CONTRÔLE GLYCÉMIQUE PERIOPERATOIRE EN CHIRURGIE CARDIAQUE

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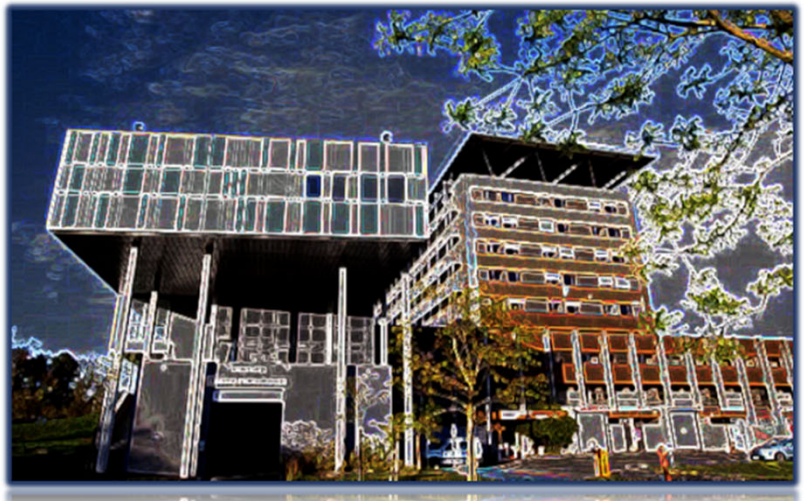
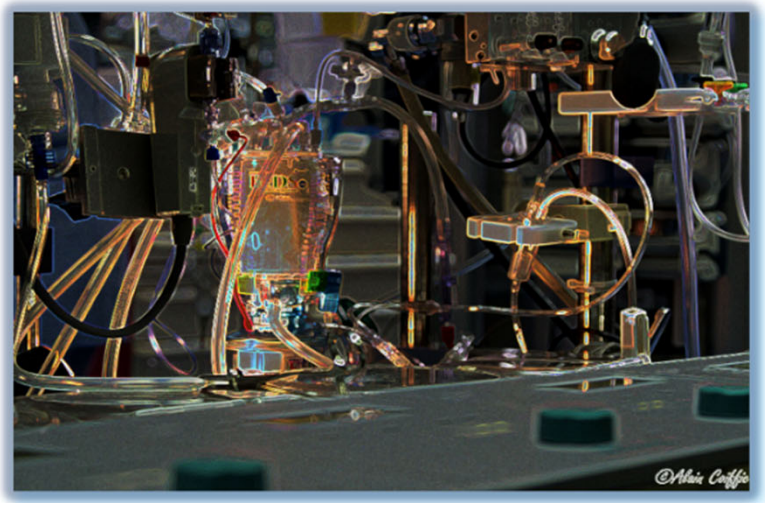
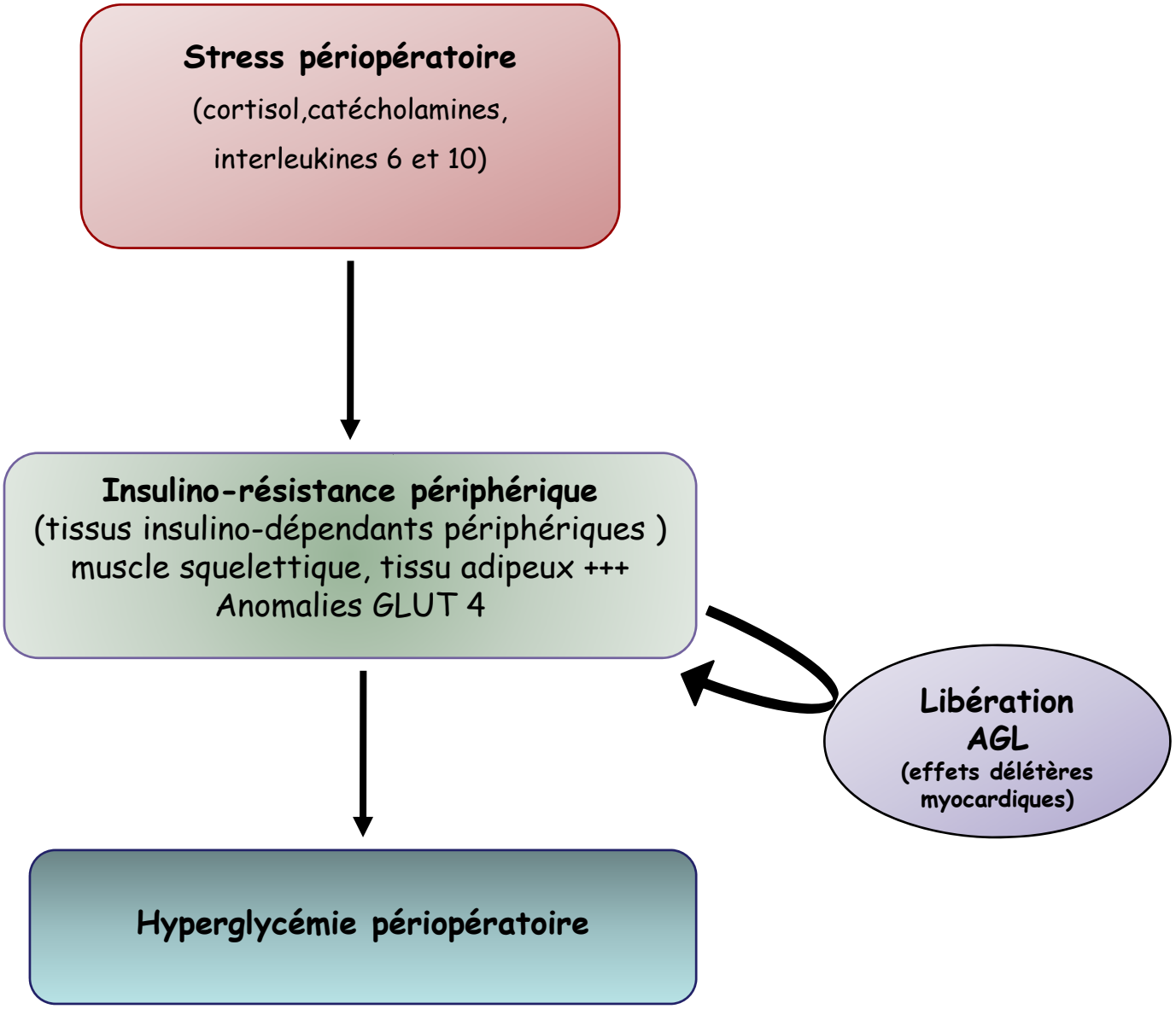






Ljungqvist et al. Surgery 2000;28:757-60

Lipshutz et al. Anesthesiology 2009;110:408-21

Précoce

- *Quelques heures après début de la chirurgie*
- *Peropératoire, postopératoire immédiat,…*

Persiste plusieurs jours après chirurgie (2-3 semaines)

Insulino-résistance périphérique
(tissus insulino-dépendants périphériques)
muscle squelettique, tissu adipeux

Facteurs aggravants

- *Obésité (tissu adipeux +++)*
- *Chirurgie invasive (hémorragique, longue durée, hémodynamiquement intense, circulation extra-corporelle, …)*
- *Jeun préopératoire prolongé (en limiter le temps ou apport glucosé…)*
- *Hypothermie*
- *Immobilisation prolongée (réhabilitation précoce)*

Insulin resistance after abdominal surgery

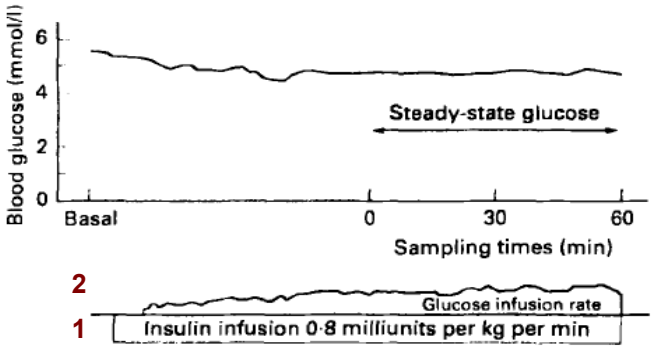
British Journal of Surgery 1994, 81, 59–63

A. THORELL, S. EFENDIC*, M. GUTNIAK*, T. HÄGGMARK and O. LJUNGQVIST

Détermination et évolution insulino-sensibilité

Cholécystectomie réglée

Clamp euglycémique hyperinsulinique



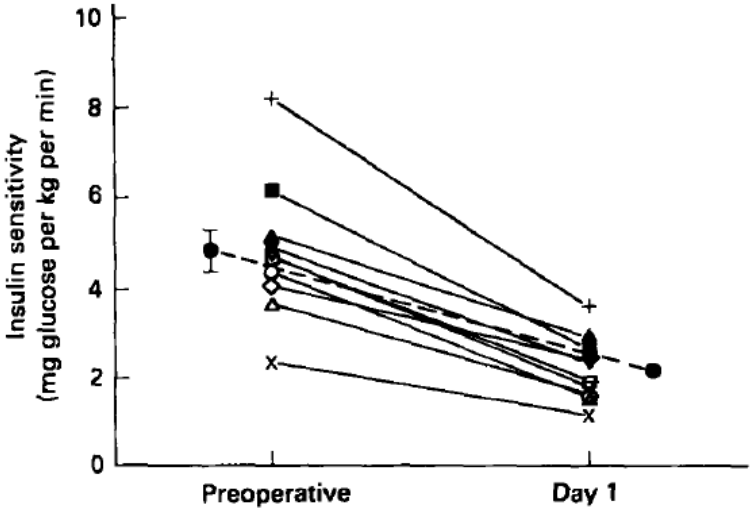


Fig. 2 Insulin sensitivity (M value) for all participating subjects before and on the first day after open cholecystectomy ($n = 10$). ---, Mean(s.e.m.)

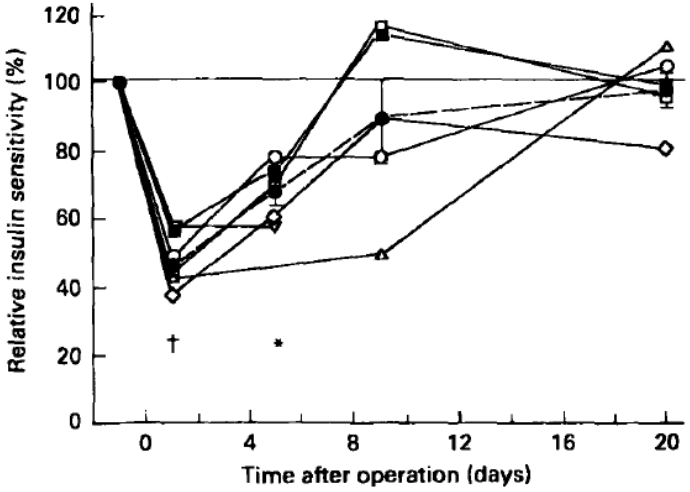
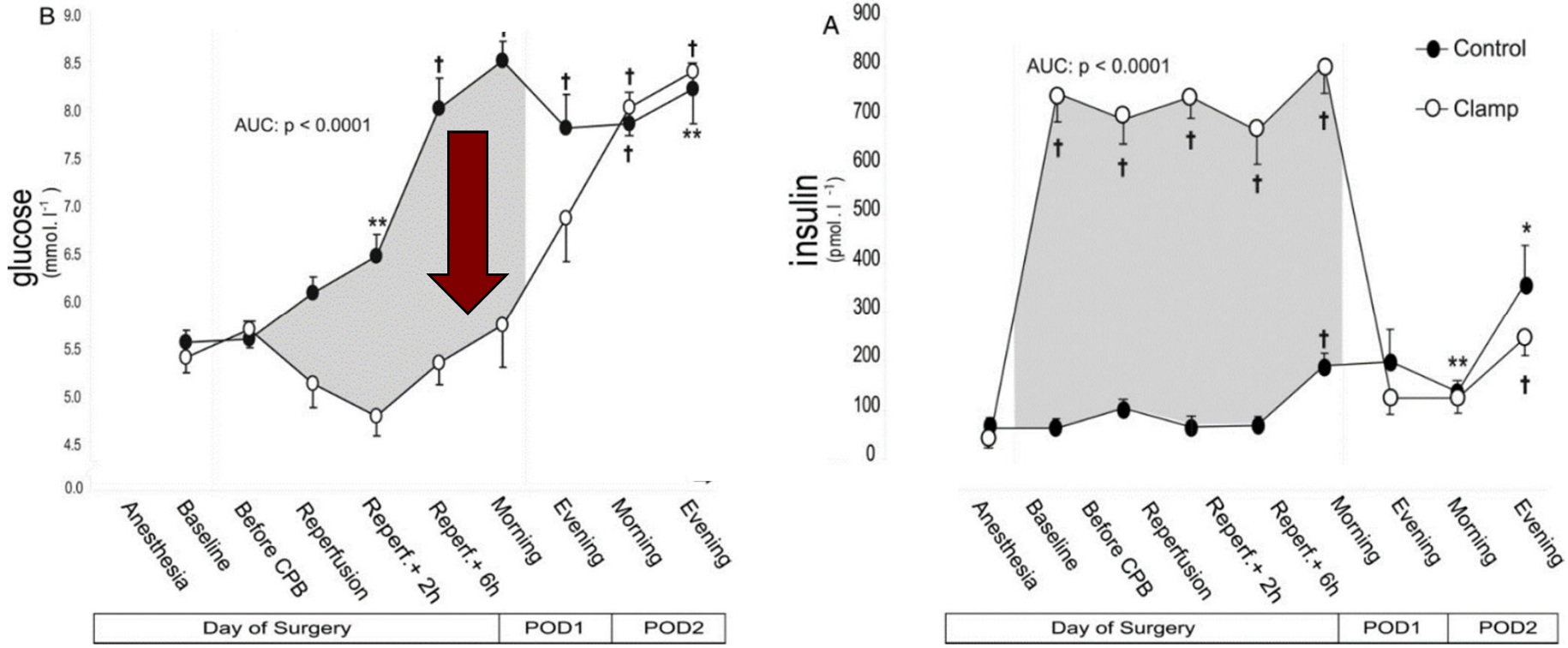


Fig. 3 Relative insulin sensitivity (calculated as postoperative M value/preoperative M value) before and on days 1, 5, 9 and 20 after open cholecystectomy. ---, Mean(s.e.m.) ($n = 5$ or 10). *$P < 0{\cdot}01$, †$P < 0{\cdot}001$ (*versus* preoperative values, Student's *t* test)

Insulino-résistance «relative»

Corrigée par un apport exogène insulinique

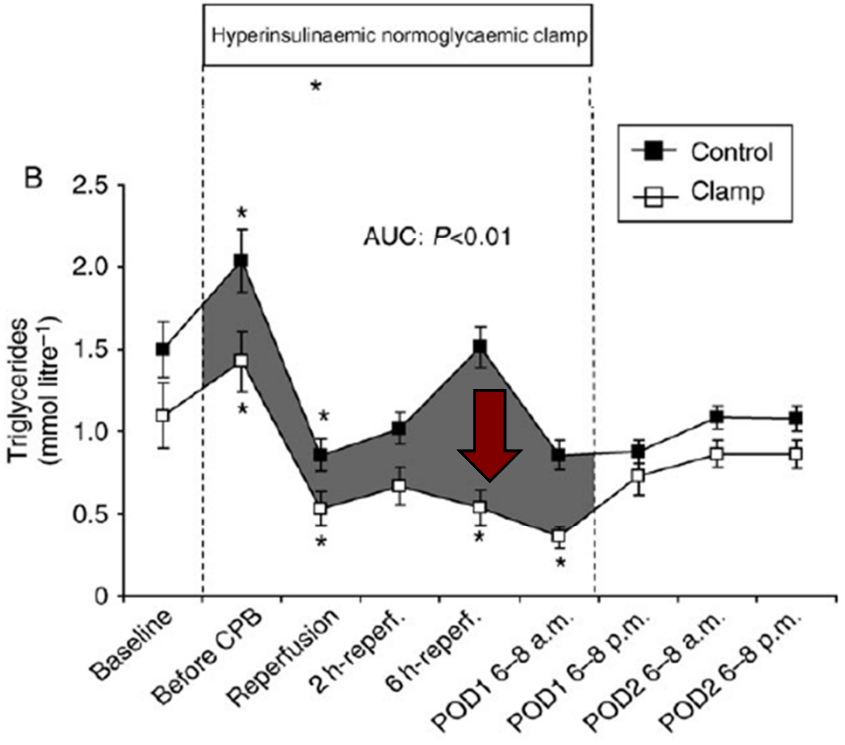


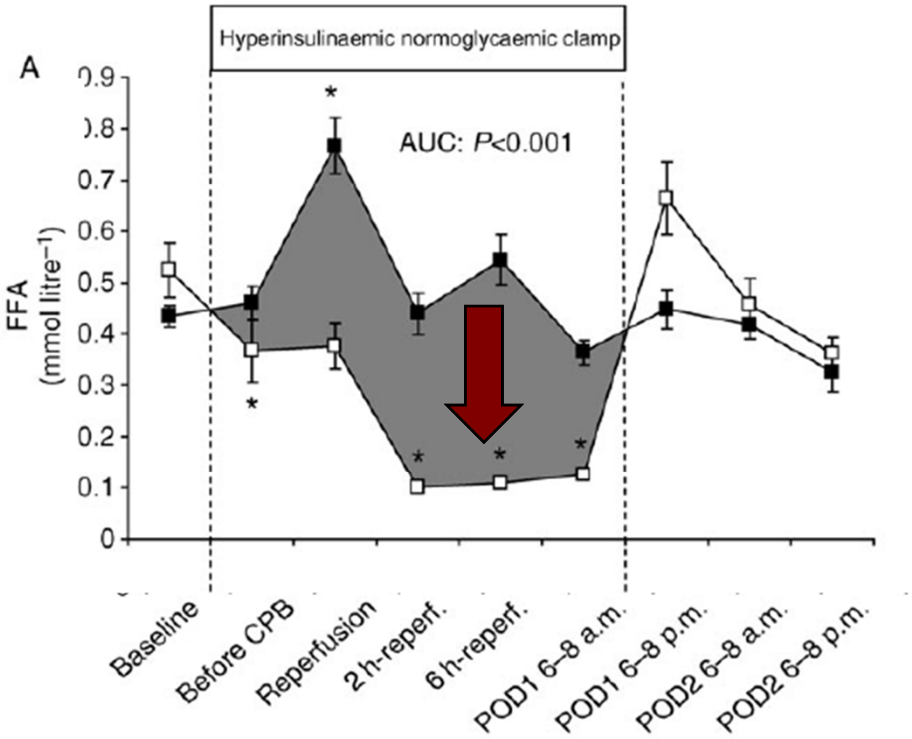
Van Wezel HB et al. J Clin Endocrinol Metab 2006;91:4144-53

Perioperative hyperinsulinaemic normoglycaemic clamp causes hypolipidaemia after coronary artery surgery†

C. J. Zuurbier[1]*, F. J. Hoek[5], J. van Dijk[1], N. G. Abeling[6], J. C. M. Meijers[4], J. H. M. Levels[4], E. de Jonge[3], B. A. de Mol[2] and H. B. Van Wezel[1]

Br J Anaesth 2008; **100**: 442–50





Hyperglycémie périopératoire

Les mécanismes…

- ✓ Insulino-résistance +++

Tsubo T et al. Can J Anaesth 1990; 37:645-9

- ✓ Stimulation production endogène de glucose

Schricker T et al. Nutrition 1997; 13:191-5

- ✓ Augmentation résorption rénale de glucose

Braden H et al. Ann Thorac Surg 1998;65:1588-93

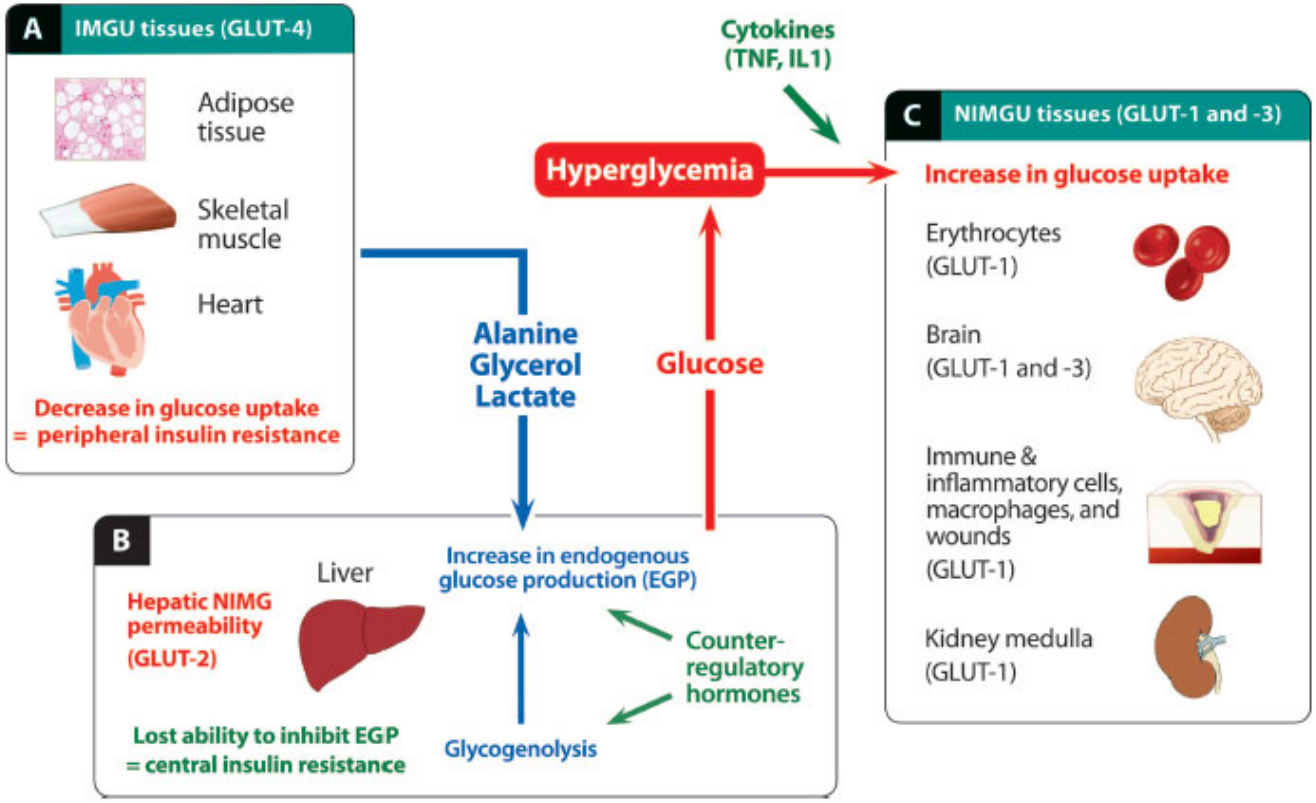
- ✓ Diminution clairance du glucose

Sicardi Salomon Z et al. Acta Anaesthesiol Scand 2006;50:848-54

Glycemic Control in the Intensive Care Unit and during the Postoperative Period

Diane Lena, M.D.,* Pierre Kalfon, M.D.,† Jean-Charles Preiser, M.D., Ph.D.,‡
Carole Ichai, M.D., Ph.D.§

Anesthesiology 2011; 114:438–44



Acute hyperglycemia abolishes ischemic preconditioning in vivo

Am J Physiol 1998; 275:H721-25

JUDY R. KERSTEN, TODD J. SCHMELING, KARL G. ORTH,
PAUL S. PAGEL, AND DAVID C. WARLTIER
Departments of Anesthesiology and Pharmacology and Toxicology and Division of Cardiovascular Diseases, Department of Medicine, Medical College of Wisconsin, Milwaukee, Wisconsin 53226

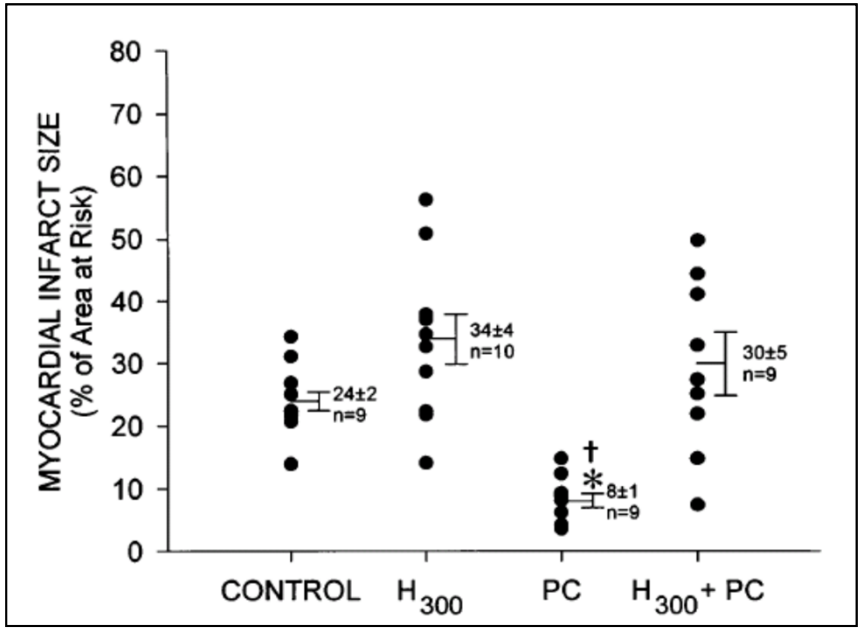
Etude expérimentale (cœurs de chien)

Ischémie (60 min) + Reperfusion (3h)

Pré-conditionnement ischémique = 4 X 5 min occlusion avant IR

Contrôle et Hyperglycémique (≈ 300 mg/dL) induite par perf IV

Critère de jugement = Taille de l'infarctus myocardique par rapport à la taille de la zone à risque



Reactive oxygen species modulate coronary wall shear stress and endothelial function during hyperglycemia

Stress oxydatif

Eric R. Gross,[1,2,3*] John F. LaDisa, Jr.,[1,3*] Dorothee Weihrauch,[1]
Lars E. Olson,[3] Tobias T. Kress,[1] Douglas A. Hettrick,[1,3]
Paul S. Pagel,[1,3] David C. Warltier,[1,2,3,4] and Judy R. Kersten[1,2]

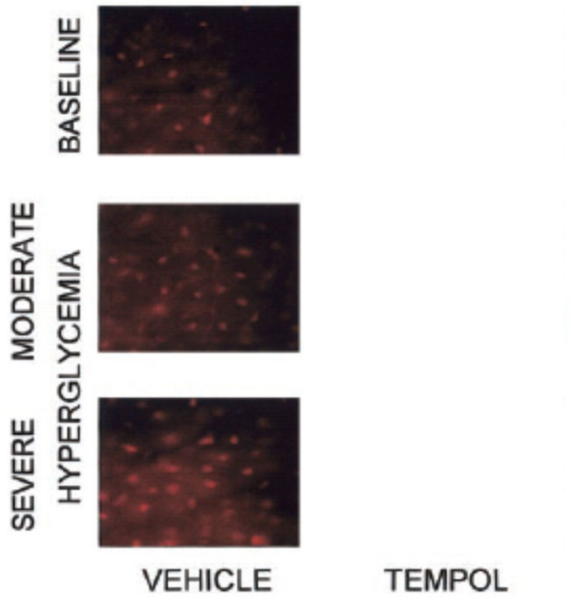
Am J Physiol Heart Circ Physiol 284: H1552–H1559, 2003.

Modèle ***in vivo*** canin

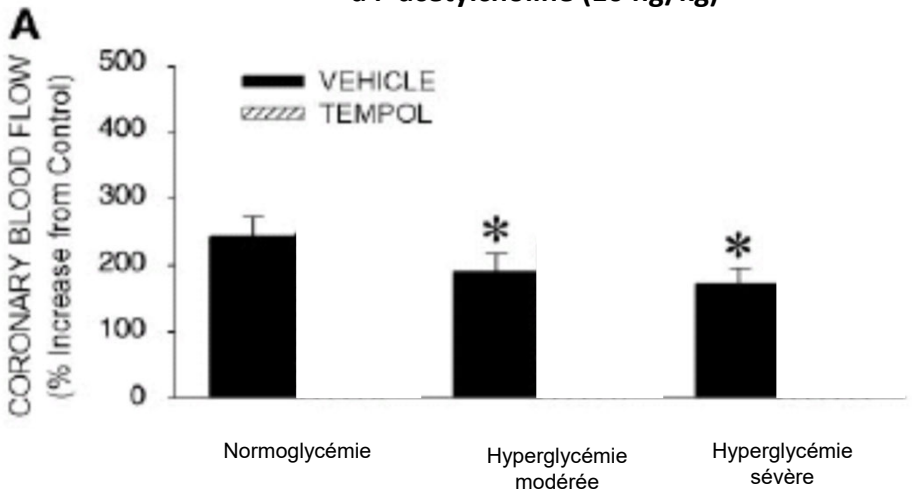
Effets hyperglycémie 350 et 600 mg/dL (Glucose IV) sur production de RL et fonction endothéliale

Antagonisation par le tempol = analogue de la SOD (scavenger des radicaux libres)

Détection par fluorescence des RL dans les biopsies myocardiques



Réponse vasodilatatrice coronaire à l'acétylcholine (10 ng/kg)



Tight Glycemic Control in Diabetic Coronary Artery Bypass Graft Patients Improves Perioperative Outcomes and Decreases Recurrent Ischemic Events

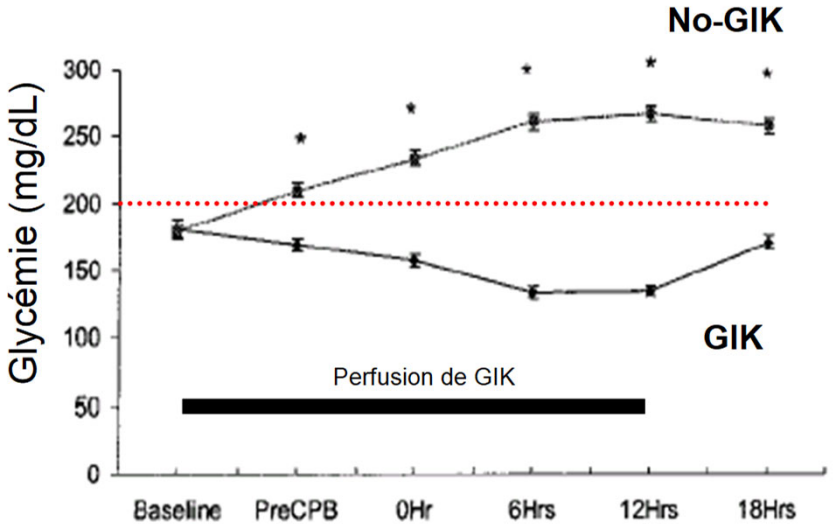
Harold L. Lazar, MD; Stuart R. Chipkin, MD; Carmel A. Fitzgerald, RN; Yusheng Bao, MD; Howard Cabral, PhD; Carl S. Apstein, MD

Circulation. 2004;109:1497-1502.

Etude prospective randomisée

Patients diabétiques en chirurgie coronaire

- Groupe HGT 125-200 mg/dL (n=72)
- Groupe HGT <250 mg/dL (n=69)



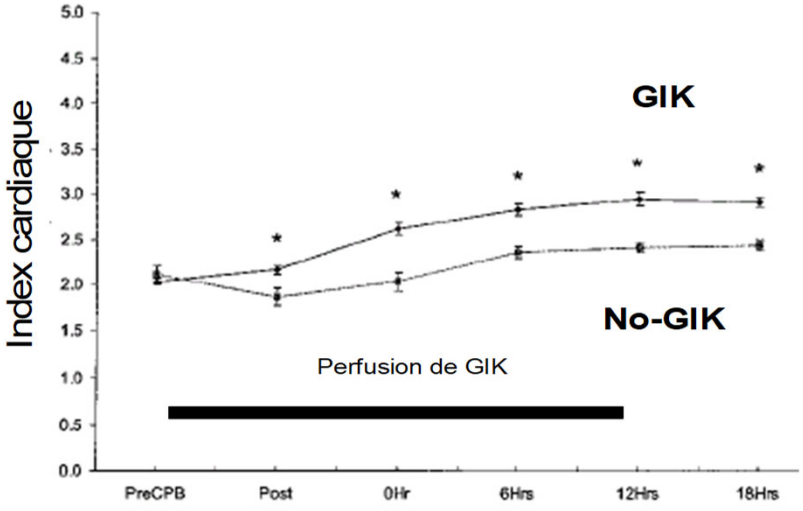


***Tableau**: Morbi-mortalité postopératoire*

	GIK (n=69)	No-GIK (n=72)
IDM postopératoire	0%	2,8%
ACFA	**17%**	42%*
Infections (pneumopathie ou cicatrice)	**0%**	13%*
Durée de ventilation (h)	**6,9 ± 0,3**	10,7 ± 0,6*
Score inotrope	**1,2 ± 0,1**	2,2 ± 0,2*
Durée de séjour en réa (h)	**17,3 ± 1,0**	32,8 ± 2,6*
Durée de séjour totale (j)	**6,5 ± 0,1**	9,2 ± 0,3*
Mortalité à 30 jours	0%	0%

Les résultats sont exprimés en % ou moyenne ± écart-type. P<0,05 versus groupe GIK

Moderate hyperglycemia worsens acute blood-brain barrier injury after forebrain ischemia in rats

Dietrich WD et al. Stroke 1993; 24: 111-116

Etude expérimentale *in vivo* chez **rats**

Effets hyperglycémie sur les lésions d'ischémie cérébrale en normothermie

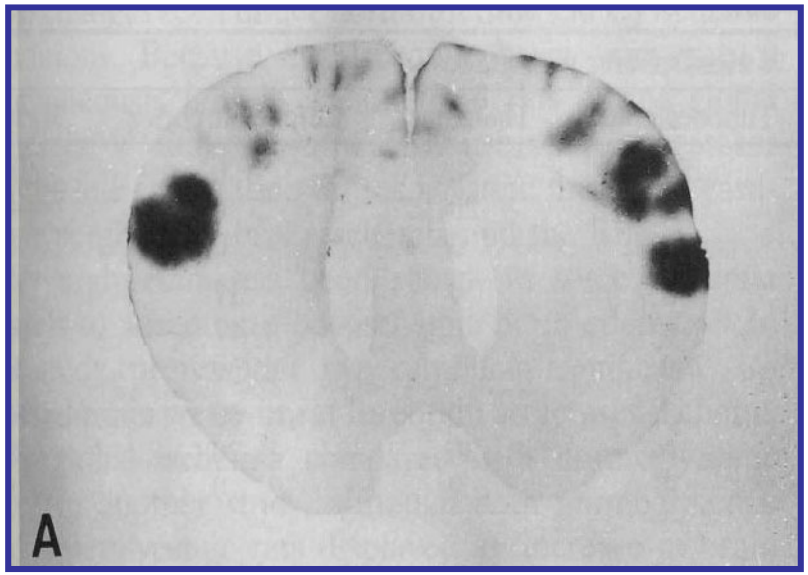
Ischémie globale **20 min** (occlusion 4 axes vasculaires)

Reperfusion **30 min** puis sacrifice des animaux

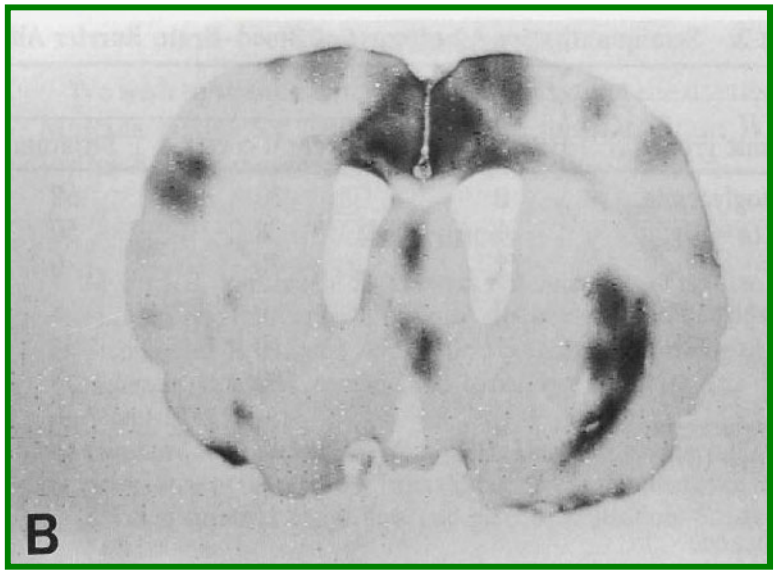
2 groupes - **Hyperglycémique (n=6)** Inj IP G50%, 15 min avant ischémie

- **Normoglycémique** (n=5) Inj IP solution saline avant ischémie

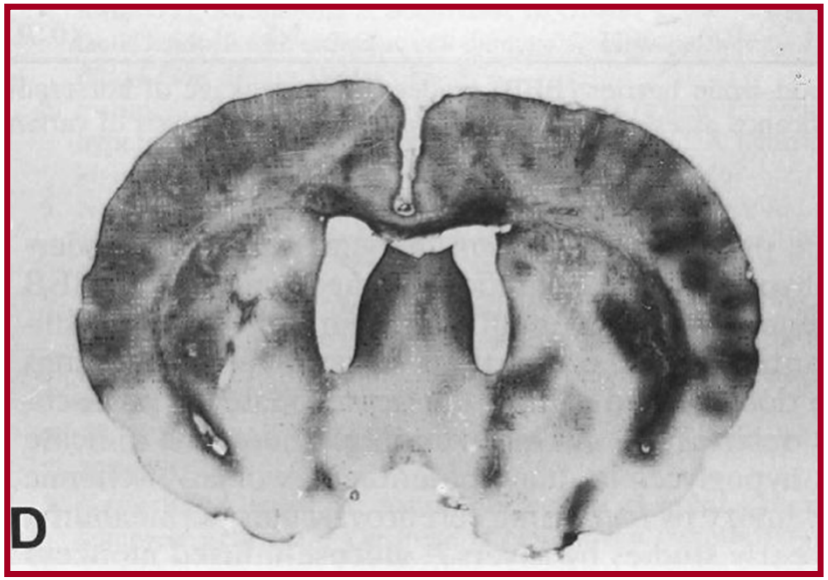
Quantification des lésions barrière hémato-encéphalique (BHE) par de l'extravasation du horseradish peroxidase (HRP) sur coupes histologiques



Normoglycémie (107 mg/dL)



Hyperglycémie (253 mg/dL)



Hyperglycémie (322 mg/dL)

Persistent Poststroke Hyperglycemia Is Independently Associated With Infarct Expansion and Worse Clinical Outcome

Tracey A. Baird, Mark W. Parsons, Thanh Phanh, Ken S. Butcher, Patricia M. Desmond, Brian M. Tress, Peter G. Colman, Brian R. Chambers and Stephen M. Davis

Stroke. 2003;34:2208-2214

TABLE 2. Correlations Between Glycemic Parameters and Outcome Measures

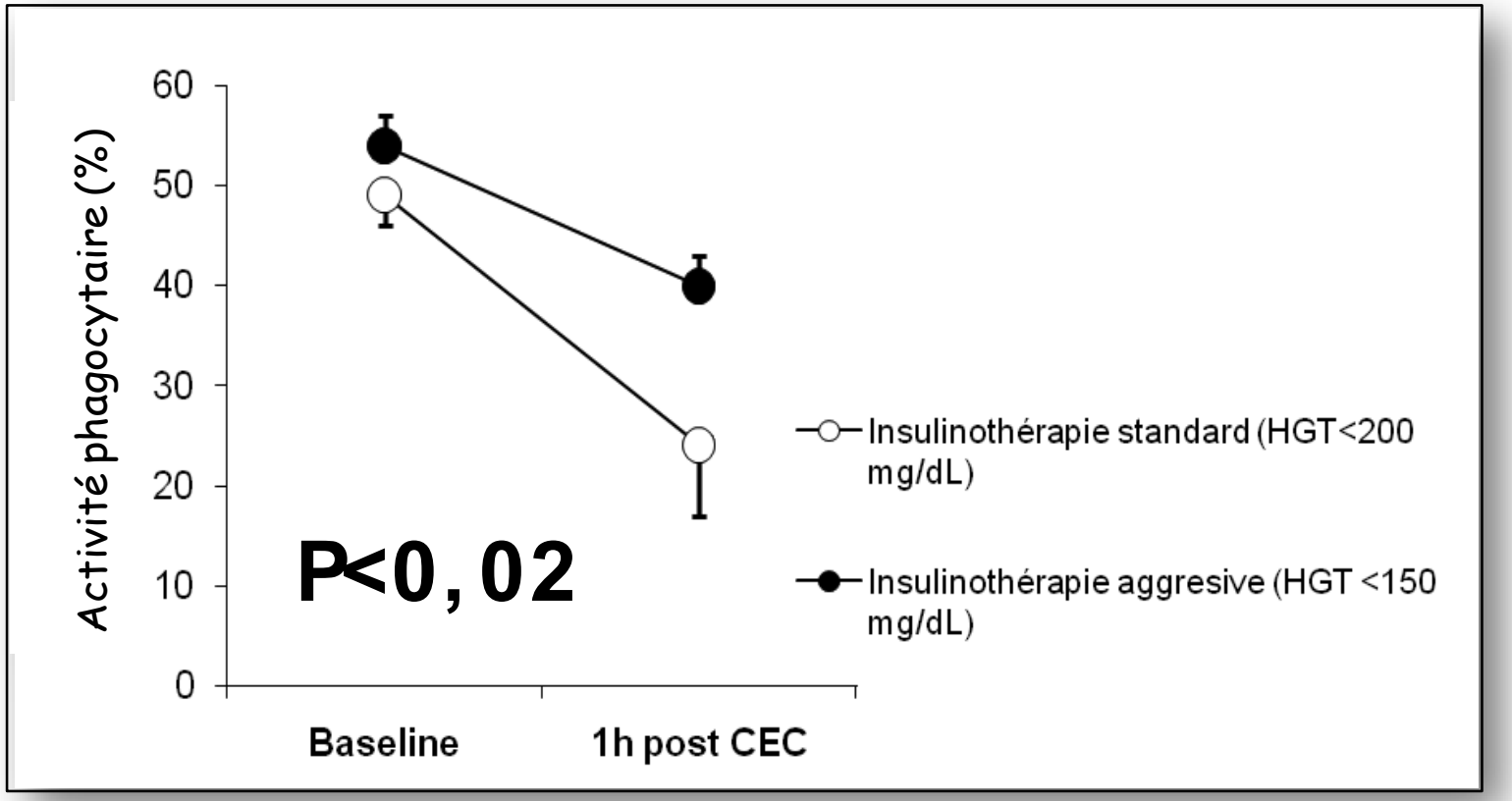
	r

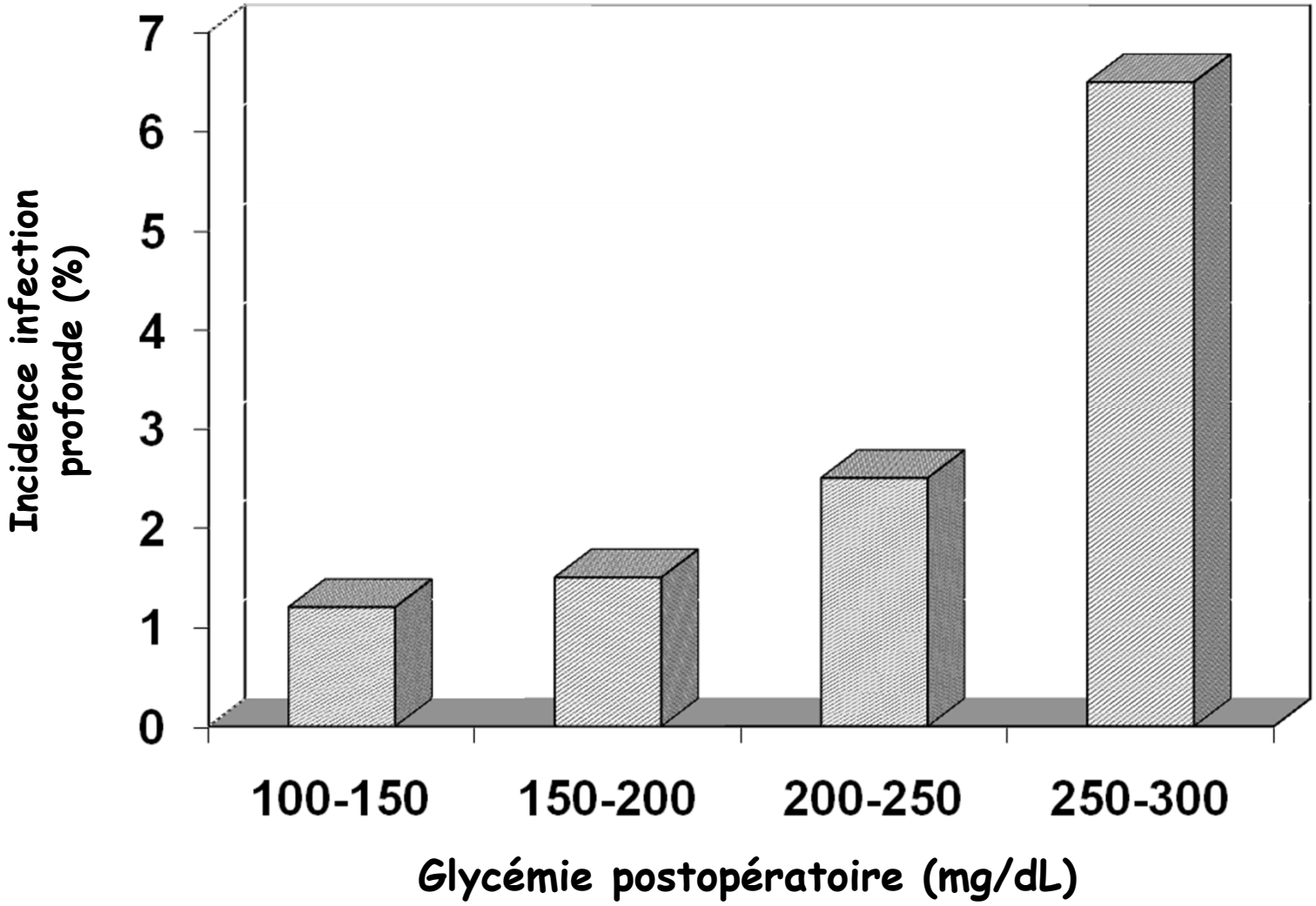
*$P<0.05$.

**mRS= modified Rankin Score*

Insulin Infusion Improves Neutrophil Function in Diabetic Cardiac Surgery Patients

Rassias AJ et al. Anesth Analg 1999





Zerr et al. Ann Thorac Surg 1997

Continuous insulin infusion reduces mortality in patients with diabetes undergoing coronary artery bypass grafting

Anthony P. Furnary, MD,[a,d] Guangqiang Gao, MD,[a] Gary L. Grunkemeier, PhD,[b] YingXing Wu, MD,[b] Kathryn J. Zerr, MBA,[b] Stephen O. Bookin, MD,[c] H. Storm Floten, MD,[a,d] and Albert Starr, MD[a,d]

J Thorac Cardiovasc Surg 2003

Etude prospective historique sur 15 ans

Patients diabétiques (n=3554)

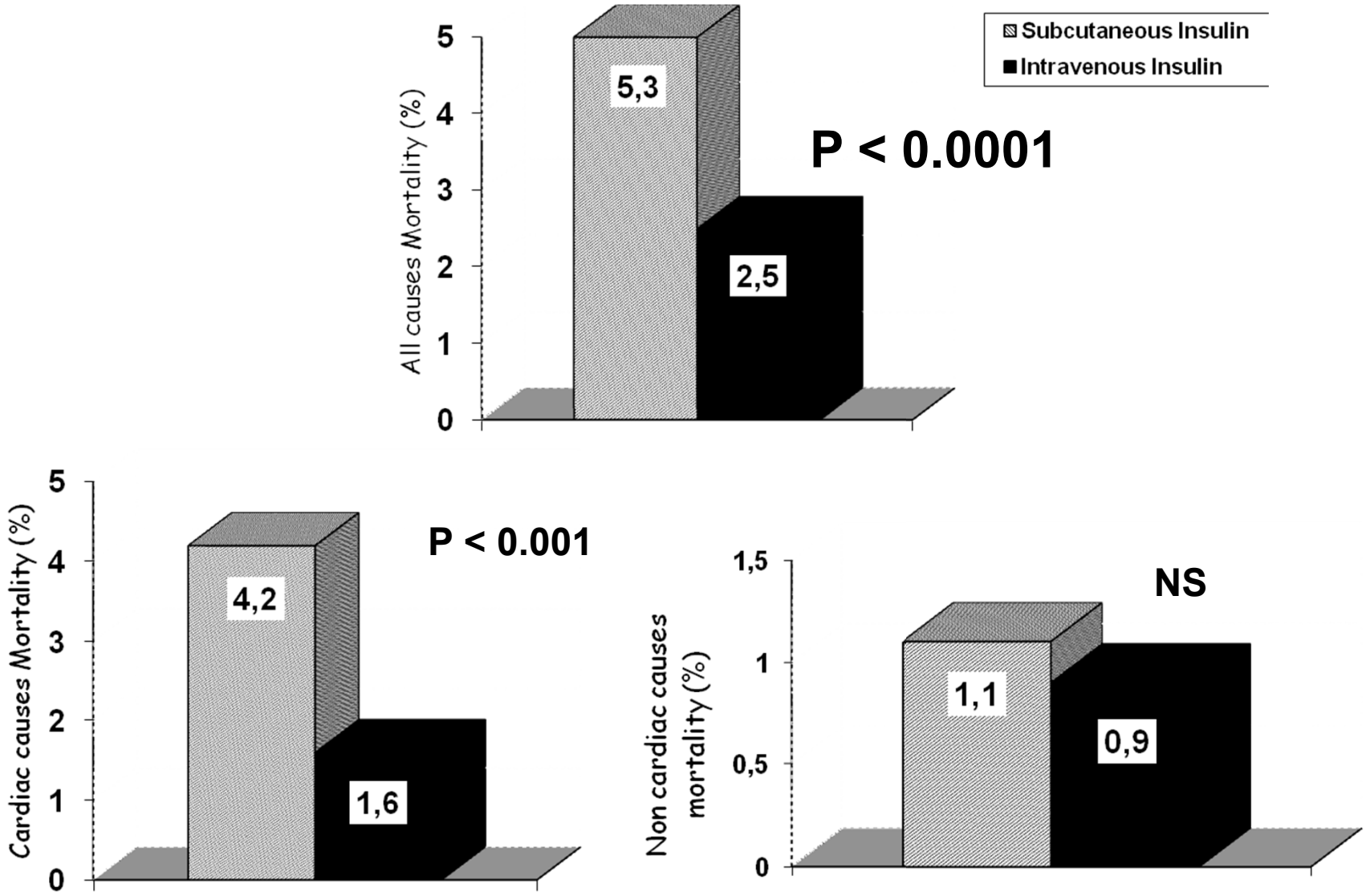
Chirurgie coronaire

Groupe Insulinothérapie sous-cutanée 1987-1991 (n=942)

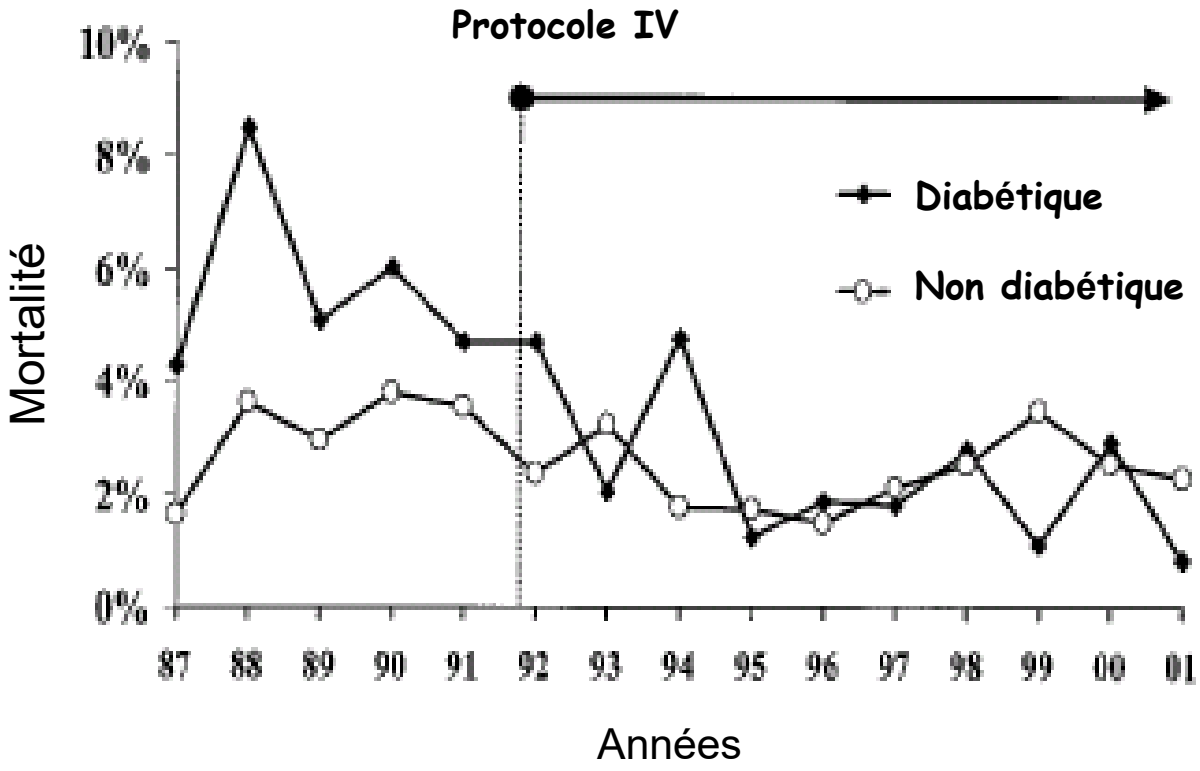
Objectif: <200 mg/dL

Groupe Insulinothérapie IVSE 1991-2001 (n=2612)

Objectifs de plus en plus audacieux (100-150 mg/dL)



J Thorac Cardiovasc Surg 2003



Furnary et al. *J Thorac Cardiovasc Surg* 2003

The New England
Journal of Medicine

VOLUME 345 NOVEMBER 8, 2001 NUMBER 19

INTENSIVE INSULIN THERAPY IN CRITICALLY ILL PATIENTS

GREET VAN DEN BERGHE, M.D., PH.D., PIETER WOUTERS, M.SC., FRANK WEEKERS, M.D., CHARLES VERWAEST, M.D., FRANS BRUYNINCKX, M.D., MIET SCHETZ, M.D., PH.D., DIRK VLASSELAERS, M.D., PATRICK FERDINANDE, M.D., PH.D., PETER LAUWERS, M.D., AND ROGER BOUILLON, M.D., PH.D.

Etude prospective randomisée unicentrique

Réanimation chirurgicale (cardiaque +++)

Insulinothérapie Intensive (80-110 mg/dL) n=765

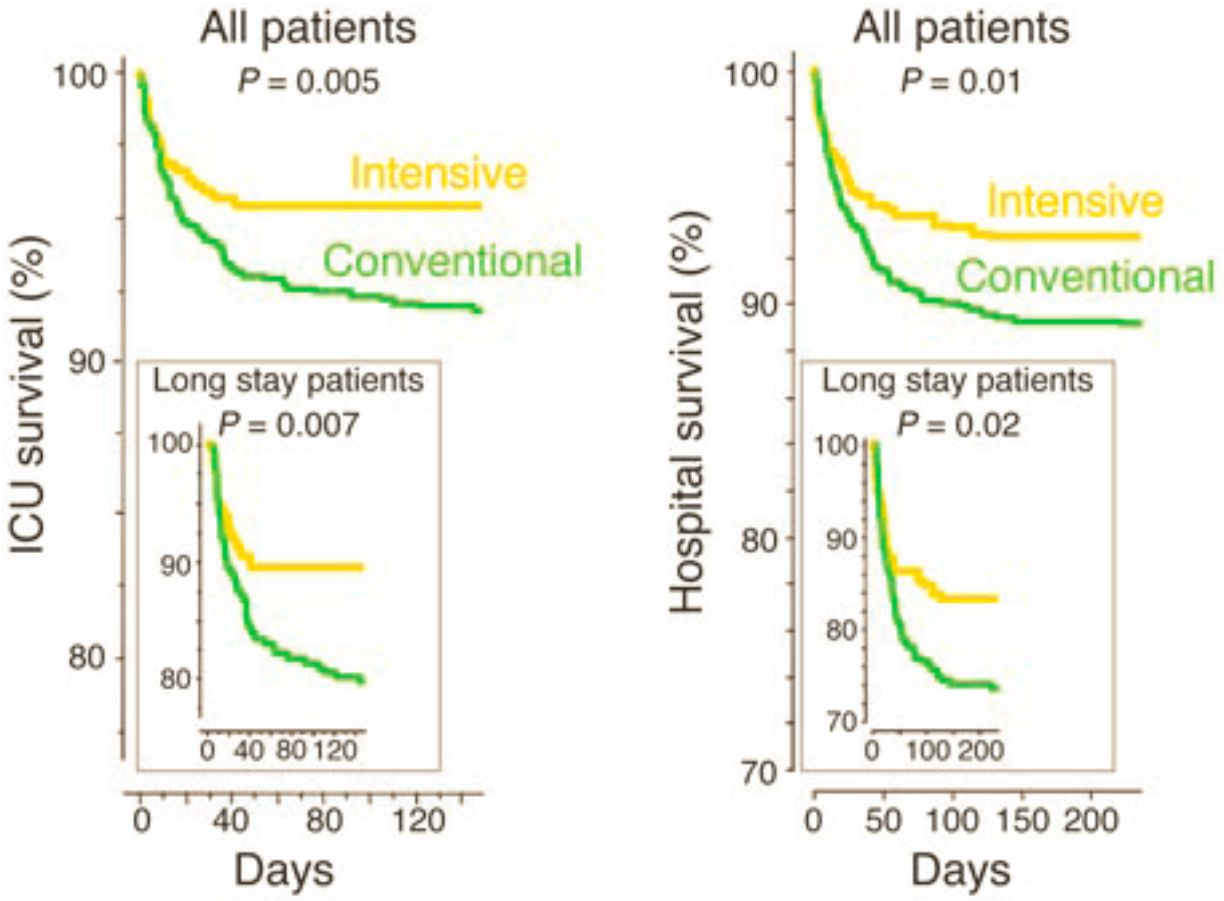
versus

Insulinothérapie conventionnelle (180-200 mg/dL) n=783

Critère de jugement principal = Mortalité durant le séjour en réanimation

TABLE 1. BASE-LINE CHARACTERISTICS OF THE PATIENTS.*

CHARACTERISTIC	CONVENTIONAL TREATMENT (N=783)	INTENSIVE TREATMENT (N=765)
Male sex — no. (%)	557 (71)	544 (71)
Age — yr	62.2±13.9	63.4±13.6
Body-mass index†	25.8±4.7	26.2±4.4
Reason for intensive care — no. (%)		
Cardiac surgery	493 (63)	477 (62)
Noncardiac indication	290 (37)	288 (38)
Neurologic disease, cerebral trauma, or brain surgery	30 (4)	33 (4)
Thoracic surgery, respiratory insufficiency, or both	56 (7)	66 (9)
Abdominal surgery or peritonitis	58 (7)	45 (6)
Vascular surgery	32 (4)	30 (4)
Multiple trauma or severe burns	35 (4)	33 (4)
Transplantation	44 (6)	46 (6)
Other	35 (4)	35 (5)



Van den Berghe G et al. N Engl J Med 2001

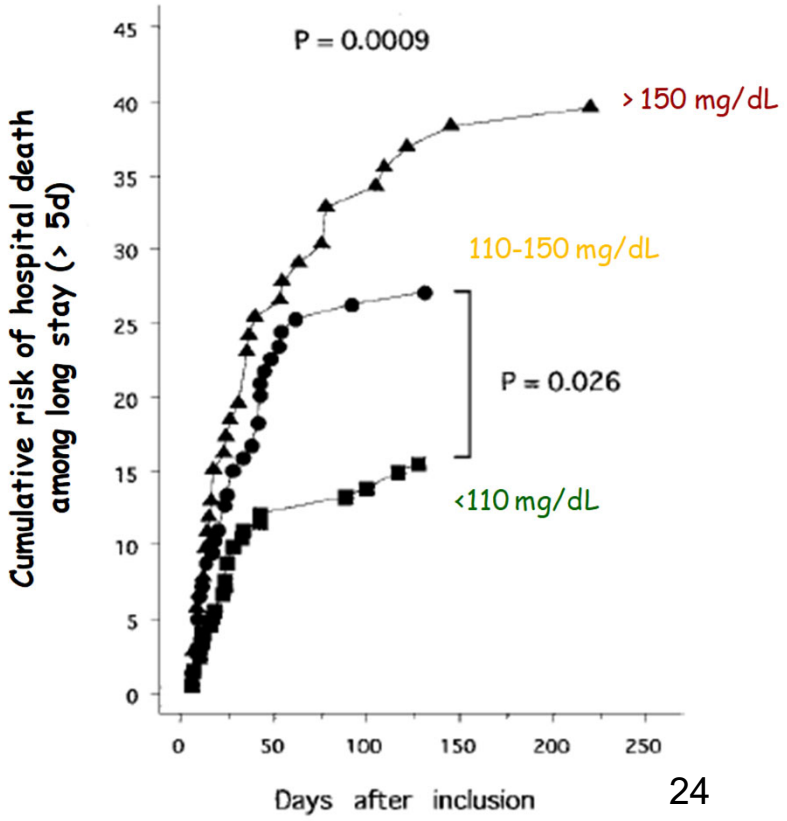
Outcome benefit of intensive insulin therapy in the critically ill: Insulin dose versus glycemic control*

Greet Van den Berghe, MD, PhD; Pieter J. Wouters, MSc; Roger Bouillon, MD, PhD; Frank Weekers, MD; Charles Verwaest, MD; Miet Schetz, MD, PhD; Dirk Vlasselaers, MD; Patrick Ferdinande, MD, PhD; Peter Lauwers, MD

Crit Care Med 2003; 31:359–366

Facteurs prédictifs de mortalité (régression logistique)

	OR	95% CI	*p* Value
Age (per 10 yrs added)	1.360	1.140–1.580	.001
Delayed ICU admission	1.882	1.069–3.314	.03
At-admission APACHE II >9	5.054	2.524–10.120	<.0001
Reason for ICU admission (vs. cardiac surgery OR 1)			
Multiple trauma or severe burns	4.851	1.664–14.141	.004
Neurologic disease, cerebral trauma, or brain surgery	4.814	2.044–11.339	.0003
Thoracic surgery and/or respiratory insufficiency	2.966	1.242–7.084	.01
Abdominal surgery and/or peritonitis	2.466	1.017–5.979	.05
Transplantation	0.746	0.197–2.820	.7
Vascular surgery	1.336	0.433–4.123	.6
Other	1.904	0.642–5.644	.2
History of malignancy	1.504	0.779–2.905	.2
At-admission hyperglycemia (≥200 mg/dL)	1.128	0.601–2.116	.7
History of diabetes	0.356	0.158–0.803	.01
Daily insulin dose (per 10 units added)	1.060	1.020–1.090	.005
Mean blood glucose level (per 20 mg/dL added)	1.300[a]	1.180–1.420	<.0001



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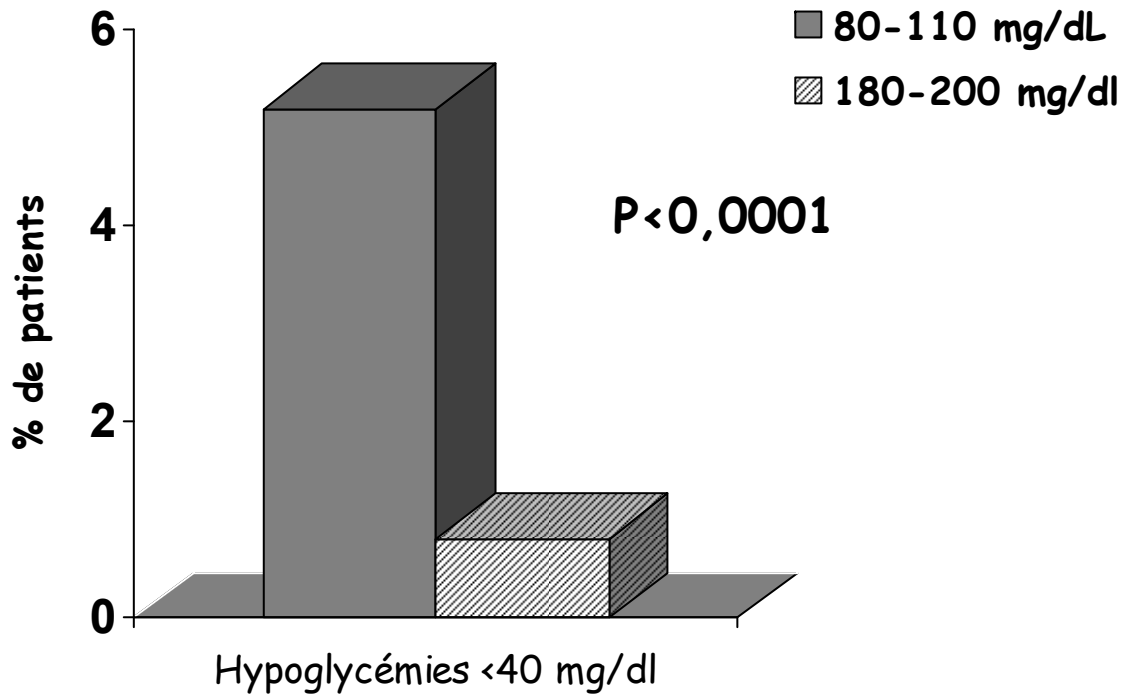
INTENSIVE INSULIN THERAPY IN CRITICALLY ILL PATIENTS



Benefits and Risks of Tight Glucose Control in Critically Ill Adults

A Meta-analysis

JAMA. 2008;300(8):933-944

Renda Soylemez Wiener, MD, MPH
Daniel C. Wiener, MD
Robin J. Larson, MD, MPH

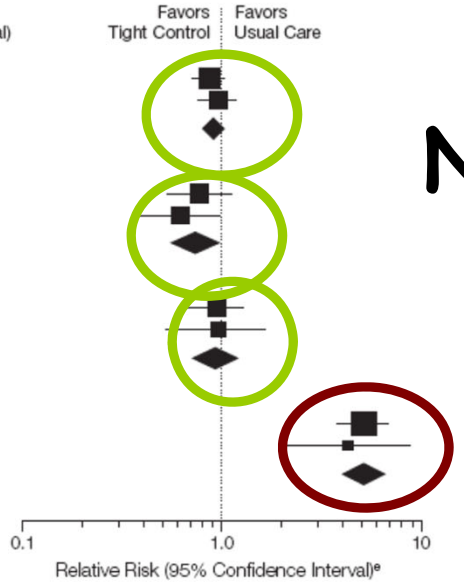
Méta-analyse: 29 RCT (n=8432)

Impact contrôle glycémique strict (<110 mg.dL^{-1}) versus conventionnel

Mortalité 30 jours

Figure 3. Association of Tight Glucose Control vs Usual Care With Outcomes Among Critically Ill Adults, Stratified by Glucose Goal in Tight Control Group

Subgroup	No. of Studies	Outcome, No./Total No. of Patients (%)		Relative Risk (95% Confidence Interval)
		Tight Control	Usual Care	
Hospital mortality[a]				
Very tight control	14	702/3031 (23.2)	781/3099 (25.2)	0.90 (0.77-1.04)
Moderately tight control	13	190/1096 (17.3)	196/1089 (18.0)	0.99 (0.83-1.18)
Overall	27	892/4127 (21.6)	977/4188 (23.3)	0.93 (0.85-1.03)
Septicemia[b]				
Very tight control	4	186/1654 (11.2)	221/1672 (13.2)	0.80 (0.57-1.11)
Moderately tight control	5	26/295 (8.8)	43/295 (14.6)	0.64 (0.41-1.00)
Overall	9	212/1949 (10.9)	264/1967 (13.4)	0.76 (0.59-0.97)
New need for dialysis[c]				
Very tight control	5	172/1424 (12.1)	193/1475 (13.1)	0.95 (0.70-1.29)
Moderately tight control	4	28/366 (7.7)	29/364 (8.0)	0.98 (0.59-1.61)
Overall	9	200/1790 (11.2)	222/1839 (12.1)	0.96 (0.76-1.20)
Hypoglycemia (glucose ≤40 mg/dL)[d]				
Very tight control	11	409/2895 (14.1)	75/2952 (2.5)	5.23 (4.12-6.64)
Moderately tight control	4	41/380 (10.8)	9/386 (2.3)	4.37 (2.19-8.72)
Overall	15	450/3275 (13.7)	84/3338 (2.5)	5.13 (4.09-6.43)



NS

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 MARCH 26, 2009 VOL. 360 NO. 13

N Engl J Med 2009;360:1283-97.

Intensive versus Conventional Glucose Control in Critically Ill Patients

The NICE-SUGAR Study Investigators*

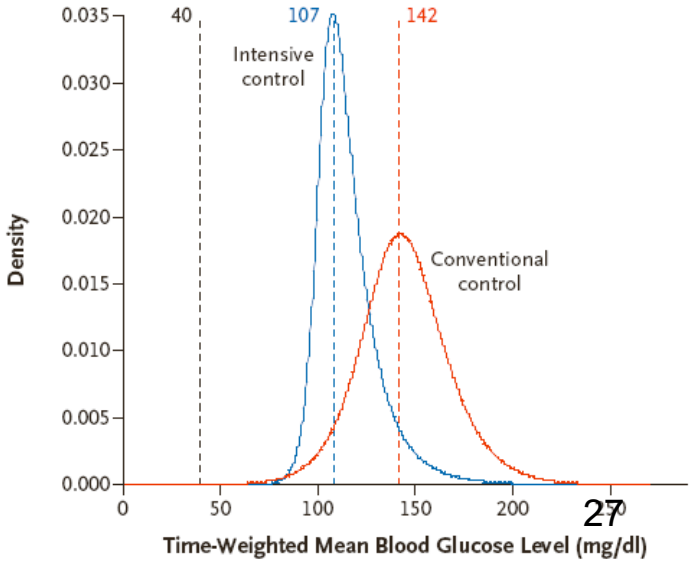
Etude prospective, randomisée, multicentrique (42 centres australo-canadiens+++)

Réanimation polyvalente entre 2004-2008 (n=6030)

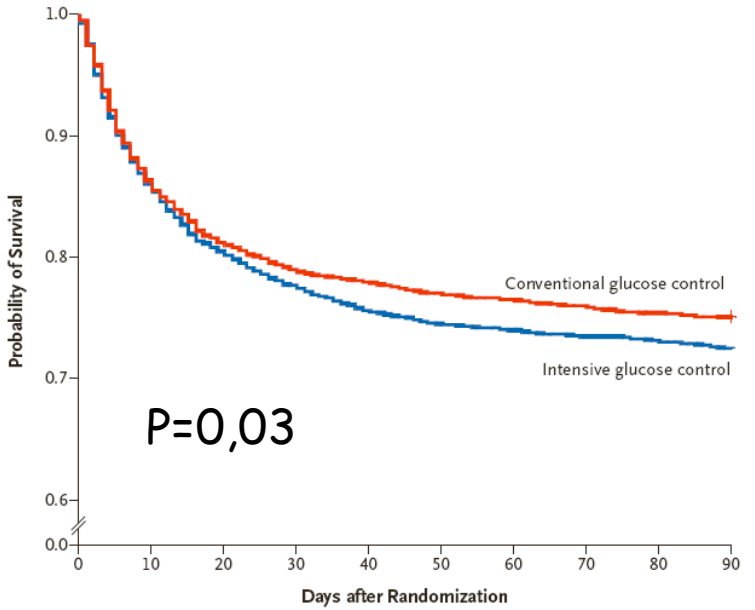
Insulinothérapie intensive (81-110 mg/dL) versus conventionnelle (<180 mg/dL)

Critère de jugement principal : mortalité à J90

Insulinothérapie IV : 97,2% versus 69,0% (p<0,001)



Outcome Measure	Intensive Glucose Control	Conventional Glucose Control	Odds Ratio or Absolute Difference (95% CI)†	Statistical Test	P Value
Death — no. of patients/total no. (%)				Logistic regression	
At day 90	829/3010 (27.5)	751/3012 (24.9)	1.14 (1.02 to 1.28)		0.02
At day 28	670/3010 (22.3)	627/3012 (20.8)	1.09 (0.96 to 1.23)		0.17



No. at Risk

Conventional control	3014	2379	2304	2261
Intensive control	3016	2337	2227	2182

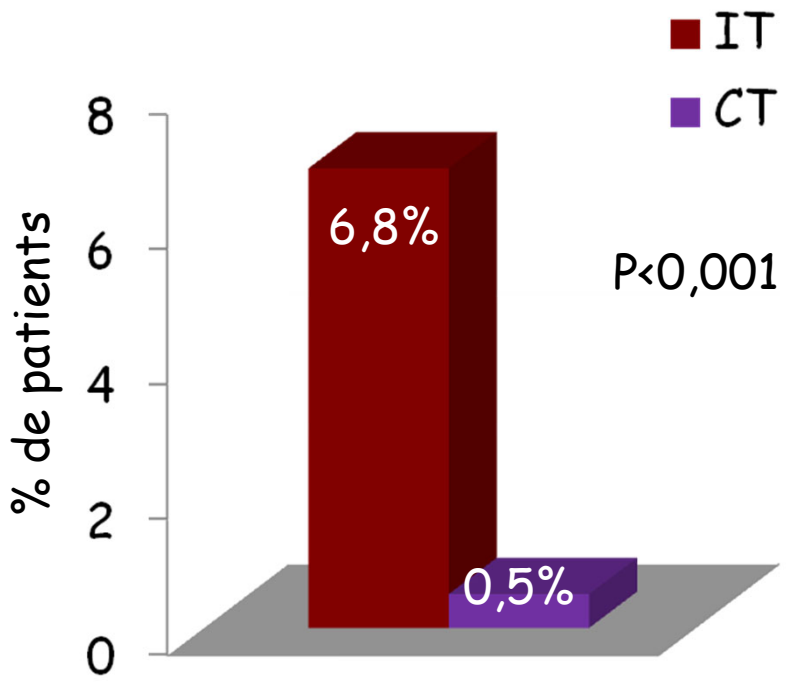
A multi-centre, open label, randomised controlled trial of two target ranges for glycaemic control in Intensive Care Unit patients

THE NICE STUDY

THE NICE STUDY

Décès d' origine cardiovasculaire 42% versus 36% (P<0,02)

Hypoglycémie (<40 mg/dL)

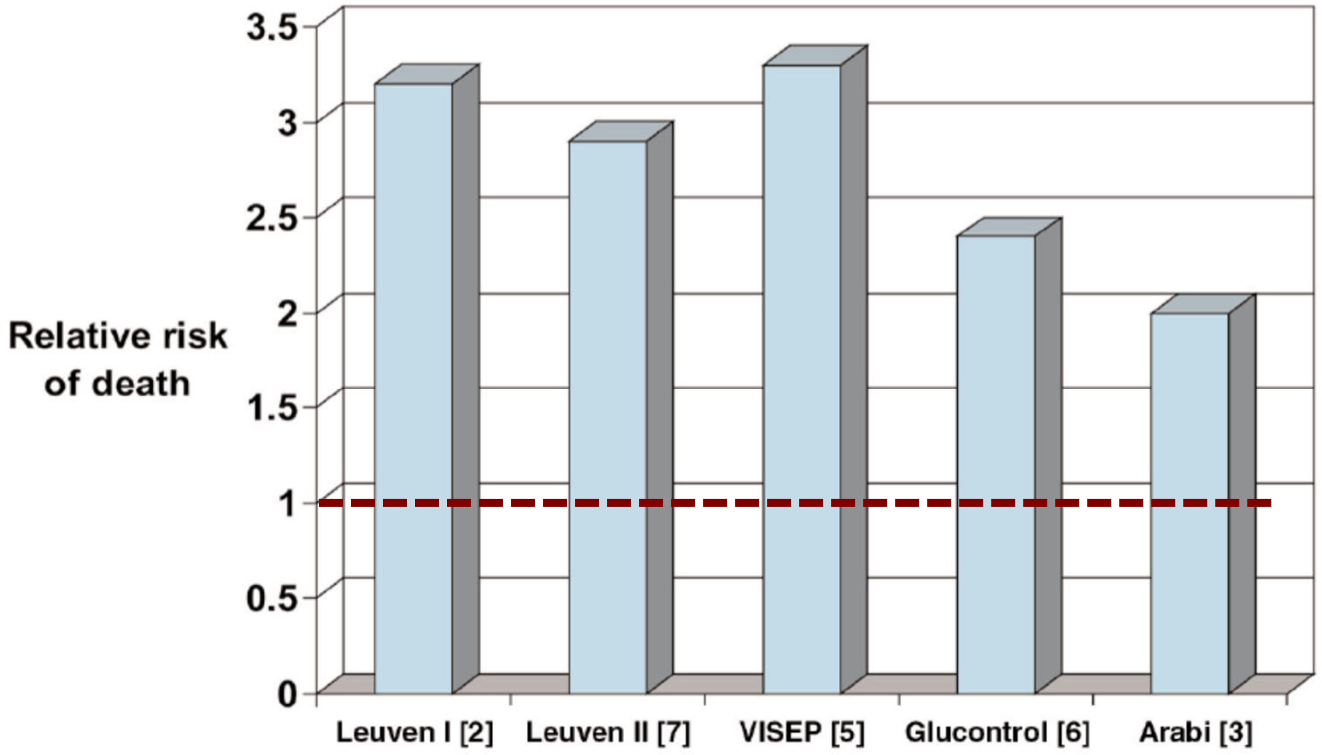


NICE-SUGAR: the end of a sweet dream?

Jean-Charles Preiser

Critical Care 2009, **13**:143 (doi:10.1186/cc7790)

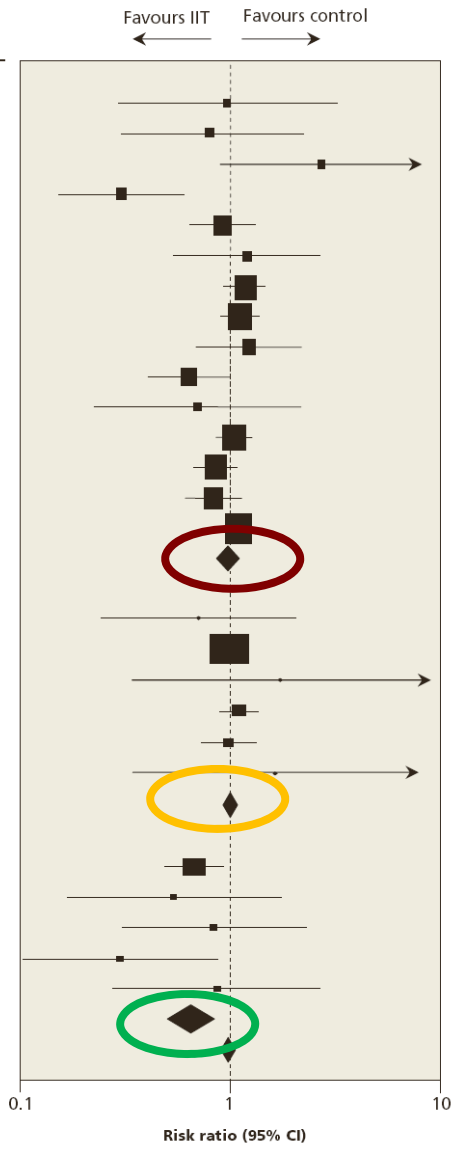
Impact de l' hypoglycémie sur la mortalité (RR)



Griesdale DEG et al. CMAJ 2009;180:821-7

Intensive insulin therapy and mortality among critically ill patients: a meta-analysis including NICE-SUGAR study data

Study	No. deaths / total no. patients: IIT	Control	Risk ratio (95% CI)
Mixed ICU			
[illegible] et al.[39]	4/28	4/27	0.96 (0.27–3.47)
Henderson et al.[31]	5/32	7/35	0.78 (0.28–2.22)
Mitchell et al.[35]	9/35	3/35	3.00 (0.89–10.16)
Wang et al.[38]	7/58	26/58	0.27 (0.13–0.57)
Azevedo et al.[22]	38/168	42/169	0.91 (0.62–1.34)
McMullin et al.[34]	6/11	4/9	1.23 (0.49–3.04)
Devos et al.[13]	107/550	89/551	1.20 (0.93–1.55)
Brunkhorst et al.[11]	98/247	102/288	1.12 (0.90–1.39)
Iapichino et al.[32]	15/45	12/45	1.25 (0.66–2.36)
He et al.[30]	16/58	29/64	0.61 (0.37–1.00)
Zhang et al.[40]	4/168	6/170	0.67 (0.19–2.35)
De La Rosa Gdel et al.[12]	102/254	96/250	1.05 (0.84–1.30)
Arabi et al.[10]	72/266	83/257	0.84 (0.64–1.09)
Mackenzie et al.[33]	39/121	47/119	0.82 (0.58–1.15)
NICE-SUGAR[18]	829/3010	751/3012	1.10 (1.01–1.20)
All mixed ICU patients	1351/5051	1301/5089	0.99 (0.87–1.12)
Medical ICU			
[illegible] et al.[25]	1/5	2/5	0.50 (0.06–3.91)
Van den Berghe et al.[9]	214/595	228/605	0.95 (0.82–1.11)
Walters et al.[37]	1/13	0/12	2.79 (0.12–62.48)
Farah et al.[27]	22/41	22/48	1.17 (0.77–1.78)
Oksanen et al.[36]	13/39	18/51	0.94 (0.53–1.68)
Bruno et al.[26]	2/31	0/15	2.50 (0.13–49.05)
All medical ICU patients	253/724	270/736	1.00 (0.78–1.28)
Surgical ICU			
Van den Berghe et al.[8]	55/765	85/783	0.66 (0.48–0.92)
Grey et al.[28]	4/34	6/27	0.53 (0.17–1.69)
Bilotta et al.[24]	6/40	7/38	0.81 (0.30–2.20)
He et al.[29]	7/150	6/38	0.30 (0.11–0.83)
Bilotta et al.[23]	5/48	6/49	0.85 (0.28–2.60)
All surgical ICU patients	77/1037	110/935	0.63 (0.44–0.91)
All ICU patients	1681/6812	1681/6760	0.93 (0.83–1.04)



Continuous insulin infusion reduces mortality in patients with diabetes undergoing coronary artery bypass grafting

Anthony P. Furnary, MD,[a,d] Guangqiang Gao, MD,[a] Gary L. Grunkemeier, PhD,[b] YingXing Wu, MD,[b] Kathryn J. Zerr, MBA,[b] Stephen O. Bookin, MD,[c] H. Storm Floten, MD,[a,d] and Albert Starr, MD[a,d]

Left to right: Grunkemeier, Furnary, Wu, Gao, Zerr (*front ro*

4. *Cardiac-related mortality* referred to all deaths in which arrhythmia or pump failure was identified as the seminal cause of death.

Study Groups

All patients with DM were divided into two sequential groups according to the type of perioperative glycemic control that they received.

Subcutaneous insulin group. Patients operated on between January 1987 and September 1991, the subcutaneous insulin (SQI) group (n = 942), received subcutaneous insulin injections every 4 hours in a directed attempt to maintain blood glucose levels below 200 mg/dL. Sliding scale dosage of insulin was titrated to each patient's glycemic response during the previous 4 hours. These sliding scale SQI injections were continued every 4 hours throughout the patients' hospital course, even after the resumption of their preoperative glucose control regimen.

CII group. All patients with DM undergoing CABG operated on between October 1991 and December 2001, the CII group (n = 2612), received a CII infusion titrated per protocol in the perioperative period (Portland protocol).[16] The current Portland protocol

patient was transferred to the telemetry unit. In January 1996, the protocol was expanded with initiation in the operating room (before sternotomy and after induction of anesthesia, with continuation during cardiopulmonary bypass) and uniform continuation until 7 AM of the third POD, even for patients who had transferred out of the ICU.

set. By means of this nationally recognized risk assessment, all patients were assigned an expected probability of death. Predicted and observed hospital mortalities were then compared, along with the observed/expected risk ratios. The composite STS risk score was calculated as the logit of the probability of death.

Univariate analyses between groups were done with *t* tests and χ^2 analyses. The Bonferroni correction was applied to adjust for multiple comparisons between groups. Stepwise logistic regression was used to produce risk models for hospital death. to measure model discrimination, c-statistics (area under the receiver operating characteristic curve) were used, and the Hosmer-Lemeshow statistic was used to measure calibration.[17] The purpose was to make internal comparisons rather than to produce a prediction equation for use outside of this data set. Thus all patients were used, rather than separating the data into training and testing subsets or applying shrinkage methods to the coefficients. All statistical analyses were performed with SPSS software (version 10.0; SPSS, Inc, Chicago, Ill).

Results

Data Analysis

In-hospital mortality was the primary end point of this study. Patient groups were analyzed on an intent-to-treat basis. According to this method, intraoperative and first POD deaths were included in the end point analysis even though those patients did not complete the 3-day SQI or CII treatment protocols. This was considered to be the most rigorous method to test our hypothesis.

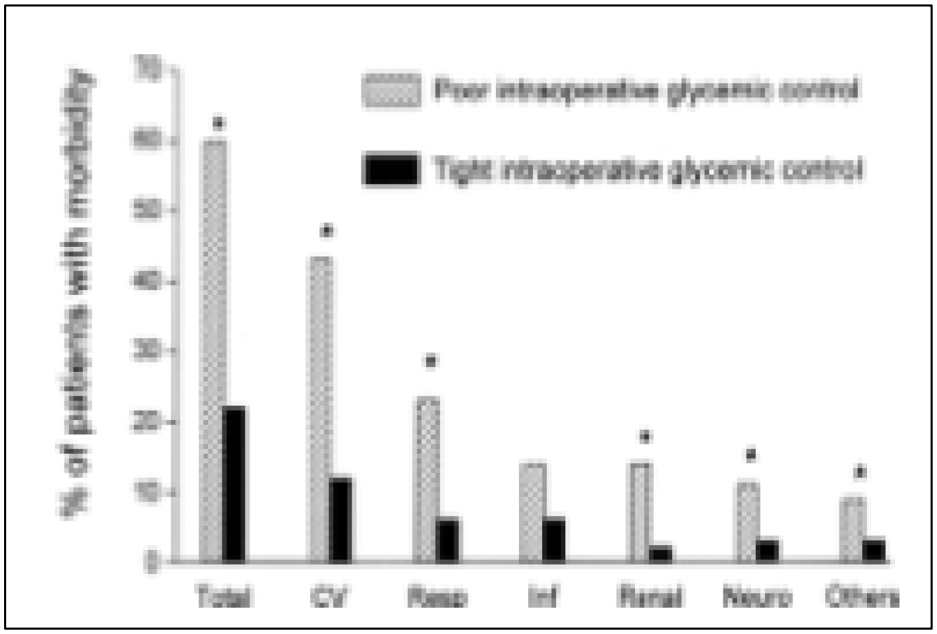
An internal logistic regression model was developed to determine the effect of perioperative hyperglycemic treatment method (SQI vs CII) on operative mortality after adjustment for other known preoperative risk factors. The external risk model was taken from the 1996 STS risk algorithm.[2] The 1996 model was chosen because that year contained the median patient of the study's data

room or within 24 hours of surgery (n = 24) had "operative catastrophes" and did not survive long enough to derive a benefit from initiation of CII therapy. If our analysis were to be evaluated on an actual treatment (as opposed to intent-to-treat) basis, SQI mortality would have been 4.5% (n = 42/934), and CII mortality would have been 1.9% (n = 49/2596, $P < .0001$).

Cause of Death

The seminal causes of death for each of the 115 patients who died were pump failure in 54% (n = 62), arrhythmia in 17% (n = 20), neurologic causes in 19% (n = 22), respiratory failure in 5% (n = 6), renal failure in 3% (n = 3),

Poor Intraoperative Blood Glucose Control Is Associated with a Worsened Hospital Outcome after Cardiac Surgery in Diabetic Patients



Poor intraoperative glucose control was defined by > 200 $mg.dL^{-1}$

Table 4. Independent Risk Factors of Severe Adverse In-hospital Outcome in Diabetic Patients after On-pump Cardiac Surgery (n = 200)

Variable	Odds Ratio (95% CI)	*P* Value
Pulmonary hypertension*	12.4 (2.7–57.4)	0.001
Poor intraoperative glycemic control	7.2 (2.7–19.0)	< 0.001
Intraoperative erythrocyte transfusion	5.4 (2.3–12.6)	< 0.001
Hypothermic CPB†	3.0 (1.2–7.3)	0.01
Preoperative plasma creatinine	1.02 (1.00–1.03)‡	0.001
Cardiopulmonary bypass time	1.02 (1.01–1.04)§	0.01

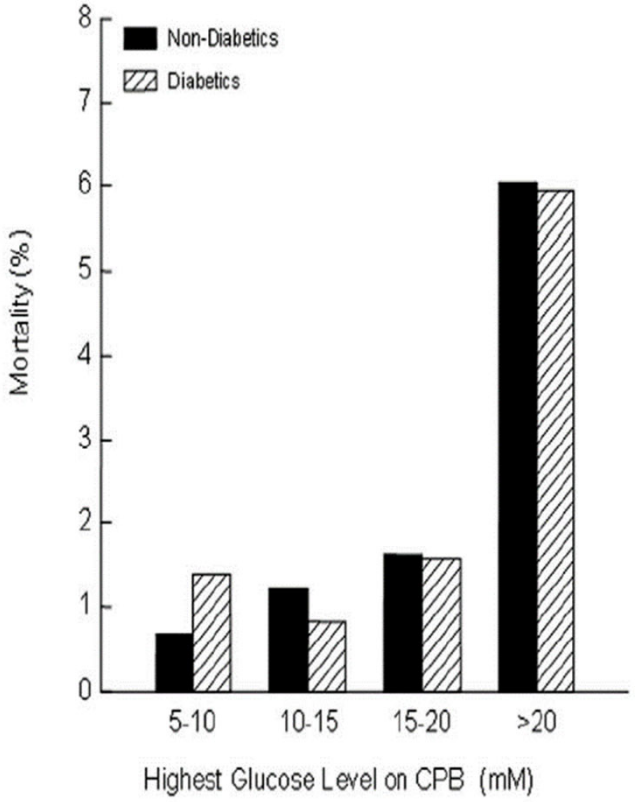
Ouattara A et al. Anesthesiology 2005;103:687-94

Hyperglycemia during cardiopulmonary bypass is an independent risk factor for mortality in patients undergoing cardiac surgery

Torsten Doenst, MD, PhD,[a,c] Duminda Wijeysundera, MD,[b] Keyvan Karkouti, MD,[b] Christoph Zechner, MD,[c] Manjula Maganti, MSc,[a] Vivek Rao, MD, PhD,[a] and Michael A. Borger, MD, PhD[a]

J Thorac Cardiovasc Surg 2005;130:1144-50

Corrélation entre le pic de glycémie peropératoire et la mortalité postopératoire chez les patients non diabétiques (4701) et les diabétiques (1579) opérés entre 1999 et 2001

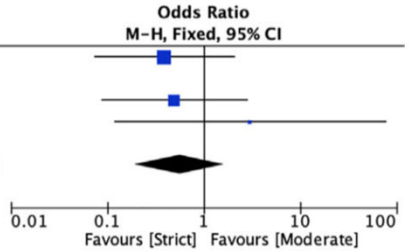


Jin X et al. Front Endocrinol 2020.11:513073

Mortalité postopératoire

Study or Subgroup	Strict Events	Strict Total	Moderate Events	Moderate Total	Weight	Odds Ratio M-H, Fixed, 95% CI
Kirdemir 2008	2	100	5	100	53.0%	0.39 [0.07, 2.05]
Lazar 2011	0	40	0	42		Not estimable
Wahby 2016	2	67	4	68	41.7%	0.49 [0.09, 2.78]
Zadeh 2016	1	38	0	37	5.3%	3.00 [0.12, 76.03]
Total (95% CI)		**245**		**247**	**100.0%**	**0.57 [0.20, 1.66]**
Total events	5		9			

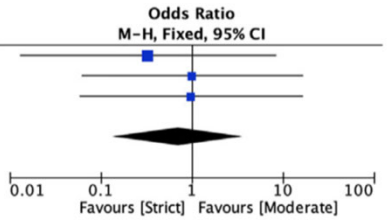
Heterogeneity: $Chi^2 = 1.25$, $df = 2$ ($P = 0.54$); $I^2 = 0\%$
Test for overall effect: $Z = 1.03$ ($P = 0.30$)



AVC postopératoire

Study or Subgroup	Experimental Events	Experimental Total	Control Events	Control Total	Weight	Odds Ratio M-H, Fixed, 95% CI
Asida 2013	0	50	1	50	42.9%	0.33 [0.01, 8.21]
Kirdemir 2008	1	100	1	100	28.6%	1.00 [0.06, 16.21]
Zadeh 2016	1	38	1	37	28.5%	0.97 [0.06, 16.15]
Total (95% CI)		**188**		**187**	**100.0%**	**0.70 [0.14, 3.62]**
Total events	2		3			

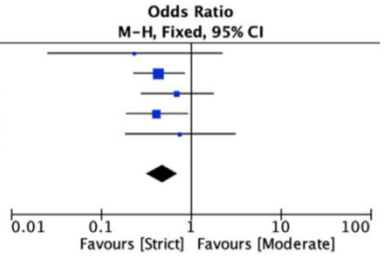
Heterogeneity: $Chi^2 = 0.33$, $df = 2$ ($P = 0.85$); $I^2 = 0\%$
Test for overall effect: $Z = 0.42$ ($P = 0.67$)



ACFA postopératoire

Study or Subgroup	Experimental Events	Experimental Total	Control Events	Control Total	Weight	Odds Ratio M-H, Fixed, 95% CI
Asida 2013	1	50	4	50	5.8%	0.23 [0.03, 2.18]
Kirdemir 2008	19	100	35	100	41.9%	0.44 [0.23, 0.83]
Lazar 2011	12	40	16	42	16.1%	0.70 [0.28, 1.75]
Wahby 2016	13	67	25	68	29.5%	0.41 [0.19, 0.90]
Zadeh 2016	4	38	5	37	6.7%	0.75 [0.19, 3.06]
Total (95% CI)		**295**		**297**	**100.0%**	**0.48 [0.32, 0.72]**
Total events	49		85			

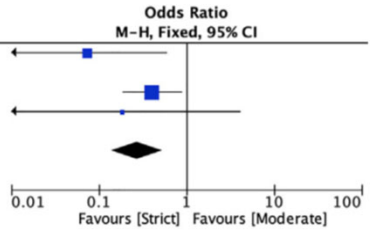
Heterogeneity: $Chi^2 = 1.65$, $df = 4$ ($P = 0.80$); $I^2 = 0\%$
Test for overall effect: $Z = 3.51$ ($P = 0.0004$)



Médiastinite postopératoire

Study or Subgroup	Experimental Events	Experimental Total	Control Events	Control Total	Weight	Odds Ratio M-H, Fixed, 95% CI
Kirdemir 2008	1	100	12	100	33.4%	0.07 [0.01, 0.58]
Lazar 2011	0	40	0	42		Not estimable
Wahby 2016	14	67	27	68	59.6%	0.40 [0.19, 0.86]
Zadeh 2016	0	38	2	37	7.0%	0.18 [0.01, 3.97]
Total (95% CI)		**245**		**247**	**100.0%**	**0.28 [0.14, 0.54]**
Total events	15		41			

Heterogeneity: $Chi^2 = 2.55$, $df = 2$ ($P = 0.28$); $I^2 = 22\%$
Test for overall effect: $Z = 3.74$ ($P = 0.0002$)



Intensive Intraoperative Insulin Therapy versus Conventional Glucose Management during Cardiac Surgery

A Randomized Trial

Ann Intern Med. 2007;146:233-243.

Gunjan Y. Gandhi, MD, MSc; Gregory A. Nuttall, MD; Martin D. Abel, MD; Charles J. Mullany, MD; Hartzell V. Schaff, MD; Peter C. O'Brien, PhD; Matthew G. Johnson, MPH; Arthur R. Williams, PhD; Susanne M. Cutshall, RN; Lisa M. Mundy, RN; Robert A. Rizza, MD; and M. Molly McMahon, MD

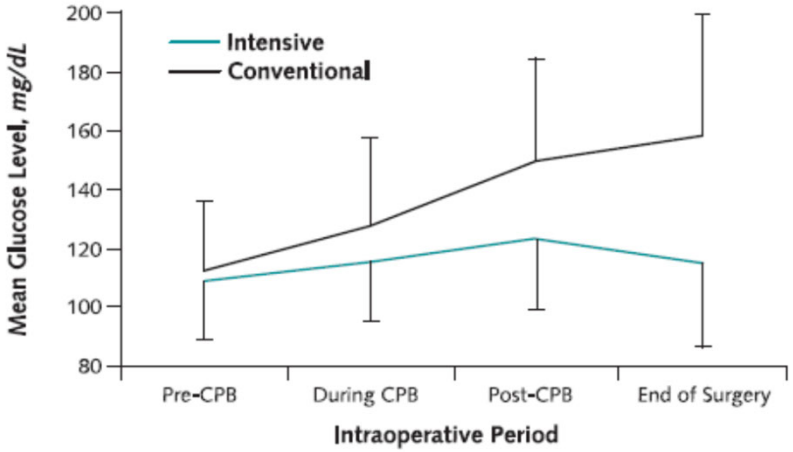
Etude prospective, randomisée, unicentrique (Rochester, MN)

Chirurgie cardiaque avec CEC (n=400)

- Groupe insulinothérapie intensive si BGL>100 mg/dl (80-100 mg/dl)
- Groupe conventionnel insulinothérapie si BGL >200 mg/dl (<150 mg/dl)

Contrôle glycémique strict postopératoire (80-110 mg/dl) pdt 24 heures

Critères de jugement (composite) : morbi-mortalité postopératoire à 30 jours



Hypoglycémie

- perop: n=1 dans chaque groupe

- postop: n=8 (intensive)
  n=14 (conventionnel)

Table 4. **Comparison of Primary and Secondary Outcomes***

Outcome	Intensive Treatment Group (n = 185), n (%)	Conventional Treatment Group (n = 186), n (%)	Relative Risk or Odds Ratio (95% CI)†	P Value‡
Any event§	82 (44)	86 (46)	1.0 (0.8 to 1.2)	0.71
In hospital	78 (42)	82 (44)		
Postdischarge (up to 30 days after surgery)	8 (4)	9 (5)		
Death	4 (2)	0 (0)	∞ (0.9 to ∞)	0.061
In hospital	4 (2)	0 (0)		
Postdischarge (up to 30 days after surgery)	0 (0)	0 (0)		
Stroke	8 (4)	1 (1)	8.0 (1.0 to 63.7)	0.020
In hospital	7 (4)	1 (1)		
Postdischarge (up to 30 days after surgery)	1 (1)	0 (0)		
Deep sternal infection	6 (3)	7 (4)	0.9 (0.3 to 2.5)	0.79
In hospital	3 (2)	1 (1)		
Postdischarge (up to 30 days after surgery)	3 (2)	6 (3)		
Cardiac arrest	1 (1)	0 (0)	∞ (0.1 to ∞)	0.50
In hospital	1 (1)	0 (0)		
Postdischarge (up to 30 days after surgery)	0 (0)	0 (0)		

Pas de différence significative sur la durée de séjour en réa ou à l' hôpital

Because of cardiopulmonary bypass (CPB)-induced alterations in insulin secretion and resistance, profound hyperglycemia occurs in patients undergoing cardiac surgery (levels may approach 1000 mg/dL). Hyperglycemia has been shown to worsen neurologic injury after focal and global cerebral ischemia, probably because of anaerobic glycolysis-induced conversion of glucose to lactate, which causes intracellular acidosis and impairment of cellular metabolism (1,2). The appropriate

intraoperative management of hyperg
whether it adversely affects neurologic o
tients after cardiac surgery, remains co
7).[1,2,3] Previous investigations have asses
injury as either perioperative gross neurol
perioperative changes in a battery of neu
examinations. However, interpretation ar
nificance of perioperative changes in neu

Attempting intraoperative tight glucose control may be difficult

Intraoperative insulin resistance (hypothermia, stress hormone, CPB…)

Postoperative requirement (insulin ristance) may decrease rapidly

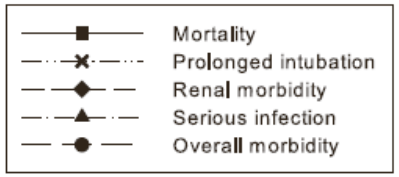
Risk of postoperative hypoglycemia

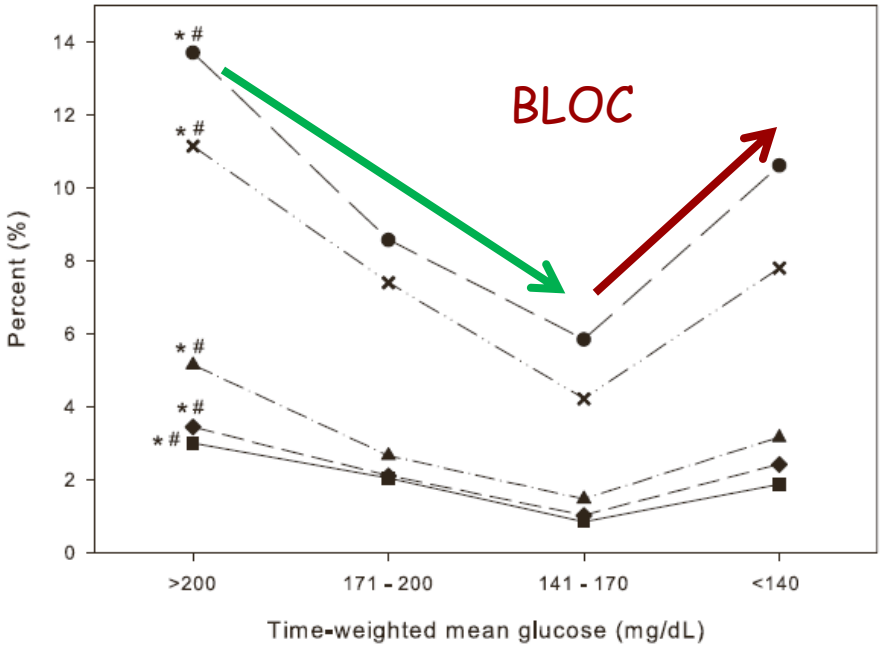
Anesth Analg 1999;89:1091-5

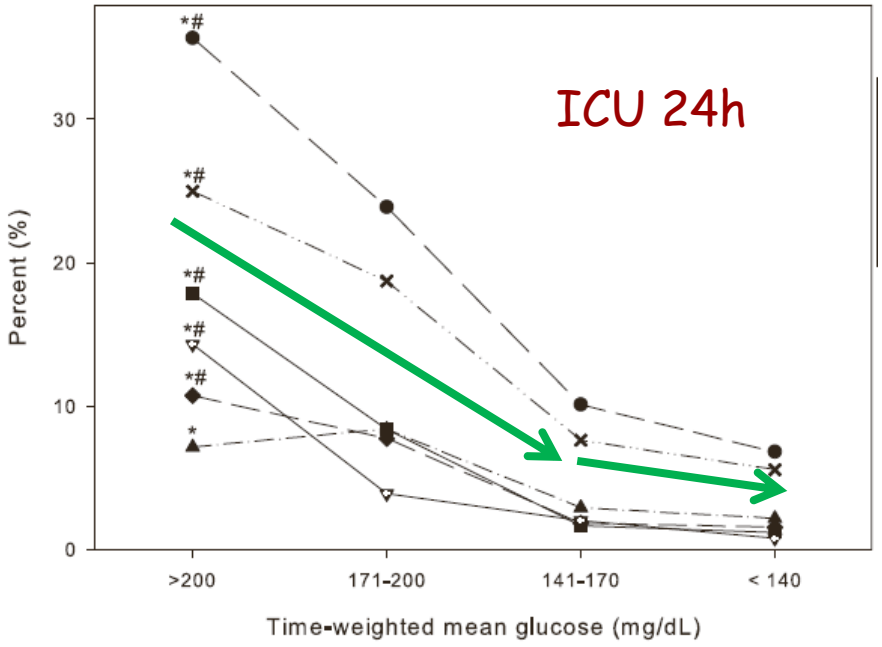
Role of Intraoperative and Postoperative Blood Glucose Concentrations in Predicting Outcomes after Cardiac Surgery

Anesthesiology 2010; 112:860–71

Andra E. Duncan, M.D.,* Alaa Abd-Elsayed, M.D.,† Ankit Maheshwari, M.D.,‡ Meng Xu, M.S.,§ Edward Soltesz, M.D., M.P.H.,‖ Colleen G. Koch, M.D., M.S.#







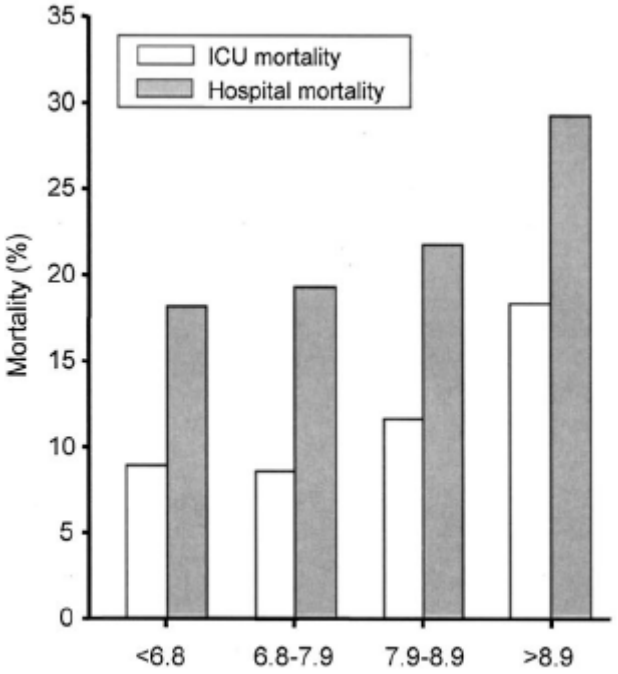
Variability of Blood Glucose Concentration and Short-term Mortality in Critically Ill Patients

Moritoki Egi, M.D., Rinaldo Bellomo, M.D., F.J.F.I.C.M.,† Edward Stachowski, M.D.,‡ Craig J. French, M.D.,§ Graeme Hart, M.D.‖*

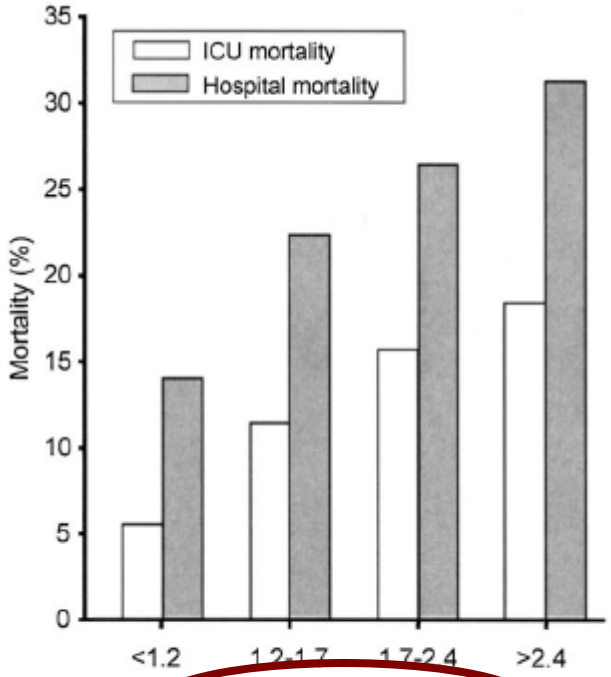
Anesthesiology 2006; 105:244–52

Etude multicentrique rétrospective (2000-2004)

Réanimation polyvalente (n=7049)



Valeur moyenne de glycémie (mmol/L)



Déviation standard
des valeurs de glycémie (mmol/L)

Increased Glucose Variability Is Associated With Major Adverse Events After Coronary Artery Bypass

Clement KC et al. Ann Thorac Surg 2019;108:1307-13

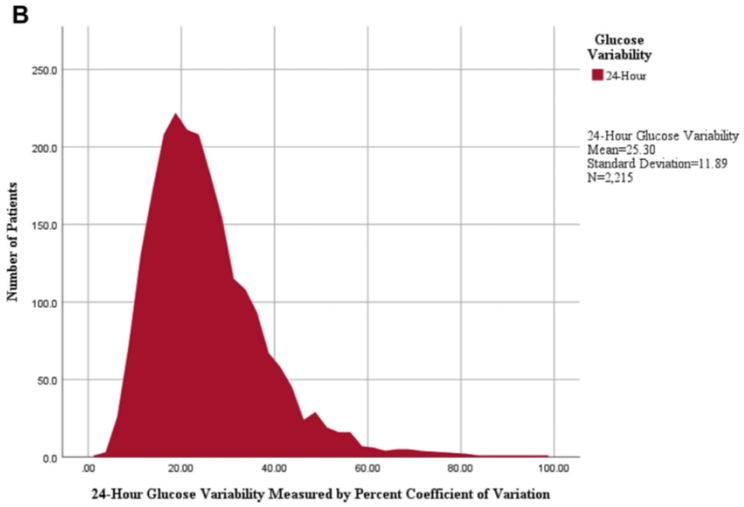


Table 2. Multivariate Logistic Regression of Postoperative Glucose Variability and Major Adverse Events[a]

Variable	Odds Ratio	95% CI Lower	95% CI Upper	*P* Value
Preoperative factors and 12-hour glucose variability				
Age	1.02	1.01	1.04	.003
Hemoglobin A_{1c}	0.97	0.88	1.07	.54
Ejection fraction	0.97	0.96	0.98	<.001
Hyperlipidemia	1.49	1.05	2.13	.027
Hypertension	0.62	0.40	0.97	.036
Square root of Cardiopulmonary bypass time (min)	1.15	1.07	1.24	<.001
Total blood products transfused in OR	1.52	1.38	1.68	<.001
12-hour glucose variability	1.06	0.87	1.29	.58
Constant	0.02			<.001

Postoeprative MAE= cardiac arrest, pneumonia, renal failure, stroke, sepsis, reoperation, 30-day mortality

Increased Glycemic Variability in Patients with Elevated Preoperative HbA1C Predicts Adverse Outcomes Following Coronary Artery Bypass Grafting Surgery

Balachundhar Subramaniam, MD, MPH,* Adam Lerner, MD,* Victor Novack, MD, PhD,†‡ Kamal Khabbaz, MD,§ Maya Paryente-Wiesmann, MD,† Philip Hess, MD, PhD,* and Daniel Talmor, MD, MPH*

(Anesth Analg 2014;118:277–87)

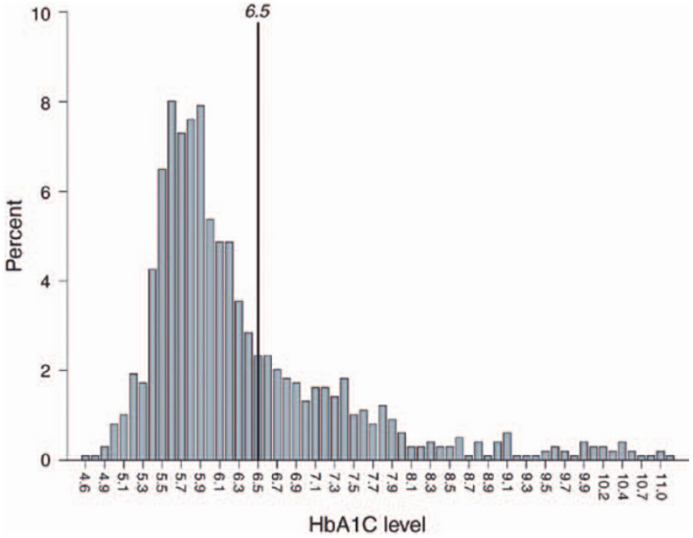


Figure 1. Hemoglobin A1C (HbA1C) distribution in our patient population. HbA1C levels (%) are seen in the x-axis.

Table 2. Comparison Between Outcome Variables of Patients with HbA1C >6.5% and <6.5%

Variables	All patients (*n* = 1461)	HbA1C <6.5% (*n* = 1003)	HbA1C ≥6.5% (*n* = 458)	*P*
Perioperative/postoperative complications	143 (9.8%)	87 (8.7%)	56 (12.2%)	0.034
MI	5 (0.3%)	5 (0.5%)	0	0.333
Reoperation (bleeding)	32 (2.2%)	22 (2.2%)	10 (2.2%)	0.990
Deep sternal infection	15 (1%)	5 (0.5%)	10 (2.2%)	0.008
Stroke	19 (1.3%)	12 (1.2%)	7 (1.5%)	0.603
Pneumonia	36 (2.5%)	21 (2.1%)	15 (3.3%)	0.177
Renal failure	44 (3%)	27 (2.7%)	17 (3.7%)	0.290
Tamponade	1 (0.1%)	0	1 (0.2%)	0.313
Death	42 (2.9%)	30 (3%)	12 (2.6%)	0.704
Atrial fibrillation	122 (26.4%)	264 (26.3%)	122 (26.6%)	0.90

Intensive Care Med (2007) 33:2079–2084
DOI 10.1007/s00134-007-0835-4

ORIGINAL

Accuracy of bedside capillary blood glucose measurements in critically ill patients

C. Dana Critchell
Vincent Savarese
Amy Callahan
Christine Aboud
Serge Jabbour
Paul Marik

Comparaison de la glycémie capillaire versus laboratoire
Réanimaiton polyvalente (80 patients sur 4 mois)

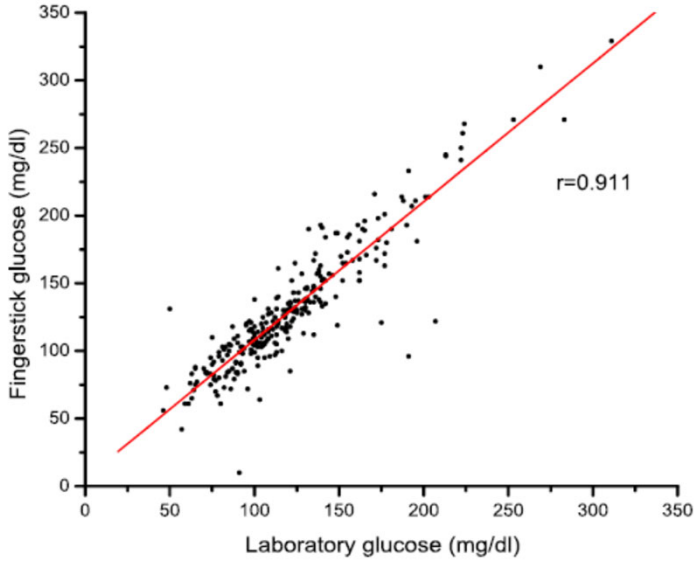
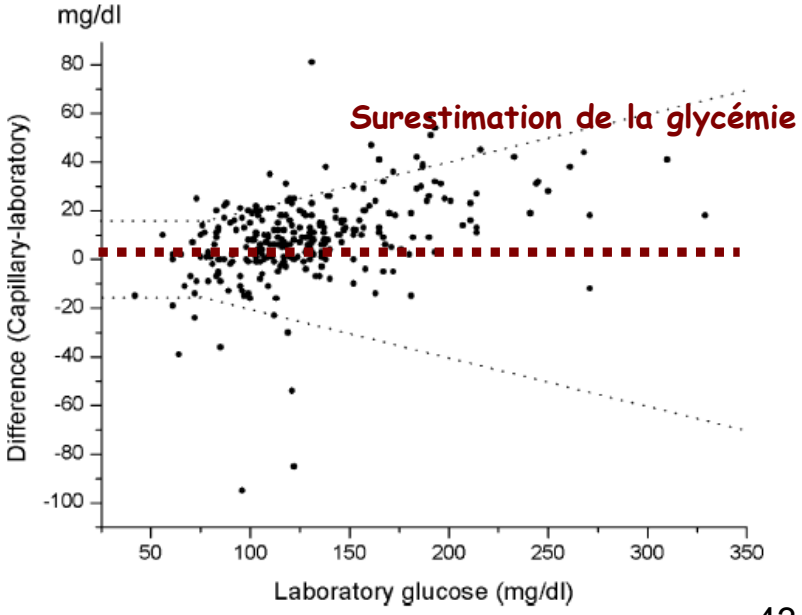


Fig. 1 Plot of laboratory glucose vs. fingerstick glucose



Glucose Measurement in the Operating Room: More Complicated than It Seems

Anesth Analg 2010;110:1056-65

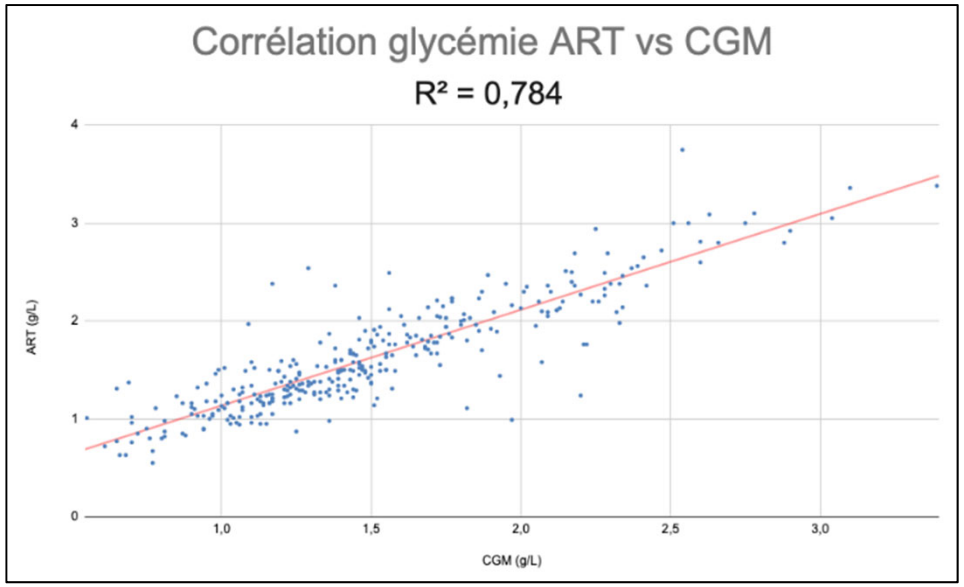
Mark J. Rice, MD,* Andrew D. Pitkin, MBBS, MRCP, FRCA,* and Douglas B. Coursin, MD†

	Methodology affected[a]	
Variable	**GO**	**GD**
Whole blood	↓	↓
Arterial	↑	↑
Capillary	↑	↑
Postprandial state	↑	↑
Hematocrit		
Anemia	↑	↑
Polycythemia	↓	↓
Oxygen concentration		
Hypoxia	↑	—
Oxygen therapy	↓	—
pH (6.8–7.55)	—	—
Low pH	—/↓	—
High pH	—/↑	—
Hypothermia	↑	↓/↑
Hypotension	↑	↓/↑
Drugs		
Ascorbic acid	↓	↓/—
Acetaminophen	↓	↑
Dopamine	—	↓
Icodextrin	—	↑
Mannitol	↑	—

Reprinted from Dungan et al.,[24] with permission.

GO = glucose oxidase; GD = glucose dehydrogenase.

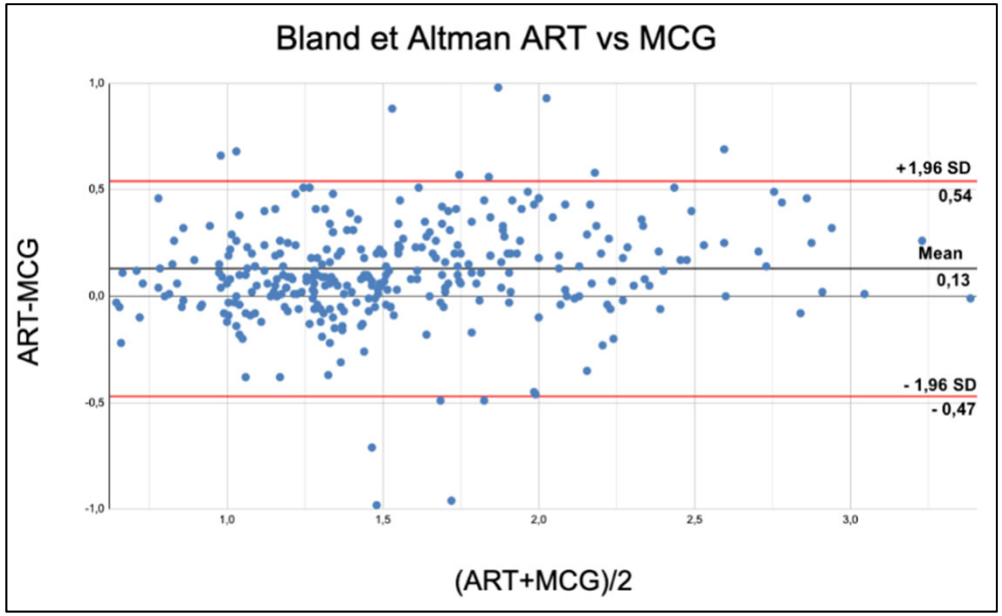
[a] Changes relative to venous plasma measured as central laboratory.



ANESTHÉSIE-RÉANIMATION CARDIOVASCULAIRE

n(323)	Déviation au protocole %	Moyenne déviation d'insuline IVSE (UI/ml)
	43	1,14 +/- 0,99

Fiabilité discutable
Valeur de glycémie interstitielle ≈ glycémie sanguine

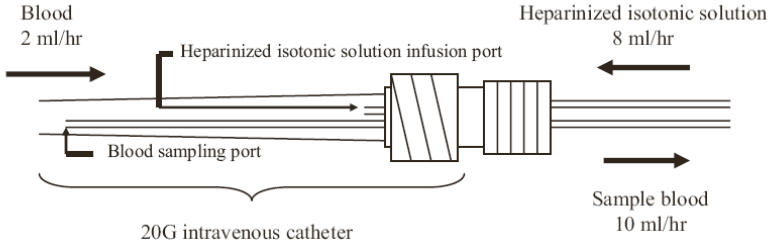


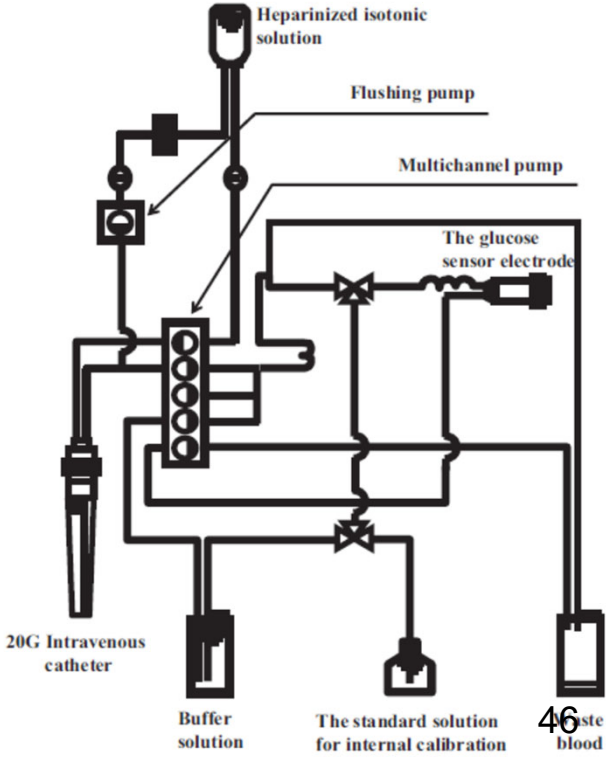
The Accuracy of a Continuous Blood Glucose Monitor During Surgery

Yamashita K et la. Anesth Analg 2008;106:160-3

Cathéter veineux à double lumière (20G)

- Rinçage en continu 8 ml/h (sérum physiologique hépariné)
- Prélèvement 10 ml/h (**2 ml de sang/h +++**)
- Capteur de glucose (glucose-oxydase) 95 µL
- Nouvelle valeur de glycémie toutes les 2 min
- Auto-calibration toutes les 4 heures





Accuracy and reliability of continuous blood glucose monitor in post-surgical patients

Yamashita K et al. Acta Anaesthesiol Scand 2009; 53:66-71

- Evaluation de la précision d'un capteur de monitorage glycémique en continue (STG-22)
  - KT veineux périphérique (20G) avant bras
- Etude observationnelle (n=50)
- Réanimation chirurgicale ***durant 16 heures***

Etude de concordance

	Hypoglycemia (<3.89 (70) mmol (mg/dl))	Normoglycemia (3.89 (70)–10 (180) mmol (mg/dl))
n	11	161
Blood glucose measured by the ABL™ 800FLEX (mg/dl)	0.33(65) ± 0.17(3)	7.39(133) ± 1.56(28)
Bias (mg/dl)	0.04(0.7) (−0.56(−1.5) ~ 0.17(3))	−0.11(−2) (−0.21(−3.7) ~ −0.02(−0.3))
Lower limits of agreement (mg/dl)	−0.33(−6) (−0.56(−10) ~ −0.11(−2))	−1.33(−24) (−1.5(−27) ~ −1.17(−21))
Upper limit of agreement (mg/dl)	0.39(7) (0.19(3.5) ~ 0.61(11))	1.11(20) (0.94(17) ~ 1.28(23))
Percent error for limits of agreement (%)	7	15

Data are expressed as mean ± SD and mean (95% confidence interval).

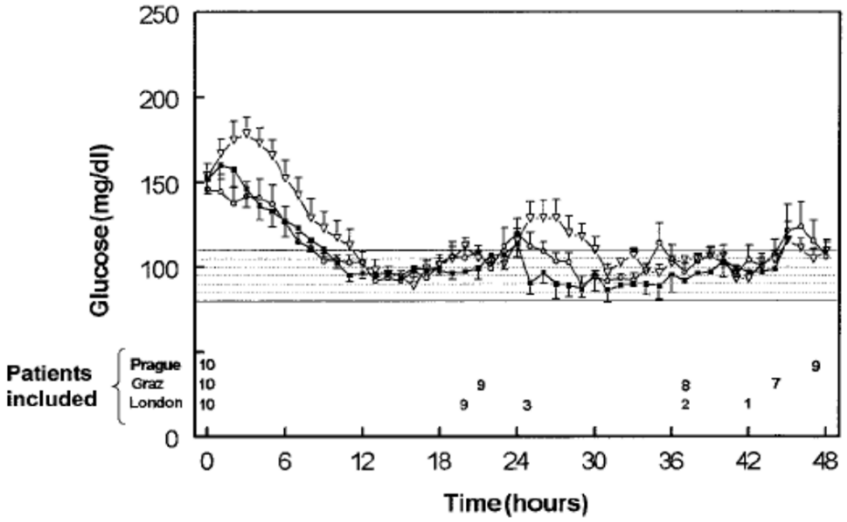
Multicentric, randomized, controlled trial to evaluate blood glucose control by the model predictive control algorithm versus routine glucose management protocols in intensive care unit patients

Plank et al. Diabetes Care 2006; 29:271-6

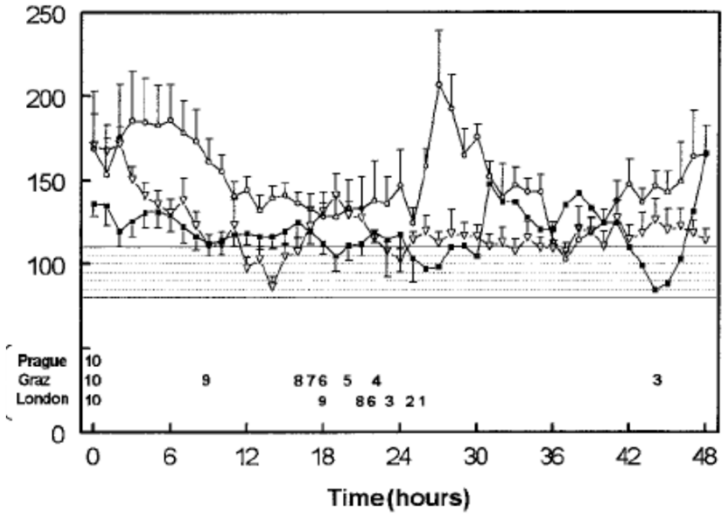
Etude multicentrique

Patients chirurgie cardiothoracique (n=60)

Variables d' entrée (glycémie, débit pompe, apport glucosé)



Avec algorithme informatisé



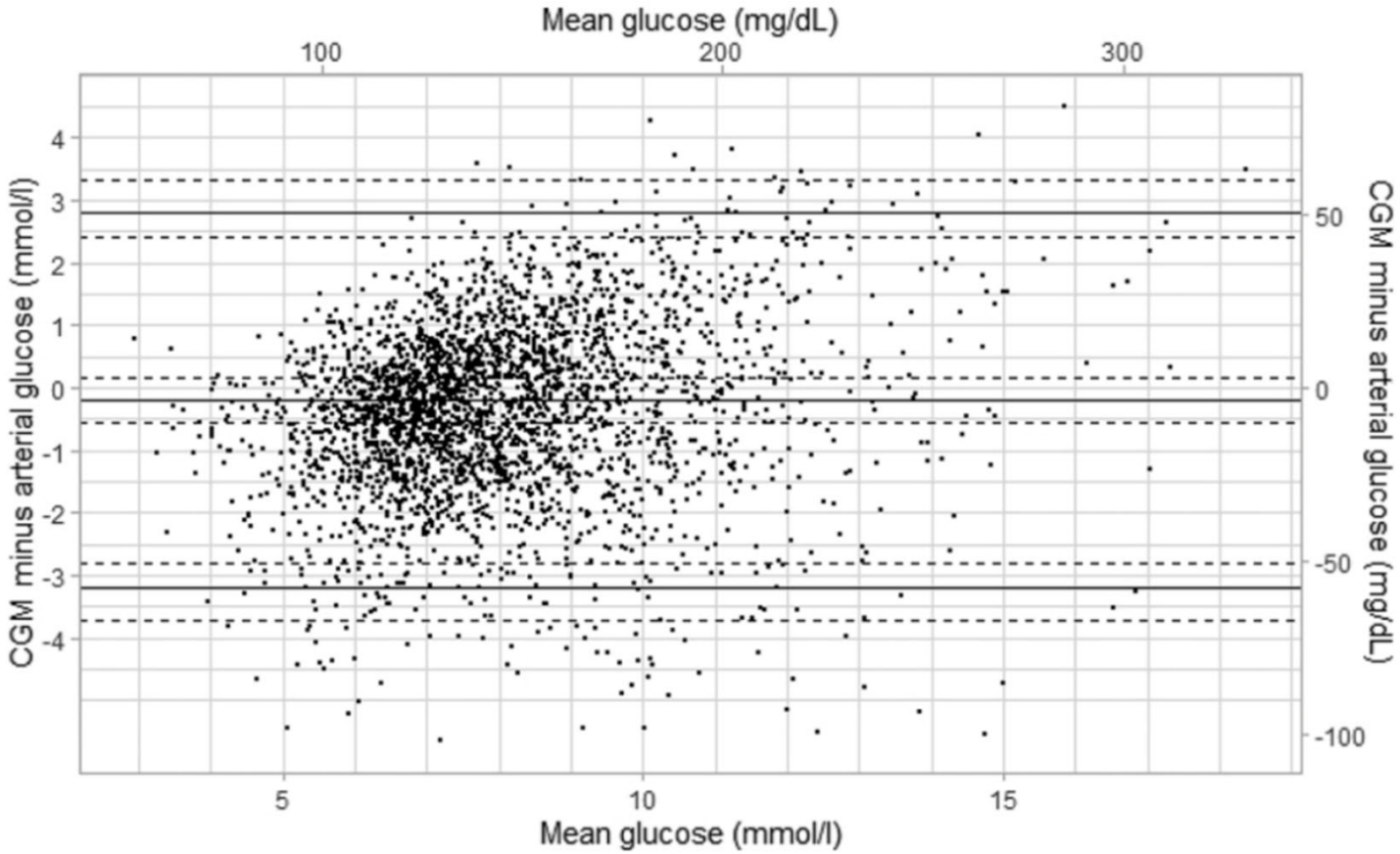
Sans algorithme informatisé

Moindre variabilité de la glycémie ++++

Performance of Subcutaneous Continuous Glucose Monitoring in Adult Critically Ill Patients Receiving Vasopressor Therapy

Diabetes Technol Ther 2024 ; 26 : 763-72

Ola Friman, RN,[1,2] Navid Soltani, MD,[1,2] Marcus Lind, MD, PhD,[3–5] Pia Zetterqvist, RN,[2] Anca Balintescu, MD, PhD,[6] Anders Perner, MD, PhD,[7,8] Anders Oldner, MD, PhD,[1,2] Olav Rooyackers, PhD,[2,9] and Johan Mårtensson, MD, PhD[1,2]



REPORT FROM STS WORKFORCE ON EVIDENCE BASED SURGERY

The Society of Thoracic Surgeons Practice Guideline Series: Blood Glucose Management During Adult Cardiac Surgery

Ann Thorac Surg 2009;87:663-9

Harold L. Lazar, MD, Marie McDonnell, MD, Stuart R. Chipkin, MD, Anthony P. Furnary, MD, Richard M. Engelman, MD, Archana R. Sadhu, MD, Charles R. Bridges, MD, ScD, Constance K. Haan, MD, MS, Rolf Svedjeholm, MD, PhD, Heinrich Taegtmeyer, MD, DPhil, and Richard J. Shemin, MD

- Insulinothérapie intraveineuse
- Relais sous-cutané possible (éducation +++)
- Contrôle glycémique peropératoire si HGT>180 mg.dL^{-1}
- Contrôle glycémique postopératoire si HGT > 150 mg.dL^{-1}

Association Between Perioperative Glycemic Control Strategy and Mortality in Patients With Diabetes Undergoing Cardiac Surgery: A Systematic Review and Meta-Analysis

Méta-analyse d'études randomisées

Relation entre le contrôle glycémique et les complications postopératoires

Patients diabétiques

3 niveaux de contrôle glycémique :

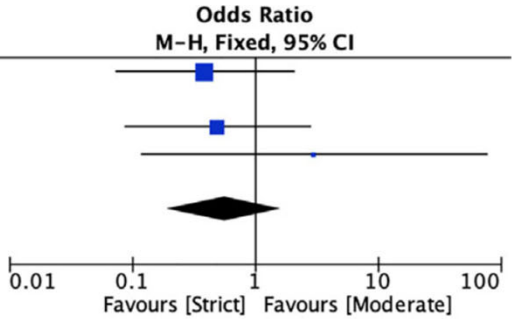
- Libéral > 180 mg/dL
- Modéré 140-180 mg/dL
- Strict < 140 mg/dL

Jin X et al. Front Endocrinol 2020.11:513073

Mortalité postopératoire

Study or Subgroup	Strict Events	Strict Total	Moderate Events	Moderate Total	Weight	Odds Ratio M-H, Fixed, 95% CI
Kirdemir 2008	2	100	5	100	53.0%	0.39 [0.07, 2.05]
Lazar 2011	0	40	0	42		Not estimable
Wahby 2016	2	67	4	68	41.7%	0.49 [0.09, 2.78]
Zadeh 2016	1	38	0	37	5.3%	3.00 [0.12, 76.03]
Total (95% CI)		**245**		**247**	**100.0%**	**0.57 [0.20, 1.66]**
Total events	5		9			

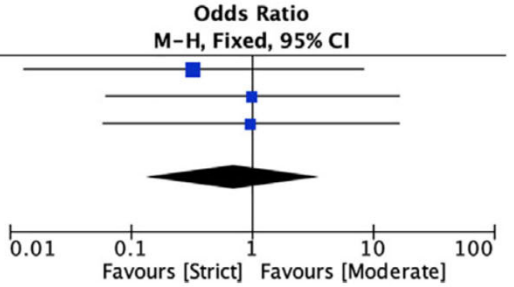
Heterogeneity: $Chi^2 = 1.25$, df = 2 (P = 0.54); $I^2 = 0\%$
Test for overall effect: Z = 1.03 (P = 0.30)



AVC postopératoire

Study or Subgroup	Experimental Events	Experimental Total	Control Events	Control Total	Weight	Odds Ratio M-H, Fixed, 95% CI
Asida 2013	0	50	1	50	42.9%	0.33 [0.01, 8.21]
Kirdemir 2008	1	100	1	100	28.6%	1.00 [0.06, 16.21]
Zadeh 2016	1	38	1	37	28.5%	0.97 [0.06, 16.15]
Total (95% CI)		**188**		**187**	**100.0%**	**0.70 [0.14, 3.62]**
Total events	2		3			

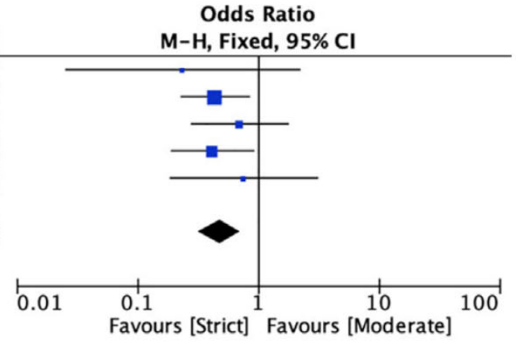
Heterogeneity: $Chi^2 = 0.33$, df = 2 (P = 0.85); $I^2 = 0\%$
Test for overall effect: Z = 0.42 (P = 0.67)



ACFA postopératoire

Study or Subgroup	Experimental Events	Experimental Total	Control Events	Control Total	Weight	Odds Ratio M-H, Fixed, 95% CI
Asida 2013	1	50	4	50	5.8%	0.23 [0.03, 2.18]
Kirdemir 2008	19	100	35	100	41.9%	0.44 [0.23, 0.83]
Lazar 2011	12	40	16	42	16.1%	0.70 [0.28, 1.75]
Wahby 2016	13	67	25	68	29.5%	0.41 [0.19, 0.90]
Zadeh 2016	4	38	5	37	6.7%	0.75 [0.19, 3.06]
Total (95% CI)		**295**		**297**	**100.0%**	**0.48 [0.32, 0.72]**
Total events	49		85			

Heterogeneity: $Chi^2 = 1.65$, df = 4 (P = 0.80); $I^2 = 0\%$
Test for overall effect: Z = 3.51 (P = 0.0004)



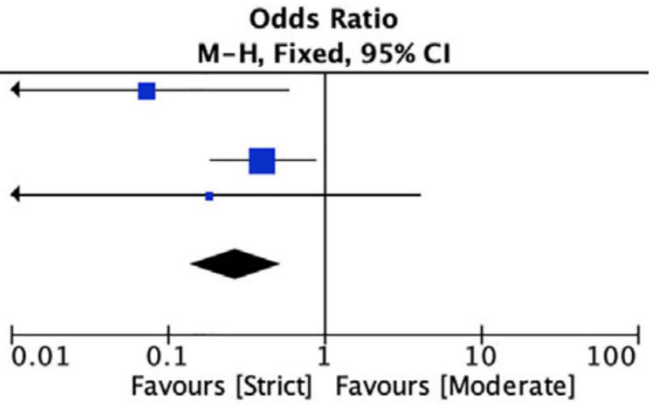
Jin X et al. Front Endocrinol 2020.11:513073

Médiastinite postopératoire

Study or Subgroup	Experimental Events	Experimental Total	Control Events	Control Total	Weight	Odds Ratio M-H, Fixed, 95% CI
Kirdemir 2008	1	100	12	100	33.4%	0.07 [0.01, 0.58]
Lazar 2011	0	40	0	42		Not estimable
Wahby 2016	14	67	27	68	59.6%	0.40 [0.19, 0.86]
Zadeh 2016	0	38	2	37	7.0%	0.18 [0.01, 3.97]
Total (95% CI)		**245**		**247**	**100.0%**	**0.28 [0.14, 0.54]**
Total events	15		41			

Heterogeneity: $Chi^2 = 2.55$, $df = 2$ ($P = 0.28$); $I^2 = 22\%$

Test for overall effect: $Z = 3.74$ ($P = 0.0002$)



Jin X et al. Front Endocrinol 2020.11:513073

Protocole d'insulinothérapie IVSE EN REANIMATION DES CARDIOPATHIES ACQUISES

Objectifs glycémiques postopératoires : 5 – 8 mmol/L (0,9 – 1,5 G/L)

Modalités d'administration :

- Dilution : analogue ultra rapide de l'insuline I UI/mL dans NaCl 0,9 %
- Voie d'abord : robinet au plus proche du patient sur KTC ou sur VVP
- **Chez un patient diabétique insulino-dépendant, ne jamais arrêter insuline sans avis du MAR**

Version du 17/12/2020
Référents : J. Rey, N. Benillan, B. Bourdarias, A. Ouattara

Glycémie		0,4 g/L 2,2 mmol/L		0,6 3,3	0,9 5	1,1 6	1,5 8	1,8 10	2,5 14	3 16,5
Initiation insuline IVSE	Bolus IVD	0	0	0	0	0	0	3 UI	4 UI	6 UI
	Débit IVSE	0	0	0	1 UI/h si DT1 (Stop si DT2 ou non diabétique connu)		1 UI/h	2 UI/h	3 UI/h	4 UI/h Prévenir médecin
Fréquence des glycémies		15 min	30 min	1h	1h	2h	1h	1h	1h	1h
Adaptation du débit d'insuline IVSE		Arrêt	Arrêt	- 1 UI/h	- 1 UI/h	Idem	+ 1 UI/h	+ 2 UI/h	+ 3 UI/h	Bolus 6 UI Prévenir médecin
		Reprise à ½ débit quand : Glyc > 5,5 mmol/L chez DT1 Glyc > 10 mmol/L chez DT2								
G30 %		2 amp (6 g) Prévenir med	1 amp (3 g)	0						

Remarques :

Privilégier les glycémies capillaires sauf pour des valeurs dans les zones « rouges » (<0,6 ou > 2,5 g/L) qui doivent faire l'objet d'un contrôle de la valeur sur gaz du sang
Lorsqu'une nutrition ou alimentation entérale ou parentérale est initiée ou arrêtée, réaliser une glycémie 1H après (pensez à bien remplir la feuille des apports).
Surveillance du dispositif d'administration : PSE et voie d'abord!! (tubulures/robinet/VVC/VVP/VALVE ANTIRETOUR)
En raison d'interaction médicamenteuses, changer la seringue d'insuline toutes les 12 heures
Surveillance état de conscience si glycémie en zone rouge
Pas de sortie du patient dans les étages sous insuline IVSE. Sur avis médical, relais SC ou arrêt de la seringue. Modalités de relais sous-cutané (cf verso).

Protocole d'insulinothérapie IVSE PEROPERATOIRE DES CARDIOPATHIES ACQUISES

Objectifs glycémiques peropératoires : 5 – 10 mmol/L (0,9 – 1,8 G/L)
Modalités d'administration :
- Dilution : analogue ultra rapide de l'insuline I UI/mL dans NaCl 0,9 %
- Voie d'abord : robinet au plus proche du patient sur KTC ou sur VVP

Version du 16/04/2021
Référents : J. Rey, N. Benillan, B. Bourdarias, A. Ouattara

Glycémie		0,4 g/L 2,2 mmol/L	0,6 3,3	1,0 5,5	1,8 10	2,5 14	3 16,5	
Initiation insuline IVSE	Bolus IVD	0	0	0	0	3 UI	4 UI	6 UI
	Débit IVSE	0	0	0	Discuter 1 UI/h pour les DT1	2 UI/h	3 UI/h	4 UI/h Prévenir médecin
Fréquence des glycémies		15 min	15 min	/30 min avec GDS				
Adaptation du débit d'insuline IVSE		Arrêt	Arrêt	- 1 UI/h	Idem	+ 1 UI/h	+ 2 UI/h	Bolus 6 UI + 3UI/h
		Reprise à ½ débit quand : Glyc > 10 mmol/L						
G30 %		2 amp (6 g) Prévenir med	1 amp (3 g)	0				

PASSAGE INSULINE IVSE / INSULINE SC

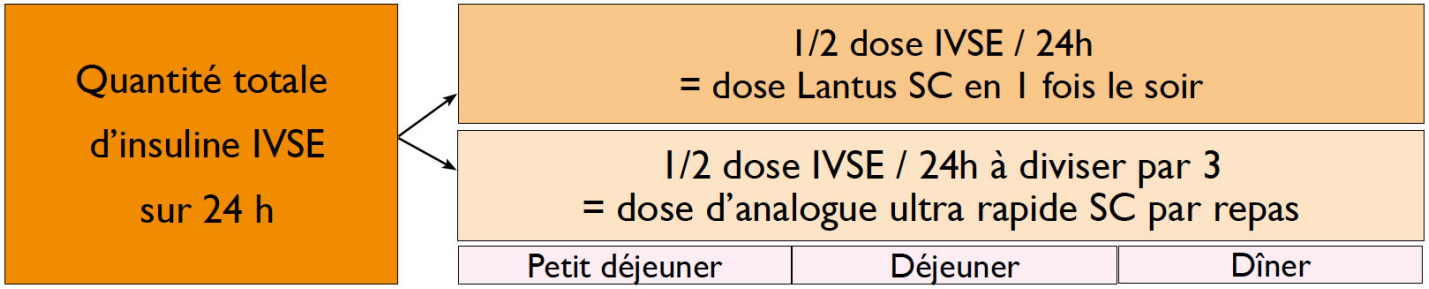
Indications

- Dès que les glycémies sont stables et ≤ 10 mmol/l
- A la reprise d'une alimentation orale
- Relais à l'arrêt de la seringue électrique d'insuline

Contre-indications

- Posologie d'insuline IVSE ≤ 0,5 UI/h
- Posologie d'insuline IVSE ≥ 5 UI/h
- Déséquilibre glycémique

Calcul de dose



Le délai entre l'arrêt de l'insuline IVS et l'injection de Lantus SC doit être IMMÉDIAT en préférant une injection à 20 h le soir. Si non faire une injection complémentaire selon le schéma suivant

Arrêt insuline IVSE	Entre 0 h et 6 h	Entre 6 h à 14 h	Entre 14 h et 18 h	Entre 18 h et 0 h
Dose de Lantus	3/4 dose	1/2 dose	1/4 dose	dose de 20 h
Dose de Lantus suivante	à 20 h le soir même			à 20 h le jour suivant

PAS D'INSULINE IVSE EN SALLE

Prise en charge pré opératoire (DT1 et DT2)

Règles de jeûne :

- Repas du soir normal
- Donner le traitement habituel : AD non insuliniques (sauf metformine) mais aussi insulines aux mêmes posologies
- Dernier repas solide à H-6 et liquides clairs autorisés jusqu'à H-2 de la chirurgie
- Mettre une VVP et perfuser avec soluté glucosé (G10% 40 mL/h) le matin à partir de 6-7h si et seulement si le patient a eu une insuline lente la veille au soir ou s'il a une pompe à insuline

Modalités d'arrêt des traitements antidiabétiques (AD) :

	Chirurgie ambulatoire	Chirurgie mineure ou majeure	Chirurgie urgente
Metformine	Pas d'arrêt	Pas de prise la veille au soir et le matin	Arrêt
Sulfamides	Pas d'arrêt	Pas de prise le matin	Arrêt
Glinides			
Inhibiteurs -glucosidases			
Inhibiteurs DDP-4			
Analogues GLP-1	Pas d'arrêt	Pas d'injection le matin	Arrêt
Insulines SC	Pas d'arrêt	Pas d'injection le matin (sauf dans le DT1)	Arrêt et relais
Pompe à insuline	Pas d'arrêt	Arrêt de la pompe à l'arrivée au bloc	Arrêt et relais

Période pré opératoire :

- Faire une glycémie capillaire avant le repas du soir, au coucher et à 6h et appliquer selon le protocole suivant :

<table>
<tr><td>Glycémie capillaire (GC)</td><td colspan="4">0,6 — 0,9 — 1,8 — 2,2 — 3 g/L
3,3 — 5 — 10 — 12 — 16,5 mmol/L</td></tr>
<tr><td>Avant le repas du soir</td><td>Sucre
15g PO
Prévenir le médecin</td><td></td><td>Insuline : analogue ultra rapide
3 UI SC | 4 UI SC
si correction non faite par le patient</td><td>6 UI SC + BU
Prévenir le médecin</td></tr>
<tr><td>19-20h</td><td colspan="4">Repas normal +/- insulines habituelles +/- AD non insuliniques sauf metformine</td></tr>
<tr><td>Au coucher 22h-0h
Si besoin 3h-4h</td><td>15g PO
GC à 15 min
Prévenir le médecin</td><td></td><td>3 UI SC | 4 UI SC</td><td>6 UI SC + BU
ou IVSE en réa
Prévenir le médecin</td></tr>
<tr><td>6h-7h</td><td colspan="3">Pas de prise d'AD non insuliniques
et G10% 40 ml/h si insuline lente la veille ou pompe à insuline en cours</td><td>VVP
NaCl 0,9%</td></tr>
<tr><td>Pré-op GC/3h</td><td>G10% 60 ml/h
Prévenir le médecin</td><td></td><td>3 UI SC | 4 UI SC</td><td>IVSE en réa
Différer le bloc</td></tr>
</table>

ATTENTION :

- Chez le patient DT1 et quelle que soit la glycémie :
NE JAMAIS ARRÊTER L'INSULINE LENTE

- Si hypoglycémie : Cf fiche
CAT devant une hypoglycémie à l'hôpital

- Si hyperglycémie : Cf fiche
CAT devant une hyperglycémie à l'hôpital

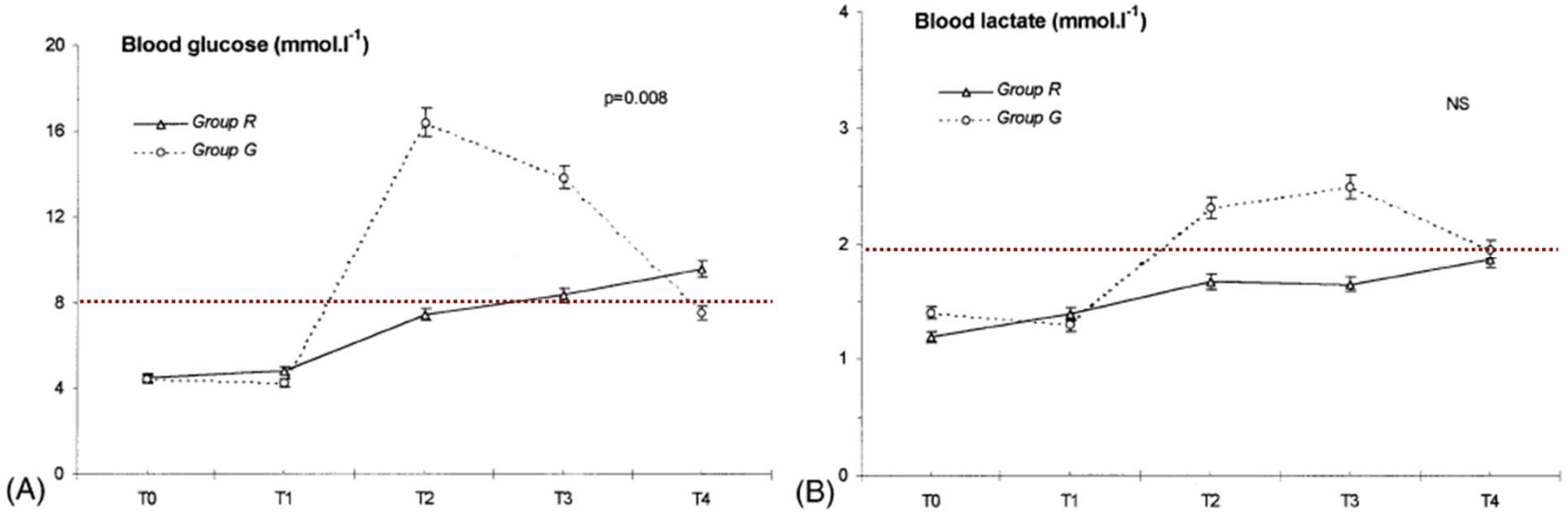
Groupe SFAR/SFD

Effects of Intraoperative Glucose Administration on Circulating Metabolites and Nitrogen Balance During Prolonged Surgery

Chambrier C et al. J Clin Anesth 1999; 11:646-51

Chirurgie abdominale ou thoracique ≥ 3 heures (ASA I-II)

Ringer lactate vs SG5% (10 ml.kg^{-1}.h^{-1} jusque H_2 postopératoire)



Si pas de glucose: pas d' hypoglycémie

Si apport de glucose= risque HG donc effets potentiellement délètéres

« …suggestions that glucose solutions should be avoided in both long and short surgical procedures. »

Conclusion

- Hyperglycémie périopératoire délétère chez patients diabétiques ou pas de chirurgie cardiaque
- Contrôle glycémique améliore leur «outcome »
- Insulinothérapie IV et relais sous-cutané
- Valeur seuil Peropératoire (<180 mg/dL)

  Réanimation (<150 mg/dL)