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NECKER-ENFANTS MALADES

SFAR le CONGRÈS du 23 au 25 Septembre 2021 PALAIS DES CONGRÈS DE PARIS

Necker ENFANTS MALADES HÔPITAL UNIVERSITAIRE

Physiopathologie en pédiatrie : 10 minutes pour comprendre Le choc hémorragique chez l'enfant

Pr. Gilles Orliaguet

Aucun lien d'intérêt

ADARPEF

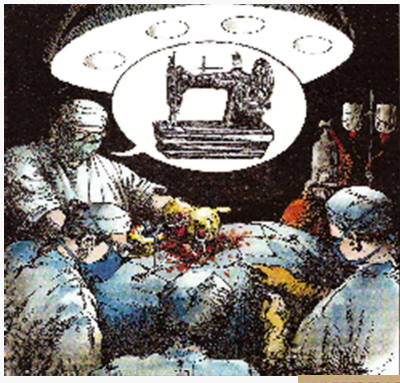


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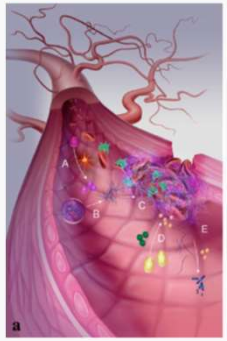
Choc hémorragique : Définition





Insuffisance circulatoire aiguë

- Caractérisée par un transport d'oxygène insuffisant pour assurer l'oxygénation tissulaire
- Due à une réduction aiguë du volume sanguin circulant



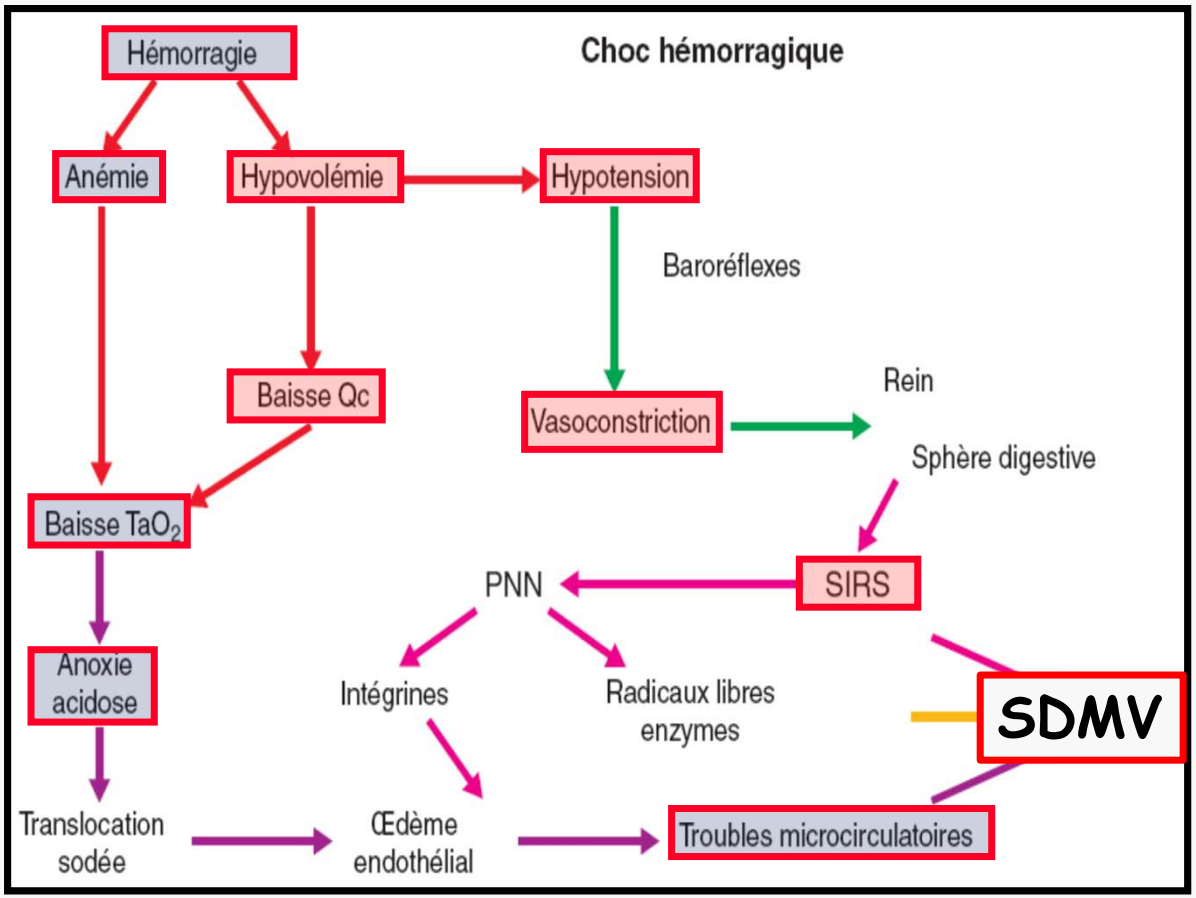


Physiopathologie du choc hémorragique chez l'enfant

- Retentissement macrocirculatoire
- Retentissement microcirculatoire
- Retentissement sur l'hémostase
- Retentissement pharmacologique

Stratégie du remplissage vasculaire en traumatologie

Mion G. EMC Médecine d'Urgence 2007. 25-200-B-40



Hémorragie

$\downarrow CaO_2 = 1.36 \times [Hb] \times SaO_2 + [0.003 \times PaO_2]$

$\downarrow Qc = \downarrow VES \times FC$

$\downarrow TaO_2 = \downarrow Qc \times \downarrow CaO_2$

THE LANCET] ORIGINAL ARTICLES [APRIL 15, 1944

POSTHAEMORRHAGIC FAINTING.
Study by Cardiac output and forearm flow.
Barcroft H et al.

Saignée — Syncope

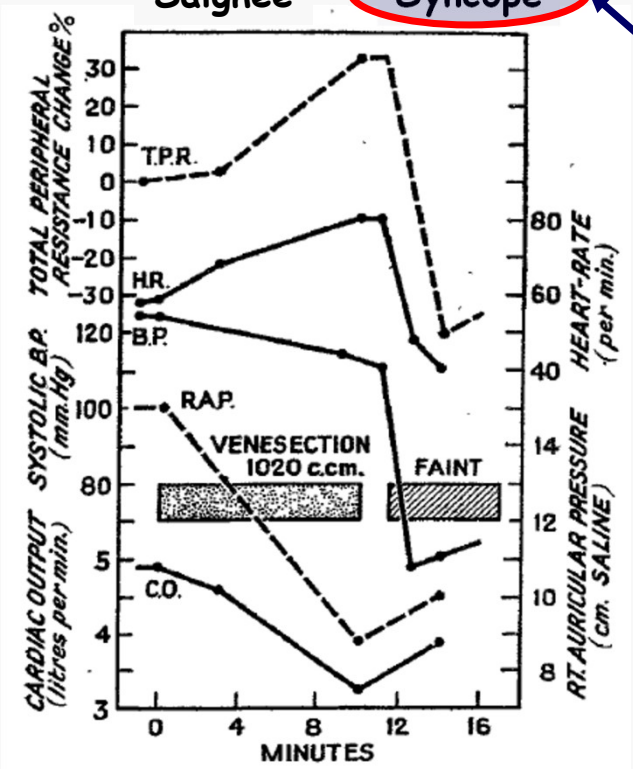


Fig. 1—Faint induced by venesection. Up to the end of the venesection, arterial pressure is maintained by peripheral vasoconstriction (increased total peripheral resistance) in spite of a falling cardiac output. During the faint the cardiac output increases slightly and the fall in blood-pressure is therefore due to decrease in peripheral resistance.

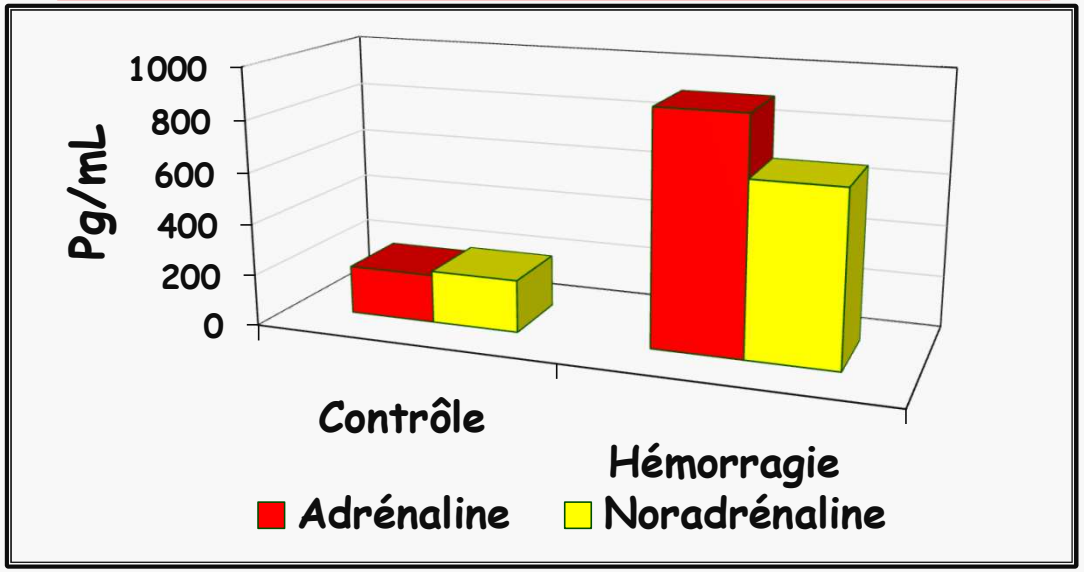
Phase sympatho-inhibitrice

Phase sympatho-excitatrice BaroR artériels, volo-R et chémoR			
Sympathique	Médullo-surrénale	ADH	Angio II
+	0	0	+

D'après Vivien B,Riou B. Physiopathologie du choc hémorragique. Physiologie humaine appliquée. Arnette 2006

Pentobarbital alters compensatory neural and humoral mechanisms in response to hemorrhage

Zimpfer M et al. Am J Physiol 1982; 243: H713-H721



N ENGL J MED 378;4 NEJM.ORG JANUARY 25, 2018

REVIEW ARTICLE

Hemorrhagic Shock

Jeremy W. Cannon, M.D.

Table 2. Classification of Hemorrhagic Shock.*

Shock Class	Blood Loss†	Heart Rate	Blood Pressure	Pulse Pressure	Respiratory Rate	Mental Status
	ml (%)	*beats/min*			*breaths/min*	
I	<750 (15)	<100	Normal	Normal	14–20	Slightly anxious
II	750–1500 (15–30)	100–120	Normal	Narrowed	20–30	Mildly anxious
III	1500–2000 (30–40)	120–140	Decreased	Narrowed	30–40	Anxious, confused
IV	>2000 (>40)	>140	Decreased	Narrowed	>35	Confused, lethargic

* Data are from the American College of Surgeons Committee on Trauma.[42]
† Blood-loss volume and percentage of total blood volume are for a male patient with a body weight of 70 kg.

Choc hémorragique chez l'enfant

Hemorrhagic Shock in Children

G. Orliaguet · E. Vergnaud · C. Duracher · P. Meyer

Méd. Intensive Réa (2016) 25:619-627

Tableau 1 Signes cliniques d'hémorragie en fonction du volume de la perte sanguine chez l'enfant [5]

	Pertes sanguines (% du volume sanguin estimé)		
Signes cliniques	< 20 %	25 %	40 %
Cardiovasculaires	Pouls filant	Pouls filant	Hypotension artérielle
	Tachycardie	Tachycardie	Tachy- voire bradycardie
Cutanés	Peau froide	Extrémités froides	Pâle
	Pouls capillaire 2–3 secondes	Cyanose	Froid
Rénaux	Oligurie modérée	Oligurie nette	Anurie
Neuropsychiques	Irritable, agressif	Confusion, léthargie	Coma

Impact of Norepinephrine and Fluid on Cerebral Oxygenation in Experimental Hemorrhagic Shock

Meybohm P et al. Pediatr Res 2007; 62: 440–444,

Modèle pédiatrique de choc hémorragique chez le porcelet (25 ml/kg)

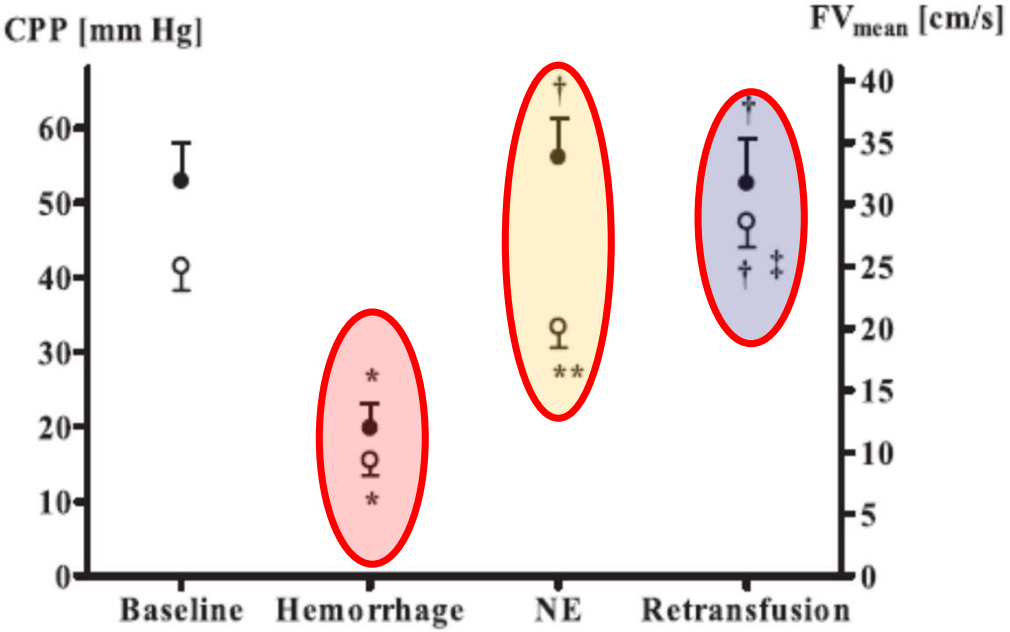


Figure 1. CPP (mm Hg, ●) and FV$_{mean}$ (cm/s, ○) at the different experimental stages ($n = 8$). *$p < 0.001$ *vs* baseline; **$p < 0.01$ *vs* hemorrhage; †$p < 0.001$ *vs* hemorrhage; ‡$p < 0.05$ *vs* NE. NE indicates norepinephrine. The values are given as mean ± SEM.

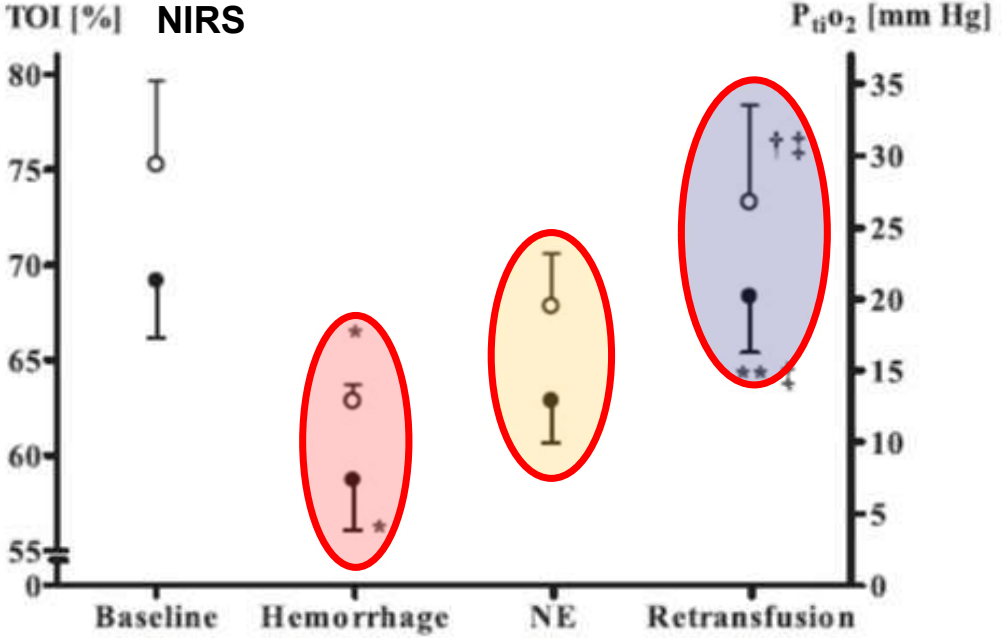


Figure 2. TOI (%, ●) and P$_{ti}$O$_2$ (mm Hg, ○) at the different experimental stages ($n = 8$). *$p < 0.001$ *vs* baseline; **$p < 0.001$ *vs* hemorrhage; †$p < 0.05$ *vs* hemorrhage; ‡$p < 0.05$ *vs* NE. NE indicates norepinephrine. The values are given as mean ± SEM.

Physiopathologie du choc hémorragique chez l'enfant

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Microcirculatory Impairment Is Associated With Multiple Organ Dysfunction Following Traumatic Hemorrhagic Shock: The MICROSHOCK Study

Hutchings SD et al. ***Crit Care Med 2018; 46:e889–e896***

D0 <12h après admission ICU
D1= D0 +24h
D2= D0 +48h

Impact des altérations de micro- et macro-circulation sur la défaillance multiviscérale à J7 (MODS D7) après choc hémorragique traumatique

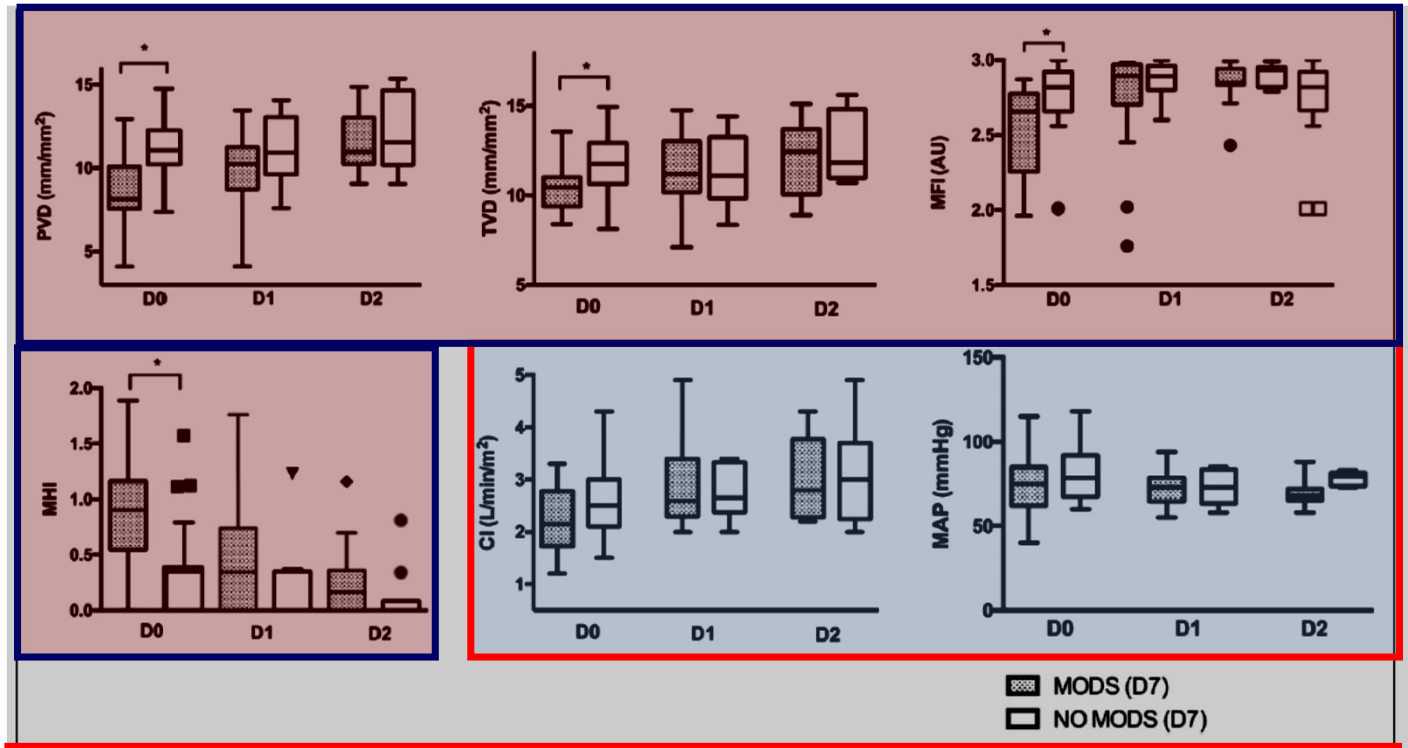


Figure 1. Microcirculatory and macrocirculatory variables at all study time points according to multiple organ dysfunction syndrome (MODS) at day 7. Patients are dichotomized into those who did or did not develop MODS by day 7 after injury. $^{*}p < 0.05$ between groups (*t* test/Mann-Whitney *U*). NB comparative analysis only performed at following bleeding control procedures, but < 12 hours after admission to the ICU (D0) time point due to patient drop out at D0 + 24 hours (D1) and D0 + 48 hours (D2). AU = arbitrary units, CI = cardiac index, MAP = mean arterial pressure, MFI = microcirculatory flow index, MHI = microcirculatory heterogeneity index, PVD = perfused vessel density, TVD = total vessel density.

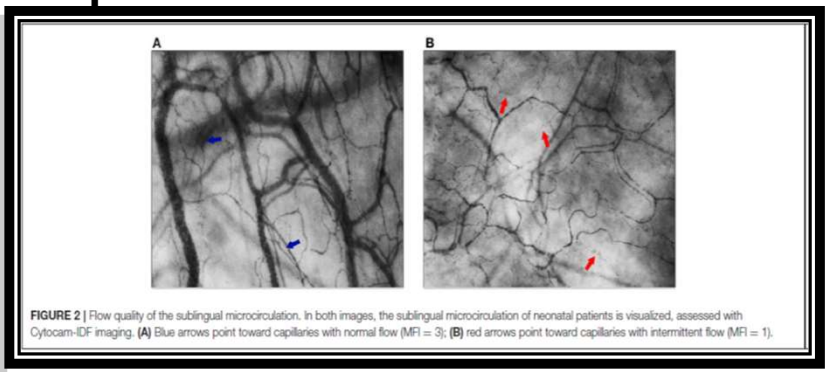


FIGURE 2 | Flow quality of the sublingual microcirculation. In both images, the sublingual microcirculation of neonatal patients is visualized, assessed with Cytocam-IDF imaging. **(A)** Blue arrows point toward capillaries with normal flow (MFI = 3); **(B)** red arrows point toward capillaries with intermittent flow (MFI = 1).

Microcirculation

⇔ **Microscopie sublinguale**

- **MFI = microcirculatory flow index**
- **MHI = microcirculation heterogeneity index**
- **PVD = perfused vessel density**
- **TVD = total vessel density**

Assessing the Microcirculation With Handheld Vital Microscopy in Critically Ill Neonates and Children: Evolution of the Technique and Its Potential for Critical Care

Erdem O et al. Front Pediatr 2019; 7:273.

TABLE 2 | Summary of findings: microcirculatory studies performed in pediatric patients.

Reference	HVM	Study population	n	Area of interest	Findings
Top (56)	OPS	Septic shock	1	Buccal mucosa	Sepsis therapy recovered macrocirculation, while microcirculation was still compromised
Top et al. (8)	OPS	Septic shock: survivors vs. non-survivors	15/3	Buccal mucosa	FVD improved on day 2 in survivors, while FVD was lower and did not change in non-survivors; persistent microcirculatory disturbances were prognostic for mortality
Top et al. (57)	OPS	Hypoxemic respiratory failure: iNO therapy	8	Buccal mucosa	iNO therapy improved FVD
Paize et al. (58)	SDF	Meningococcal disease vs. controls	20/40	Sublingual mucosa	Microcirculatory parameters were lower in meningococcal disease than in controls but recovered when patients clinically recovered
Buijs (59)	SDF	Respiratory failure: VA ECMO vs. VV ECMO	31/17	Buccal mucosa	Groups did not differ; PPV and MFI were impaired prior to start ECMO, improved one day after start ECMO and recovered in both groups
Buijs et al. (9)	SDF	Post cardiac arrest patients during TH vs. controls	22/20	Buccal mucosa	All microcirculatory parameters were impaired during TH; severe impairment at start TH was associated with mortality; microcirculatory parameters improved rapidly after TH
Nussbaum et al. (60)	SDF	Diabetes patients vs. controls	14/14	Sublingual mucosa	Glycocalyx thickness was reduced in diabetes patients compared to controls and inversely correlated with blood glucose levels; diabetes patients showed more large vessels than small vessels than controls
Nussbaum et al. (61)	SDF	Cardiac surgery vs. cardiac catheterization vs. non-cardiac surgery controls	40/6/9	Cutaneous (fossa triangularis ear)	Glycocalyx thickness was reduced after cardiac surgery and returned to baseline values after 1 week; MFI and PVD also declined and returned to baseline values after 24 h
Schinagl et al. (62)	SDF	Anemic children receiving RBC transfusion vs. controls	19/18	Buccal mucosa	Anemic children showed lower TVD lower and higher RBC velocity than controls; after transfusion, Hb and TVD increased and RBC velocity decreased; TVD and RBC velocity did not reach levels of controls
Scolletta et al. (63)	SDF	Cardiac surgery: cyanotic vs. a-cyanotic heart defects	7/17	Sublingual mucosa	Microcirculatory parameters did not change over time and were not correlated to macrocirculation in both groups; cyanotic children showed different time trends for PPV and TVD than a-cyanotic children
Gonzalez et al. (37)	IDF	Admission pediatric ICU	105	Sublingual mucosa	Microcirculatory assessment only possible in 17%, mostly intubated and the more severely ill patients; microcirculatory parameters were moderately correlated with BP, CVP, and lactate
Riedijk and Milstein (64)	IDF	Procedural sedation with propofol	7	Sublingual mucosa	Propofol induction induced a decline of BP and an increase of TVD and PVD

Physiopathologie du choc hémorragique chez l'enfant

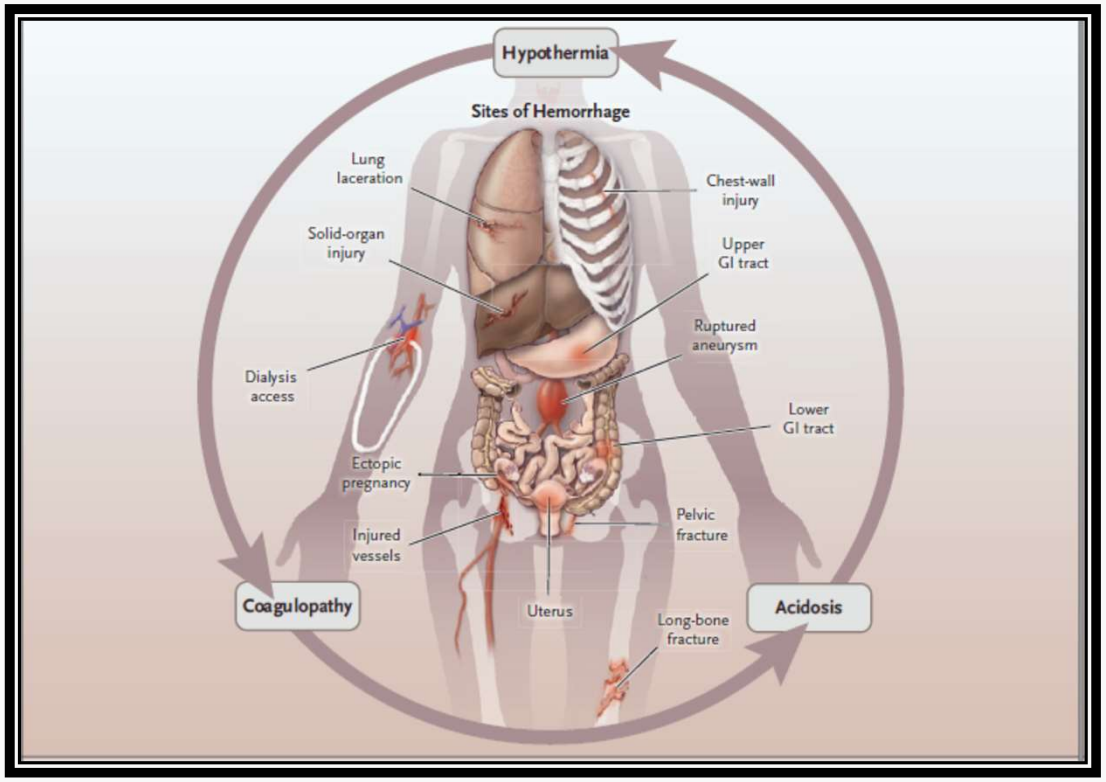
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The NEW ENGLAND JOURNAL of MEDICINE
N ENGL J MED 378;4 NEJM.ORG JANUARY 25, 2018

REVIEW ARTICLE

Hemorrhagic Shock

Jeremy W. Cannon, M.D.



The lethal triad: A bloody vicious cycle: once you get in, it's hard to get out"

Physiopathologie et prise en charge de la coagulopathie de l'hémorragie massive

Sophie Susen[1]
Charles-Marc Samama[2]
Anne Godier[2]

Hématologie 2012; 18 (6) : 325-37

Facteurs favorisant la coagulopathie
Pertes de facteurs et de plaquettes Par dilution Par consommation des facteurs de la coagulation et des plaquettes
Hypothermie
Acidose
Hypocalcémie
Hématocrite bas (rhéologie)
Fibrinolyse induite par des lésions traumatiques spécifiques
Effet propre des solutés colloïdes
Traitement antithrombotique

Pediatric Anesthesia

Pediatric Anesthesia 2010 20: 150–159 doi:10.1111/j.1460-9592.2009.03227.x

Blood loss, replacement, and associated morbidity in infants and children undergoing craniofacial surgery

Stricker PA et al. Pediatric Anesthesia 2010; 20:150-9

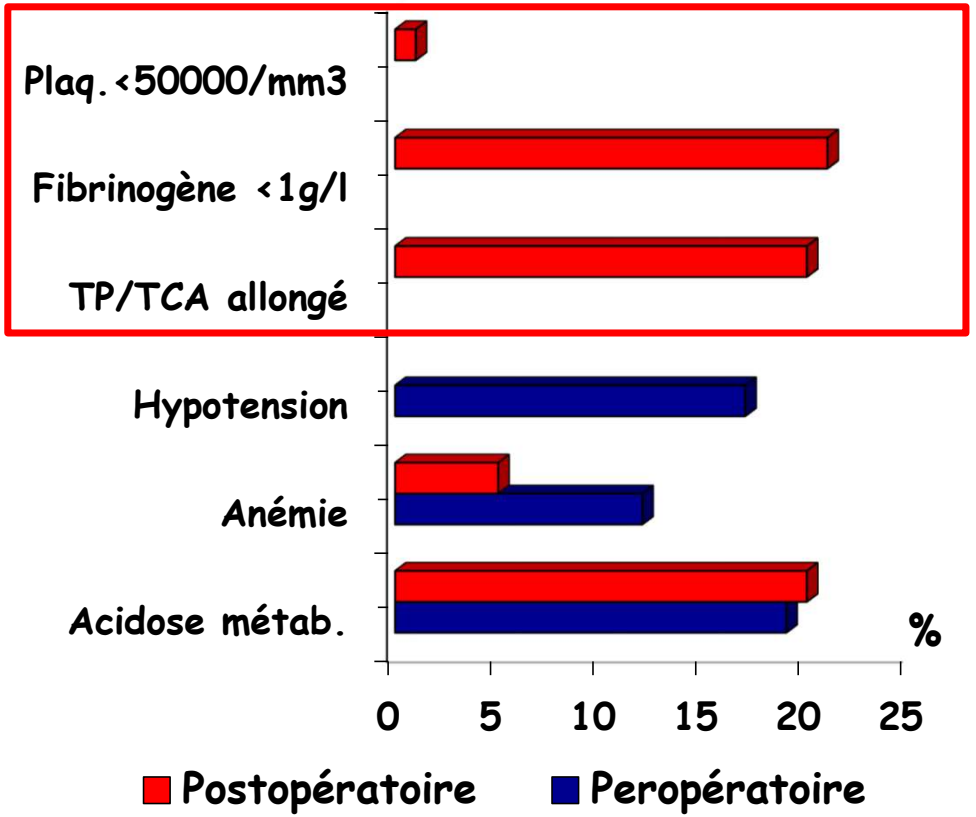
Chirurgie de craniosténose









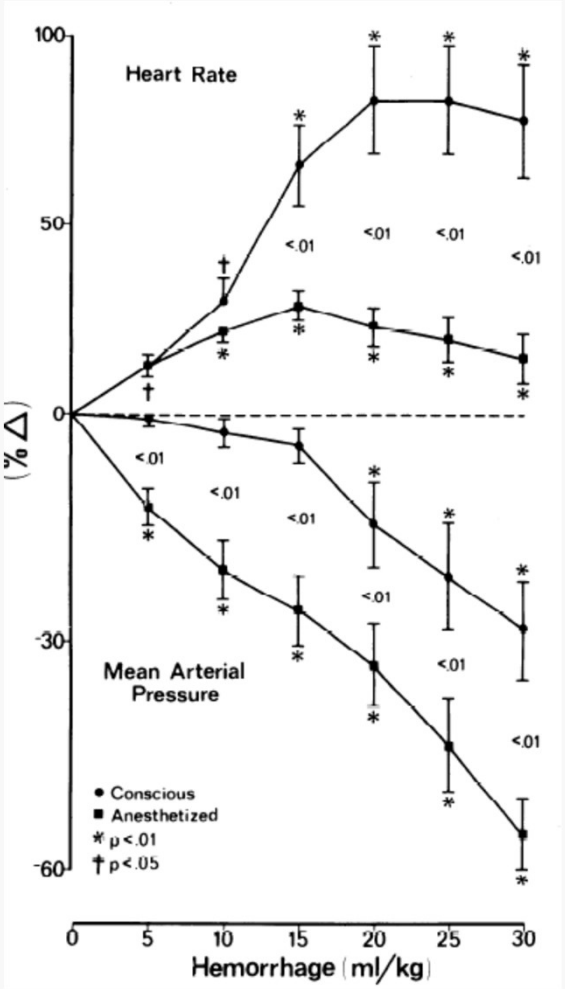


Physiopathologie du choc hémorragique chez l'enfant

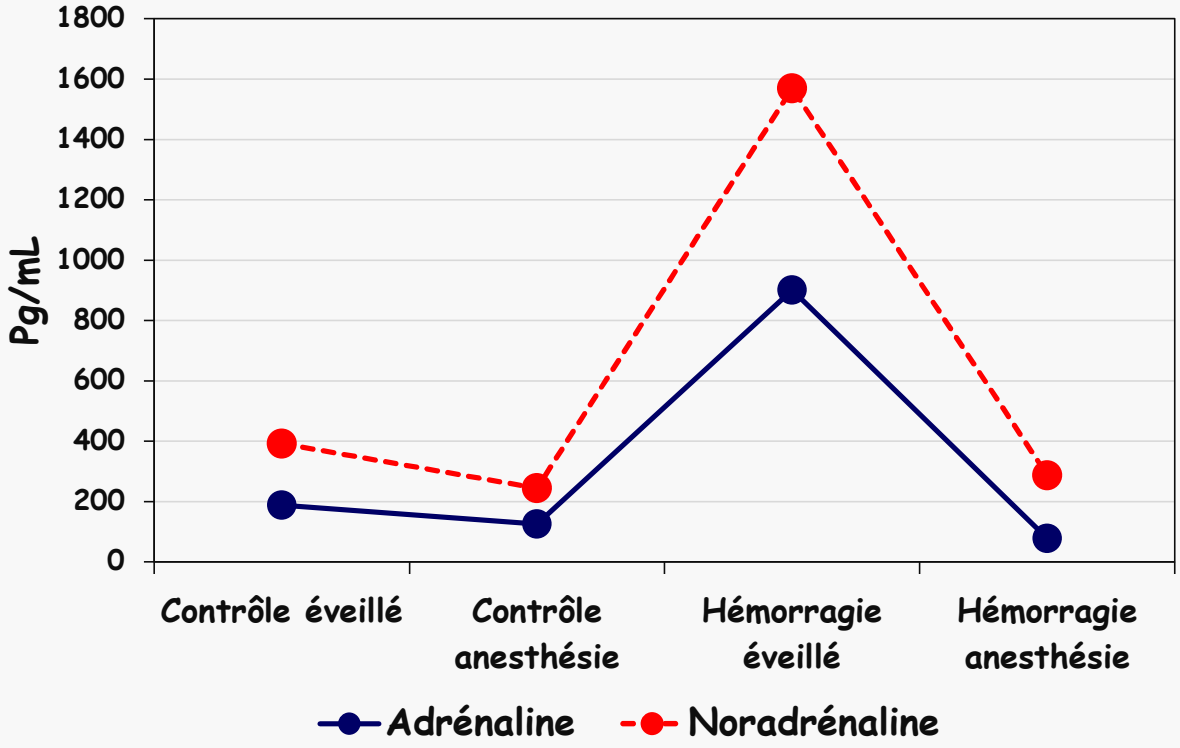
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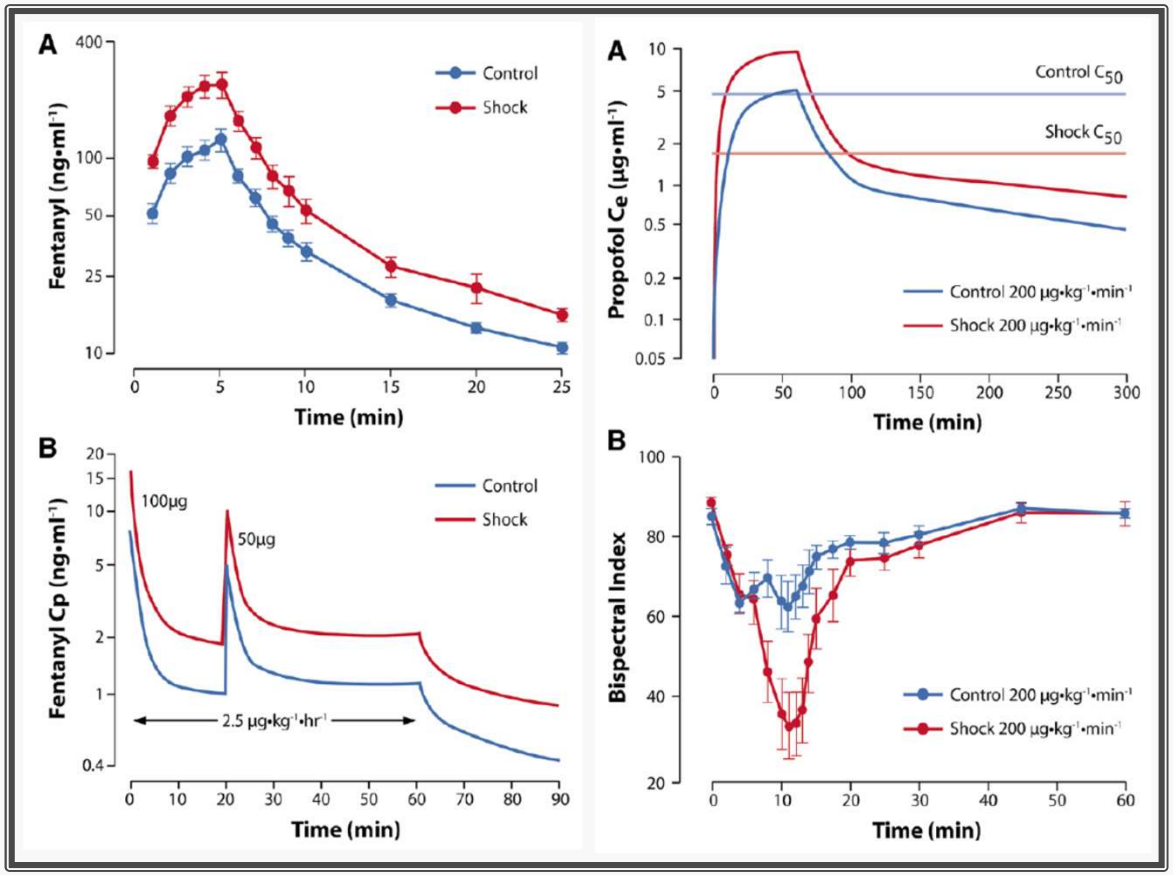
Effets de l'anesthésie sur la réponse neurohormonale à l'hémorragie

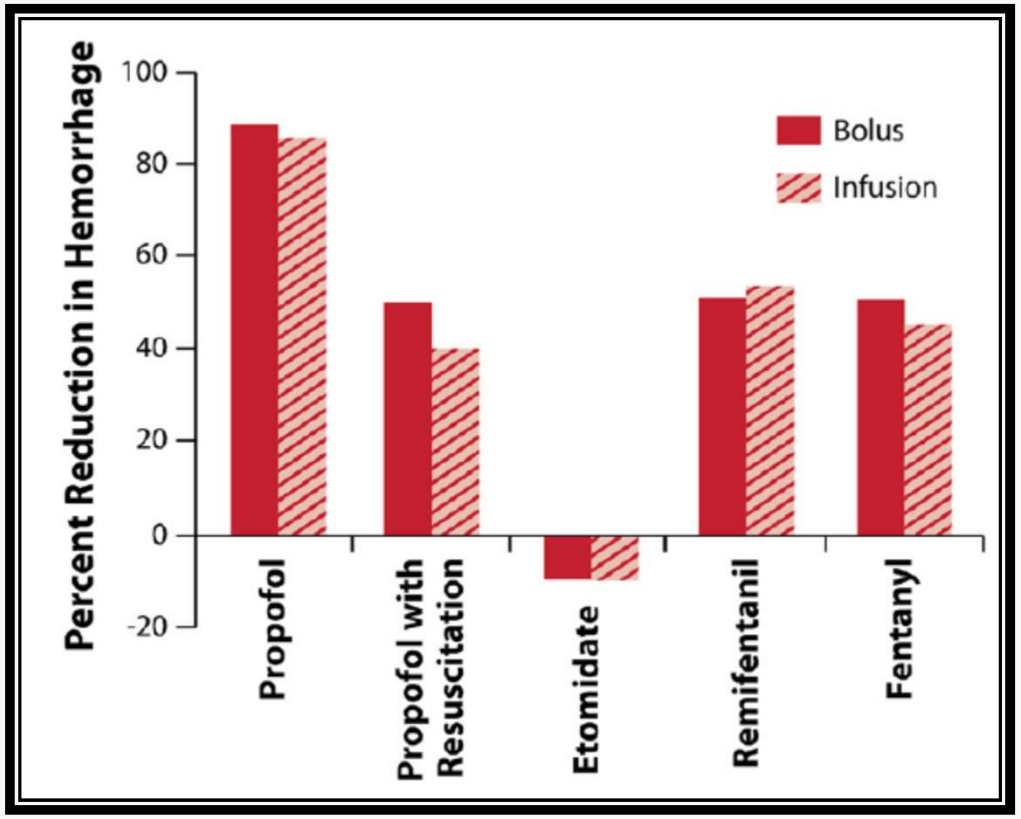


The Influence of Hemorrhagic Shock on the Disposition and Effects of Intravenous Anesthetics: A Narrative Review

Anesth Analg 2020;130:1320–30

Ezekiel D. Egan, BA, and Ken B. Johnson, MD





Physiopathologie du choc hémorragique chez l'enfant : conclusions

- Une physiopathologie complexe ≠ d'une simple perte de sang
- Des phénomènes en cascade qui s'auto-entretiennent impliquant la macro-, la micro-circulation, l'hémostase et la pharmacologie
- Des différences notables entre enfants et adultes
- Une interaction entre mécanismes compensateurs et anesthésie
- Une précocité de la prise en charge pour améliorer le pronostic

  ⇔ Damage Control Resuscitation

Enquête SFAR ADARPEF

