.18 [95% confidence interval (CI) 1.49–3.20]). The shift in distribution between GA and the MR CLEAN control group was in a similar direction (acOR 1.12 [95% CI 0.71–1.78]).

Nevertheless, there are patients who will need GA due to a decreased level of consciousness for airway protection or poor cooperation due to agitation or pain.[6] Consequently, it is important to elucidate the mechanisms by which GA negates the benefits of IAT. A recent study suggests that this detrimental effect is ultimately mediated through infarct growth.[16] The authors

One potential mechanism by which GA may be related to infarct growth and worse outcome is the time delay associated with intubation. In MR CLEAN, the time from randomization to groin puncture was 16 minutes longer in the GA group on average. However, this did not translate to a significant difference in the time from onset to revascularization. Another potential mechanism is uncorrected anesthesia-induced blood pressure reduction and altered cerebral hemodynamics at the introduction of GA and during GA. Most anesthetic agents, for example, propofol or induction dosages of thiopental, are known to be associated with hypotension.[17] Lower mean arterial blood pressures have been associated with poorer clinical outcomes in patients treated under GA compared to conscious sedation.[11,18] Furthermore, anesthetic agents may cause disturbance in cerebral autoregulation, potentially impairing cerebral blood flow and perfusion.[10] The effect of anesthetic

Another potential disadvantage of GA management is the increased risk of aspiration and at times aspiration pneumonia following intubation.[7,19] On the other hand, emergency intubation may still be required during the non-GA approach, possibly accompanied by even a higher risk of aspiration and airway trauma.[19,16]

Necessità di GA in certi pz
Ipotensione intraprocedura
Rischio VAP
Rischio IOT in emergenza

Effect of Conscious Sedation vs General Anesthesia on Early Neurological Improvement Among Patients With Ischemic Stroke Undergoing Endovascular Thrombectomy
A Randomized Clinical Trial

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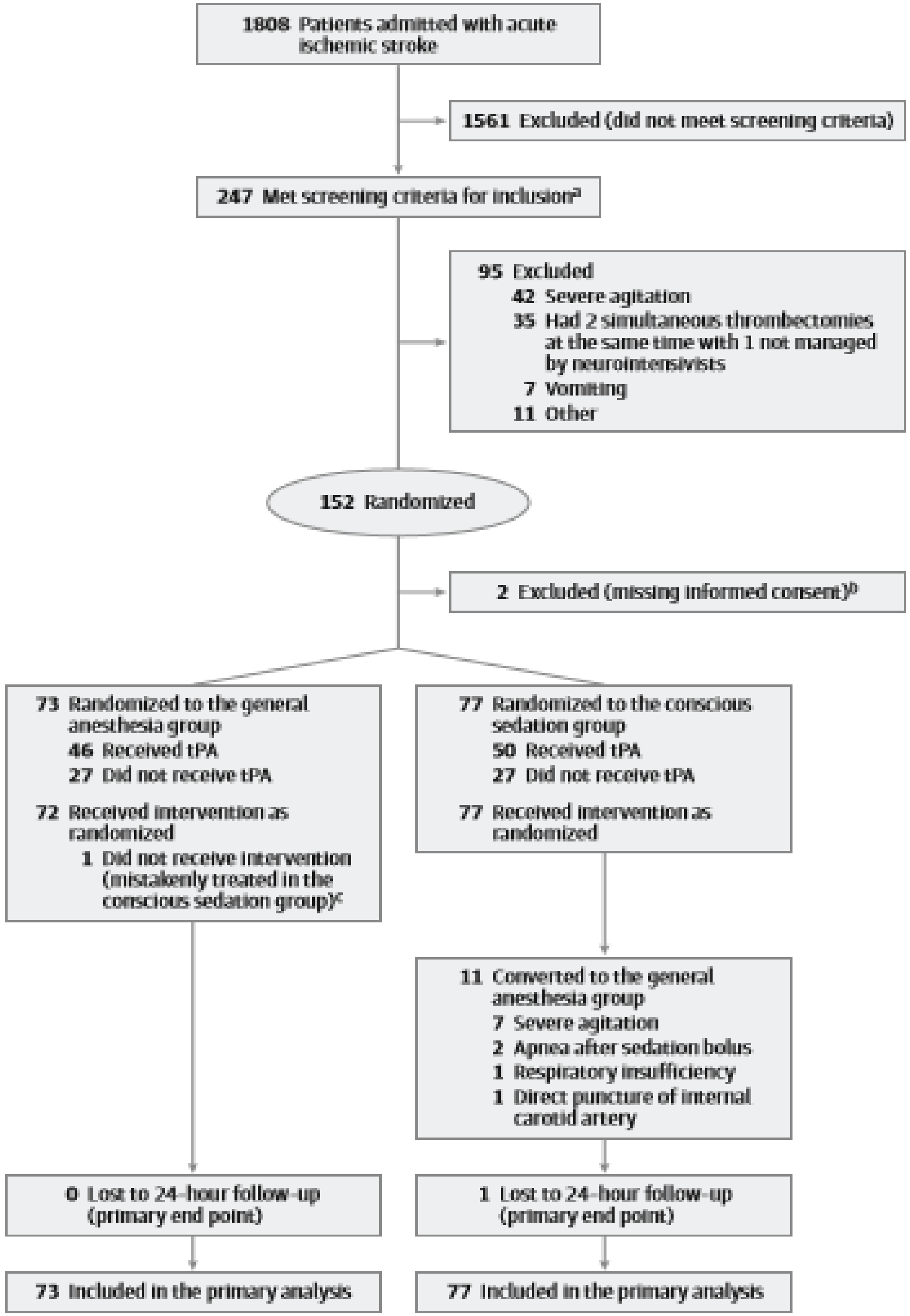
- 150 pazienti con ASI
- 73 anestesia generale
- 77 sedazione (di cui 11 convertiti in anestesia generale)
- Criteri di esclusione: immagini radiologiche non chiare, non interessamento della carotide interna o della ACM, segni di emorragia, GCS<8, severa agitazione con impossibilità a reperire accesso vascolare, perdita dei riflessi, difficoltà alla gestione vie aeree, allergie a farmaci sedativi, non IOT all'ammissione



1808 Patients admitted with acute ischemic stroke
1561 Excluded (did not meet screening criteria)
247 Met screening criteria for inclusion[a]
95 Excluded
42 Severe agitation
35 Had 2 simultaneous thrombectomies at the same time with 1 not managed by neurointensivists
7 Vomiting
11 Other
152 Randomized
2 Excluded (missing informed consent)[b]
73 Randomized to the general anesthesia group
46 Received tPA
27 Did not receive tPA
72 Received intervention as randomized
1 Did not receive intervention (mistakenly treated in the conscious sedation group)[c]
77 Randomized to the conscious sedation group
50 Received tPA
27 Did not receive tPA
77 Received intervention as randomized
11 Converted to the general anesthesia group
7 Severe agitation
2 Apnea after sedation bolus
1 Respiratory insufficiency
1 Direct puncture of internal carotid artery
0 Lost to 24-hour follow-up (primary end point)
1 Lost to 24-hour follow-up (primary end point)
73 Included in the primary analysis
77 Included in the primary analysis

- Durante il trattamento:
  - Monitoraggio parametri cardiorespiratori
  - Utilizzo degli stessi farmaci nei due gruppi a dosi differenti
  - In caso di necessità, immediata conversione di CS a AG
  - Al termine: stroke unit o ICU con risveglio appena possibile
- Primary outcome: variazione NIHSS a 24 ore
- Secondary outcomes: mRS a 3 mesi, mortalità intraH e a 3 mesi, valutazione cliniche durante la procedura (iper/ipotensione, ossigenazione, ventilazione, complicanze procedurali)

Table 2. Clinical Characteristics Assessed at Admission

Characteristic	No. (%) General Anesthesia (n = 73)	No. (%) Conscious Sedation (n = 77)
Pretreatment imaging		
CT	57 (78.1)	57 (74.0)
MRI	10 (13.7)	16 (20.8)
CT and MRI	6 (8.2)	4 (5.2)
ASPECTS[a]		
10-8	52 (71.2)	42 (56.7)
7-6	16 (21.9)	24 (32.5)
<6	5 (6.8)	8 (10.8)
Median (IQR)	8 (7-9)	8 (6.25-9)
Missing data	0	3
Premorbid modified Rankin Scale[b]		
0	40 (54.8)	39 (50.6)
1	14 (19.2)	19 (24.7)
2	10 (13.7)	13 (16.9)
>2	9 (12.3)	6 (7.8)
NIHSS[c]		
Mean (SD)	16.8 (3.9)	17.2 (3.7)
Median (IQR)	17 (13-20)	17 (14-20)
Glasgow Coma Scale[d]		
12	6 (8.2)	3 (3.9)
13	38 (52.1)	41 (53.2)
14-15	29 (39.8)	33 (42.9)
Occlusion		
Middle cerebral artery	46 (63.0)	47 (61.0)
M1	39 (53.4)	43 (55.8)
M2	7 (9.6)	4 (5.2)
Internal carotid artery	1 (1.4)	9 (11.7)
Internal carotid and middle cerebral artery	26 (35.6)	21 (27.3)
Internal carotid artery and M1	25 (34.2)	21 (27.3)
Internal carotid artery and M2	1 (1.4)	0
Right-side occlusion	28 (38.4)	35 (45.5)
Reperfusion treatments		
Intravenous thrombolysis and endovascular stroke treatment	46 (63.0)	50 (64.9)
Endovascular stroke treatment alone	26 (35.6)	27 (35.1)
No intervention	1 (1.4)	0
Types of endovascular stroke treatment		
Stent retriever	60 (82.2)	66 (85.7)
Direct aspiration	6 (8.2)	4 (5.2)
Cervical stent/angioplasty[e]	16 (21.9)	12 (15.6)
Onset-to-door time, mean (SD), min	145.0 (83.8)	118.1 (61.5)

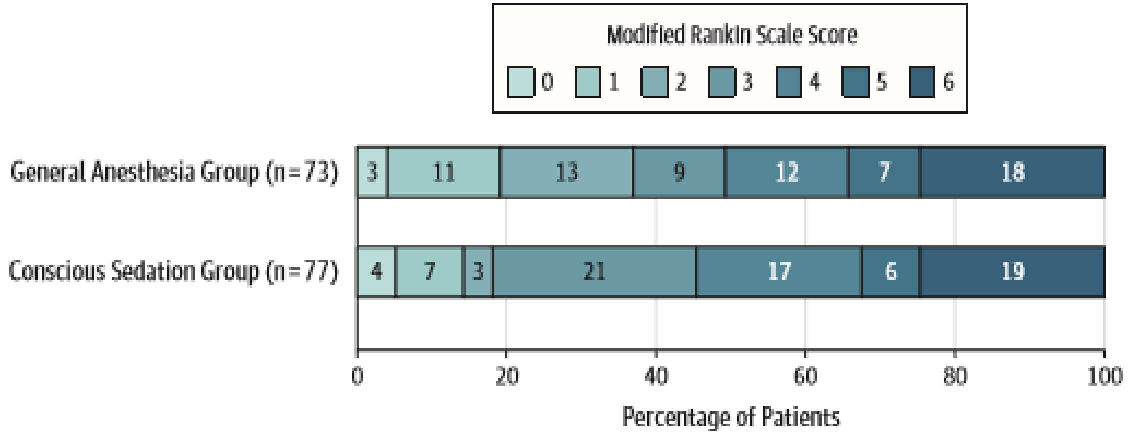
Table 3. Primary and Secondary Outcome Results

Variable	General Anesthesia (n = 73)	Conscious Sedation (n = 77)	Difference (95% CI)	P Value[a]
Primary Outcome				
Change in NIHSS[b], mean (95% CI)	−3.2 (−5.6 to −0.8)	−3.6 (−5.5 to −1.7)	−0.4 (−3.4 to 2.7)	.82[c]
Change in NIHSS, median (IQR)	−5.0 (−10.0 to 2.0)	−4.0 (−10 to 2.0)		
NIHSS after 24 h, mean (SD)	13.6 (11.1)	13.6 (9.0)	0.0 (−3.3 to 3.3)	>.99[d]
Secondary Outcomes				
Clinical				
Modified Rankin Scale after 3 mo, mean (SD)	3.5 (1.9)	3.7 (1.8)	0.2 (3.3 to 3.9)	.41[e]
Modified Rankin Scale after 3 mo, median (IQR)	4 (2 to 5)	4 (3 to 5)		
Modified Rankin Scale 0-2 after 3 mo, No. (%)	27 (37)	14 (18.2)	−18.8 (−32.8to−4.8)	.01[f]
In-hospital mortality, No. (%)	5 (6.8)	6 (7.8)	0.9 (−7.4 to 9.3)	.83[f]
Mortality after 3 mo, No. (%)	18 (24.7)	19 (24.7)	0.0 (−13.8 to 13.8)	>.99[f]

Length of stay in hospital, d	5.9 (4.2)	5.2 (4.0)	−0.7 (−2.0 to 0.6)[g]	.19[e]
Length of stay in intensive care unit, h	71.6 (106.4)	54.5 (82.2)	−17.0 (−50.4 to 15.0)[g]	.23[e]
Length of ventilation, h[h]	30.9 (90.9)	45.4 (126.8)	14.5 (−31.6 to 173.3)[g]	.91[e]
Length of stay on stroke unit, h[i]	87.4 (51.2)	85.4 (46.9)	−2.0 (−20.9 to 15.3)[g]	.89[e]
Door-to-arterial puncture time, min	75.6 (29.3)	65.6 (19.9)	−10.0 (−19.2 to −2.9)[g]	.03[e]
Door-to-reperfusion time, min[j]	165.2 (59.4)	174.4 (56.3)	9.2 (−10.0 to 28.5)[g]	.29[e]
Duration of EST, min	111.6 (62.5)	129.9 (62.5)	−18.2 (−38.4 to 2.0)	.04[e]
Feasibility of EST, No. (%)[k]				
Reperfusion grade (TICI)[l]				
0-1	4 (5.5)	7 (9.1)		.68[l]
2a	4 (5.5)	8 (10.4)		
2b	30 (41.1)	36 (46.8)		
3	35 (47.9)	26 (33.8)		
Substantial reperfusion grade 2b-3 (TICI)	65 (89.0)	62 (80.5)	−8.5 (−19.9 to 2.9)[l]	
Substantial patient movement[m]	0	7 (9.1)	9.1 (2.7 to 15.5)	.01[f]
Difficult vascular approach[n]	6 (8.2)	9 (11.7)	3.5 (−6.1 to 13.0)	.48[f]
Other[o]	5 (7.2)	1 (1.4)	−5.8 (−12.5 to 0.9)	.09[f]

Variable	General Anesthesia (n = 73)	Conscious Sedation (n = 77)	Difference (95% CI)	*P* Value[a]
Safety, No. (%)				
Complications before EST				
Incomplete cardiovascular monitoring	1 (1.4)	0	−1.4 (−4.0 to 1.3)	.30[f]
Difficulties of arterial puncture	0	1 (1.3)	1.3 (−1.2 to 3.8)	.33[f]
Other complications[p]	1 (1.4)	0	−1.4 (−4.0 to 1.3)	.30[f]
Complications during EST				
Critical hypertension or hypotension (>180 mm Hg or <120 mm Hg)	2 (2.7)	0	−2.7 (−6.5 to 1.0)	.14[f]
Critical ventilation or oxygenation disturbance[q]	3 (4.1)	3 (3.9)	−0.2 (−6.5 to 6.1)	.95[f]
Intervention-associated complications	2 (2.7)	2 (2.6)	−0.1 (−5.3 to 5.0)	.96[f]
Vessel perforation with ICH, SAH, or both	1 (1.4)	2 (2.6)	1.2 (−3.2 to 5.7)	.59[f]
Allergic reaction after application of contrast agent	1 (1.4)	0	−1.4 (−4.0 to 1.3)	.30[f]
Complications after EST				
Hypertension or hypotension (>180 mm Hg or <120 mm Hg)	17 (23.3)	10 (13.0)	−10.3 (−22.6 to 2.0)	.10[f]
Hyperthermia or hypothermia (>37.2°C or <36.0°C)	24 (32.9)	7 (9.1)	−23.8 (−36.3 to −11.2)	<.001[f]
Delayed extubation[r]	36 (49.3)	5 (6.5)	−42.8 (−55.5 to −30.1)	<.001[f]
Ventilation-associated complications[s]	10 (13.7)	3 (3.9)	−9.8 (−18.8 to −0.8)	.03[f]

Figure 3. Functional Outcome at 90-Day Follow-up in the Intent-to-Treat Population



Modified Rankin Scale range, 0 to 6 (0, no symptoms; 1, no clinically relevant disability; 2, slight disability [able to look after own affairs without assistance but not to the full extent]; 3, moderate disability [requires some help but able to walk unassisted]; 4, moderately severe disability [requires assistance and unable to walk unassisted]; 5, severe disability [requires constant nursing care]; 6, dead). Distribution of modified Rankin Scale categories were additionally tested using the Mann-Whitey *U* (Wilcoxon) statistic (*P* = .41).

- La CS è migliore della GA?

The findings of SIESTA were in contrast to data from several retrospective studies that strongly suggested that general anesthesia for thrombectomy decreases neurological recovery and increases morbidity and mortality compared with conscious sedation.[14,31] Pathophysiological considerations sup-

The main objectives of SIESTA were to determine whether conscious sedation was superior to general anesthesia with regard to early neurological improvement and to assess short-term differences of the peri-interventional clinical course, feasibility, and safety between conscious sedation and general anesthesia in a balanced group of patients with major ischemic stroke undergoing thrombectomy. The patient population

- Non supporta la superiorità della CS rispetto alla GA (morbidità e mortalità)
- Meglio sicuramente, se possibile, iniziare con la CS
- Limiti:
  - Singolo centro
  - Campione piccolo
  - NIHSS a 24 ore troppo precoce (pazienti non sempre valutabili)

These findings suggest that, clinically, both peri-interventional regimens for thrombectomy appear applicable, provided strict protocols are in place and performed by physicians trained in this setting. To start the thrombectomy procedure using conscious sedation may allow better clinical monitoring of recanalization success or complications and may save time.[41] This approach appears reasonable as long as contraindications, such as severe agitation, vomiting, or coma with loss of airway-protective reflexes are absent and an immediate conversion to general anesthesia is possible. In that case

General Anesthesia Versus Conscious Sedation for Endovascular Treatment of Acute Ischemic Stroke

The AnStroke Trial (Anesthesia During Stroke)

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(*Stroke*. 2017;48:1601-1607. DOI: 10.1161/STROKEAHA.117.016554.)

Endovascular treatment (EVT) combined with intravenous thrombolysis for acute ischemic stroke (AIS) has been proven superior to intravenous thrombolysis alone.[1] During the EVT, it is necessary that the patient is immobile, and usually some type of anesthesia/sedation is required. The 2 main alternatives are general anesthesia (GA) with intubation that gives a total control of airway, ventilation, and patient movements and, second, conscious sedation (CS) with the patient spontaneously breathing and to some extent communicable and hemodynamically more stable.[2]

The question whether choice of anesthesia technique has an impact on neurological outcome after EVT for AIS has been debated. Seventeen retrospective studies have been published on the subject, all showing better neurological outcome after CS compared with GA.[3–19] However, all retrospective studies experience selection bias, with the GA group having higher

We therefore set up the AnStroke trial (Anesthesia During Stroke) as a randomized controlled single-center study, randomizing patients with anterior AIS to GA or CS, aiming for a strict periprocedural blood pressure control and normoventilation.

AG	CS
• Propofol e remifentanil • Sevorane e remifentanil • Normoventilazione • PAS, PAD, PAM ogni 5 min • PAS 140-180 mmHg prima della ricanalizzazione • Amine se necessario • PAM baseline: prima dell'induzione	• Remifentanil • PAS, PAD, PAM ogni 5 min • PAS 140-180 mmHg prima della ricanalizzazione • Amine se necessario • PAM baseline: prima dell'induzione

Table 1. Patient Characteristics

	GA	CS
	n=45	n=45
Age, y	73 (65–80)	72 (66–82)
Male sex, n (%)	26 (58)	23 (51)
Comorbidities, n (%)		
Hypertension	27 (60)	22 (49)
Atrial fibrillation	18 (40)	18 (40)
Ischemic heart disease	9 (20)	5 (11)
Diabetes mellitus	9 (20)	7 (16)
Smoker	4 (9)	8 (18)
Hyperlipidemia	5 (11)	7 (16)
Premorbid modified Rankin Scale score ≤2, n (%)	44 (98)	44 (98)

CS indicates conscious sedation; and GA, general anesthesia.

Table 2. Stroke Characteristics

	GA	CS
	n=45	n=45
NIHSS score	20 (15.5–23)	17 (14–20.5)
Intravenous thrombolysis treatment, n (%)	33 (73.3)	36 (80)
Occlusion site, n (%)		
Carotid-T occlusion	15 (33)	9 (20)
Distal ICA	0 (0)	1 (2.2)
First segment of MCA (M1)	26 (58)	26 (58)
First segment of MCA (M1)+distal branches (A2, A3, M2, and M3)	4 (9)	1 (2)
Second segment of MCA (M2)	0 (0)	8 (18)
Left hemisphere, n (%)	26 (58)	17 (38)
ASPECTS score, 1–10	10 (8–10)	10 (9–10)
Collateral circulation, 1–5	2 (1–4)	1 (1–3)

ASPECTS indicates Alberta Stroke Program Early CT score; CS, conscious sedation; GA, general anesthesia; ICA, internal carotid artery; MCA, middle cerebral artery; and NIHSS; National Institutes of Health Stroke Scale.

	n=45	n=45	*P* Value
Time intervals, min			
From stroke onset to CT	97 (62–160)	72 (58–119)	0.2523
From stroke onset to groin puncture	183 (135–279)	180 (137–252)	0.6141
From stroke onset to recanalization/end of procedure	254 (206–373)	250 (213–316)	0.7833
From CT to groin puncture	92 (68–121)	91 (55–123)	0.9376
From CT to recanalization/end of p			
From arrival to neurointerventional			
From groin puncture to recanalizati			

Hemodynamic and respiratory data			
Baseline MAP before induction of anesthesia, mm Hg	105±16	108±17	0.4086
Blood pressure intraoperative			
MAP, mm Hg	91±8	95±11	0.0484
MAP, fraction of baseline MAP	0.88±0.10	0.89±0.9	0.5678
Highest MAP, mm Hg	116±15	114±15	0.5631
Lowest MAP, mm Hg	68±12	77±15	0.0015
Lowest MAP, fraction of baseline MAP	0.65±0.11	0.72±0.15	0.0125
Occurrence of >20% fall in MAP compared with baseline, n (%)	41 (93)	26 (60)	0.0003
Time spent with >20% fall in MAP compared with baseline, min	22 (5–57)	15 (0–55)	0.0432
Occurrence of >40% fall in MAP compared with baseline, n (%)	15 (34)	9 (21)	0.2311
Time spent with >40% fall in MAP compared with baseline, min	0 (0–9)	0 (0–0)	0.1167
Use of vasoactive drugs, n (%)	43 (98)	34 (79)	0.0073
Pao_2, kPa	17.2 (11.6–25.1)	11.2 (9.7–14.7)	<0.0001
Hemoglobin oxygen saturation, %	98±1	96±2	<0.0001
Hemoglobin concentration, g/L	128±14	131±17	0.4488
$PaCO_2$, kPa	5.4 (4.8–5.9)	5.2 (4.6–6.0)	0.5732
Blood glucose, mmol/L	7.0 (6.2–8.5)	6.9 (6.2–8.1)	0.8014

	GA	CS	
	n=45	n=45	*P* Value
mTICI 2b–3=successful recanalization, n (%)	41 (91.1)	40 (88.9)	1.000
NIHSS score after 24 h	8 (3–15)	9 (2–15)	0.5986
NIHSS score shift 24 h postoperatively	9 (4–17)	8 (2.5–13)	0.2723
NIHSS score at day 3	6 (2–14)	3 (0.5–10)	0.1685
NIHSS score shift at day 3	10 (6–18)	12 (5.5–16)	0.8272
NIHSS score at hospital discharge or at day 4–7	3 (1–10)	2 (0–9.5)	0.2484
NIHSS score shift at hospital discharge or at day 4–7	12 (9–19)	13 (9–16)	0.5713
MRI day 3 ASPECTS score	6 (3–7)	6 (4.5–7)	0.3837
MRI day 3 cerebral infarction volume, mL	20 (10–100)	20 (9–55)	0.5320
Hospital mortality, n (%)	6 (13.3)	6 (13.3)	1.000
mRS score at 3 mo	3 (1–4)	3 (1–5.5)	0.5001
mRS score at 3 mo ≤2, n (%)	19 (42.2)	18 (40.0)	1.000
Mortality at 3 mo, n (%)	6 (13.3)	11 (24.4)	0.2813
New stroke detected clinically and with MRI/CT at 3 mo	3 (6.7)	5 (11.1)	0.7136
Complications			
Symptomatic intracerebral hemorrhage*	0 (0)	3(6.7)	0.2416
Anesthesiological complications, n (%)	2 (4.4)	4 (8.9)	0.6766
Interventional complications, n (%)	11 (24.4)	6 (13.3)	0.4299

ASPECTS indicates Alberta Stroke Program Early CT score; CT, computed tomography; MRI, magnetic resonance imaging; mRS; modified Rankin Scale; mTICI, modified thrombolysis in cerebral ischemia; and NIHSS, National Institutes of Health Stroke Scale.

…quindi…..
Anestesia generale

- Ritarda tempo di IAT
- Può causare alterazioni emodinamiche: tachicardia, ipertensione, ipotensione, aritmie
- Variazioni dell'autoregolazione cerebrale con riduzione del flusso e rischio di aumento della lesione ischemica
- Brivido postoperatorio causa discomfort con aumentato consumo di ossigeno
- Elevato rischio di polmonite associata a VAM
- Meno dolore agitazione, movimenti e ridotto rischio di aspirazione

....e....
Conscious sedation

- Inizio più rapido della IAT risparmio tempo
- Monitoraggio dei miglioramenti clinici del paziente o l’insorgenza di nuovi deficit neurologici (emboli, formazione di trombi, progressione dell’ischemia, sviluppo di ematoma con effetto massa)
- Monitoraggio continuo del paziente può dare indicazioni all’operatore (quando terminare la procedura: ricanalizzazione o no)
- Possono esserci movimenti, fastidio, agitazione, vomito, eventuale IOT in emergenza
- Rischio basso di complicanze legate alla ventilazione
- Minor rischio di ipotensione

Conclusioni

- Lavoro di equipe
- Anestesista–rianimatore sempre presente..il paziente può sembrare stabile ma potrebbe peggiorare improvvisamente (caduta del GCS, sanguinamento, insufficienza respiratoria…)
- Scelta dell'anestesia non può essere stabilita con protocolli standardizzati ma deve essere valutata caso per caso


GRAZIE