ROYAUME DU MAROC
UNIVERSITE CADI AYYAD
FACULTE DE MEDECINE ET DE PHARMACIE
MARRAKECH

ROYAUME DU MAROC - MINISTERE DE LA SANTÉ

المركز الإستشفائي الجامعي
محمد السادس
مراكش

NOS VALEURS :
QUALITÉ - RESPECT - ETHIQUE - HUMANISATION
EXCELLENCE - OUVERTURE - INTÉGRITÉ

Hôpital Mère enfant ***مستشفى الأم والطفل***

Insuffisance hépatique aigue : Principes de réanimation

S. YOUNOUS: Anesthésie réanimation
Chef de Service d' Anesthésie-Réanimation
Hôpital Mère-Enfant- CHU Mohammed VI Marrakech

SOMIPEV 25-27
Mars 2016

Définitions

-**Insuffisance hépato-cellulaire sévère:**

Facteur V ou taux de prothrombine <50%

-**IHAG**: + encéphalopathie hépatique (EH).

-**Les IHAG fulminantes**:

signes neurologiques dans les 8 semaines qui suivent le début de l'atteinte hépatique, en l'absence d'une hépatopathie préexistante.

Groupe pédiatrique du King's collège: IHAG ou fulminante et jeune nourrisson

« une **maladie multiviscérale** au cours de la quelle l'insuffisance hépato-cellulaire **compliquée ou non d'EH**, s'associe à une nécrose hépatique chez un enfant sans maladie hépatique reconnue préalablement ».

Epidémiologie

Journal of Pediatric Gastroenterology and Nutrition
40:575–581 © May 2005 Lippincott Williams & Wilkins, Philadelphia

Etiology, Outcome and Prognostic Indicators of Childhood Fulminant Hepatic Failure in the United Kingdom

*Way Seah Lee, †Patrick McKiernan, and †Deirdre Anne Kelly

**Department of Paediatrics, University of Malaya Medical Centre, Kuala Lumpur, Malaysia; and †Liver Unit, Birmingham Children's Hospital, Birmingham, United Kingdom*

Kingdom.
Design: Retrospective review of all patients <17 years with fulminant hepatic failure from 1991 to 2000. Fulminant hepatic failure was defined as presence of coagulopathy (prothrombin time >24 seconds or International Normalized Ratio >2.0) with or without hepatic encephalopathy within 8 weeks of the onset of symptoms.

TABLE 3. *Outcome of 97 children with fulminant hepatic failure in the United Kingdom, according to underlying cause*

	Total (n = 97)	Survived without LT (n = 32)	LT, alive (n = 27)	LT, died (n = 13)	Died, no LT (n = 25)
Metabolic (n = 22)					
Neonatal hamochromatosis	7	3	2	1	1
Mitochondrial disorders	4	–	–	–	4
Tyrosinemia	2	2	–	–	–
Wilson's Disease	2	–	2	–	–
Other metabolic disorders	7	2	–	–	5
Infective (n = 53)					
Hepatitis B	2	–	–	1	1
Hepatitis A	9	4	3	–	2
nAnBnC hepatitis	36	8	16	6	6
Other infections	6	2	2	–	2
Drugs (n = 19)					
Paracetamol overdose	14	7	1	4	2
Drug-induced hepatotoxicity	5	2	1	1	1
Autoimmune hepatitis	3	2	–	–	1

LT, liver transplantation.

INDIAN PEDIATRICS VOL 50; JULY 15, 2013

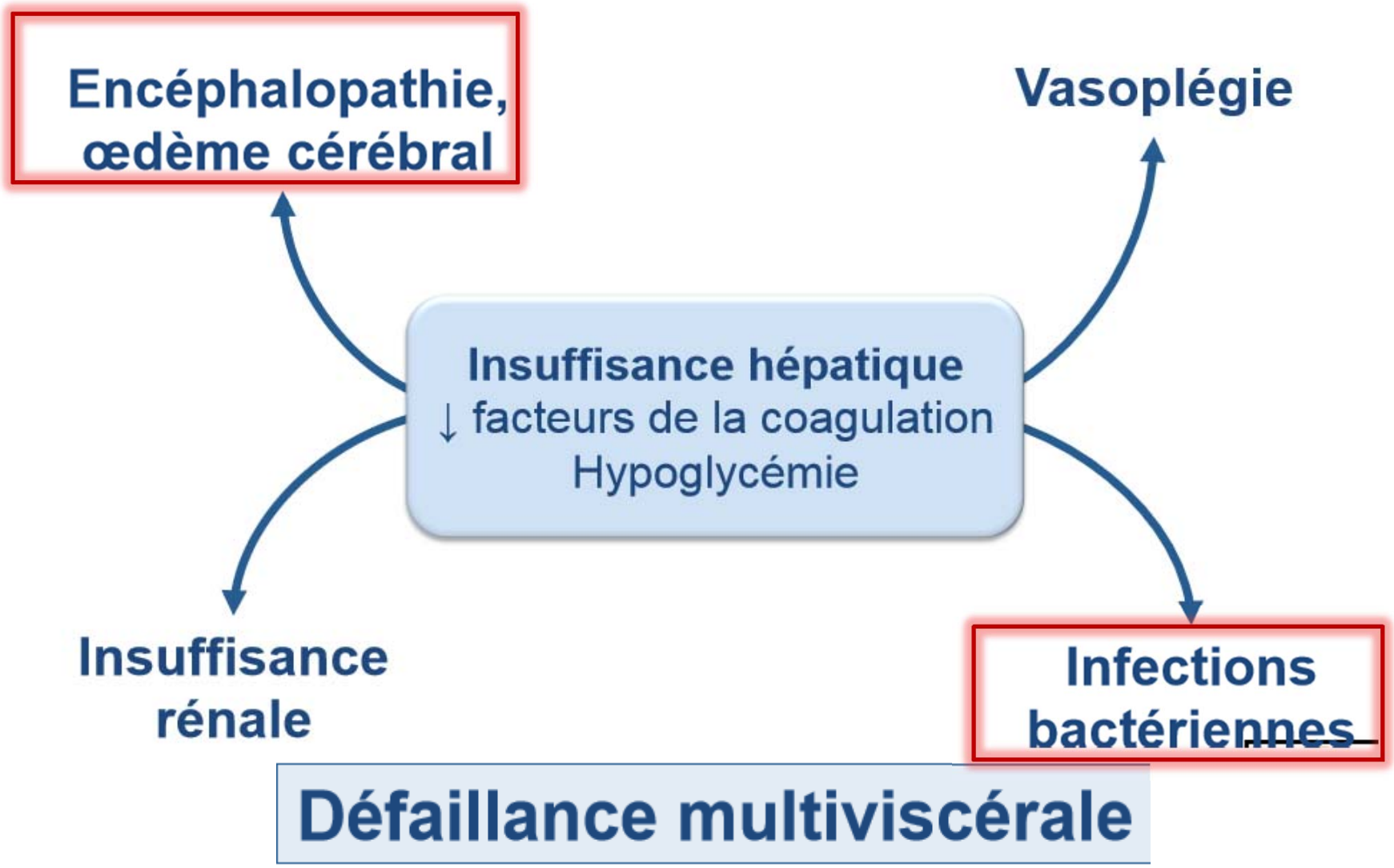
TABLE II ETIOLOGY AND ITS RELATION TO MORTALITY

Etiology	*Number N=43*	*Group A (death) N=19*	*Group B (discharged) N=24*
HAV	25 (58%)	12 (48%)	13 (52%)
HBV	2 (4.6%)	0	2 (100%)
HCV	0	0	0
HEV	2 (4.6%)	2 (100%)	0
Other infections	2 (4.6%)	0	2 (100%)
HAV plus HEV	2 (4.6%)	0	2 (100%)
Hemochromatosis	1 (2.3%)	1 (100%)	0
Galactosemia	2 (4.6%)	2 (100%)	0
Wilson's disease	2 (4.6%)	0	2 (100%)
Autoimmune hepatitis	1 (2.3%)	1 (100%)	0
Indeterminate	4 (9.2%)	1 (25%)	3 (75%)

Table 1 Outcome in 13 patients with acute liver failure caused by hepatitis A virus

Outcome	*n*	%
Spontaneous recovery	1	7.7
Death before transplantation	5	38.4
Liver transplantation	7	53.9
Alive	3	23.1
Death	4	30.8
Overall survival	4	30.8

Physiopathologie



Objectifs de la réanimation

1-Reconnaître la cause et évaluer le pronostic

2- Eviter les facteurs aggravants

3-Traiter les défaillances

4-Savoir poser l'indication de transplantation hépatique en urgence

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Pediatr Transplantation 2013: 17: 362–368

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Pediatric Transplantation
DOI: 10.1111/petr.12083

Pediatric acute liver failure: Etiology, outcomes, and the role of serial pediatric end-stage liver disease scores

Rajanayagam J, Coman D, Cartwright D, Lewindon PJ. Pediatric acute liver failure: Etiology, outcomes, and the role of serial PELD scores.

Abstract: To describe etiology, short-term outcomes and prognostic accuracy of serial PELD scores in PALF. Retrospective analysis of children aged ≤16 yr, admitted with PALF under the QLTS, Brisbane, Australia, between 1991 and 2011. PELD-MELD scores were ascertained at three time points (i) admission (ii), meeting PALF criteria, and (iii) peak value. Fifty-four children met criteria for PALF, median age 17 months (1 day–15.6 yr) and median weight 10.2 kg (1.9–57 kg). Etiology was known in 69%: 26% metabolic, 15% infective, 13% drug-induced, 6% autoimmune, and 9% hemophagocytic lymphohistiocytosis. Age <3 months and weight <4.7 kg predicted poor survival in non-transplanted children. Significant independent predictors of poor outcome (death or LT) were peak bilirubin > 220 μM/L and peak INR > 4. Serial PELD-MELD scores were higher in the 17 (32%) transplant recipients (mean: [i] 26.8, [ii] 31.8, [iii] 42.6); highest in the 12 (22%) non-transplanted non-survivors (mean: [i] 31.6, [ii] 37.2, [iii] 45.7) compared with the 25 (46%) transplant-free survivors (mean: [i] 25.3, [ii] 26.0, [iii] 30.3). PELD-MELD thresholds of ≥27 and ≥42 at (ii) meeting PALF criteria and (iii) peak predicted poor outcome ($p < 0.001$). High peak bilirubin and peak INR predict poor outcome and serial PELD-MELD is superior to single admission PELD-MELD score for predicting poor outcome.

Jeremy Rajanayagam[1,2], David Coman[2,3], David Cartwright[2,4] and Peter J. Lewindon[1,2]

[1]Department of Pediatric Gastroenterology, Royal Children's Hospital, Herston, Brisbane, Qld, Australia, [2]School of Medicine, University of Queensland, Qld, Australia, [3]Grantley Stable Neonatal Unit, Royal Brisbane and Women's Hospital, Brisbane, Qld, Australia, [4]Department of Metabolic Medicine, Royal Children's Hospital, Herston, Brisbane, Qld, Australia

Key words: pediatric acute liver failure – pediatric end-stage liver disease

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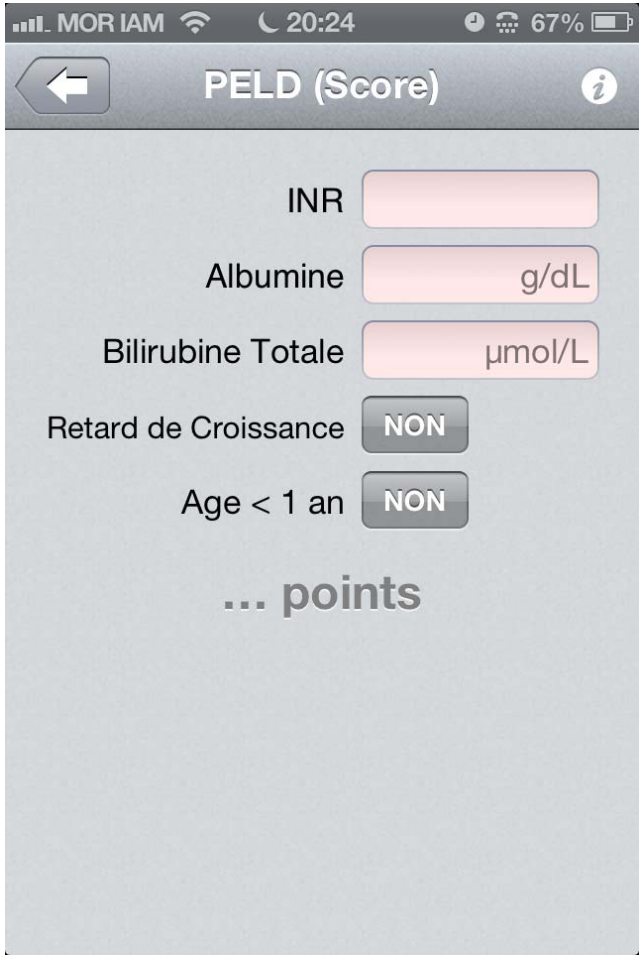
Accepted for publication 13 March 2013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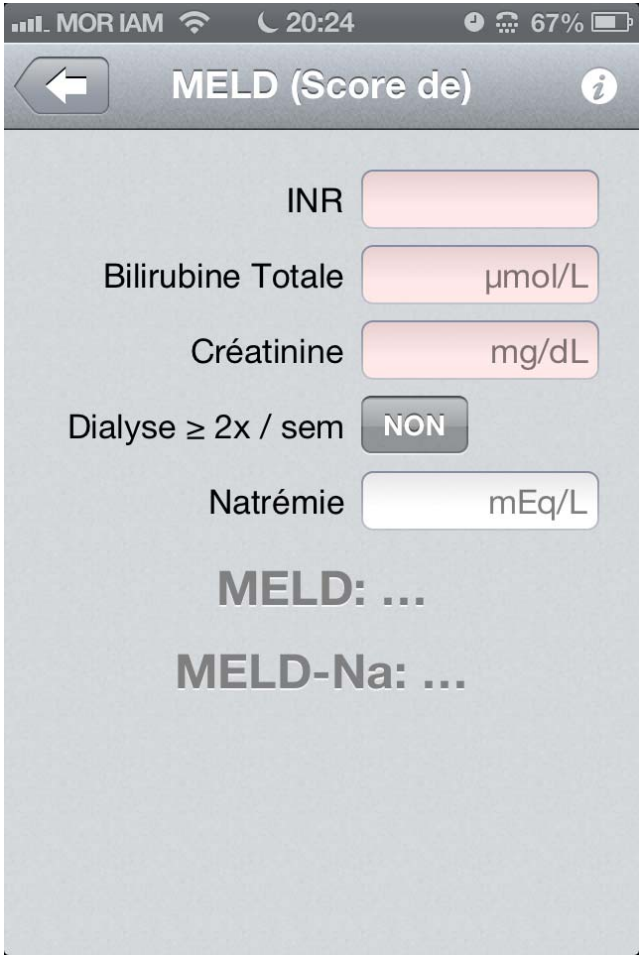
20:24
67%
PELD (Score)
INR
Albumine
g/dL
Bilirubine Totale
µmol/L
Retard de Croissance
NON
Age < 1 an
NON
... points


MOR IAM
20:24
67%
MELD (Score de)
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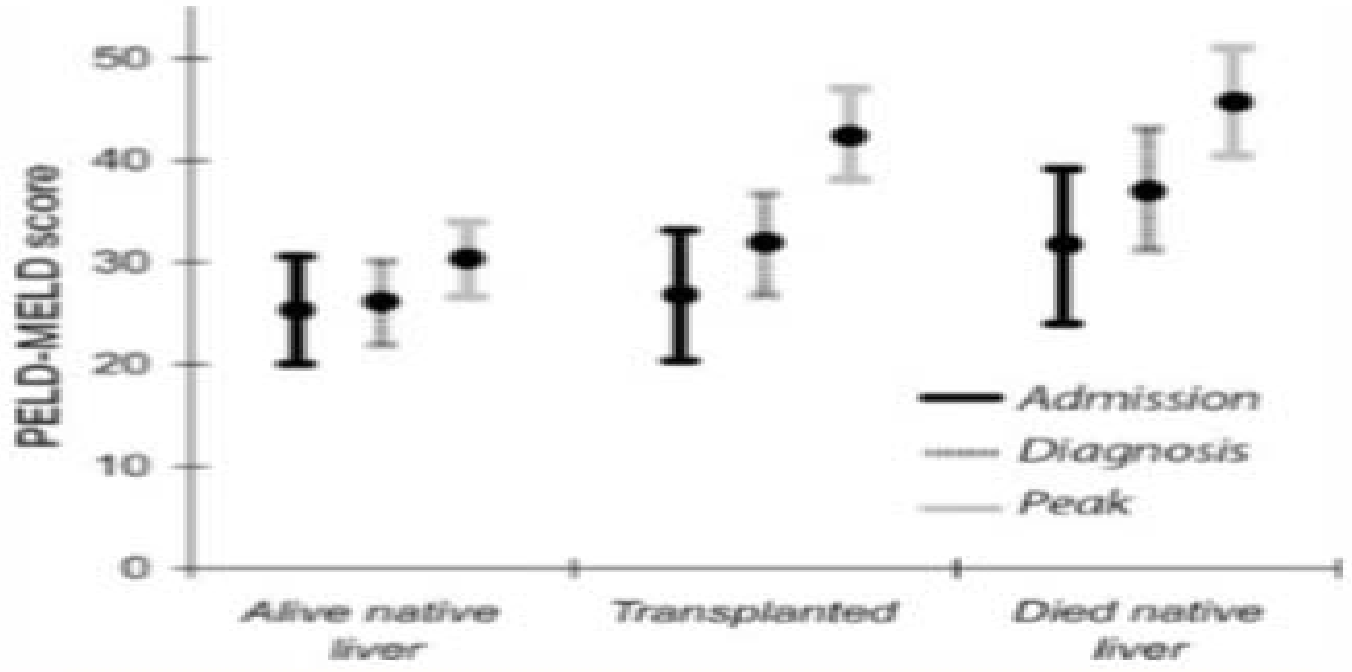

Fig. 2. **PELD-MELD scores according to outcome at different time points in PALF (a) admission, (b) diagnosis with PALF criteria, (c) peak values.**

Objectifs de la réanimation

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4-Savoir poser l'indication de transplantation hépatique en urgence

Le Foie	**Hépatotoxiques (Paracétamol)**
Le Rein	**Néphrotoxiques (AINS, aminosides, produits de contraste iodés), Hypovolémie**
Le Cerveau	Sédatifs (primpéran, BZD,…) Hypoglycémie Hyponatrémie +++
Infections	**Antibiothérapie probabiliste ?**
Hémorragies	**Traitement par IPP** **Pas de PFC systématiques, plaquettes si thrombopénie** **Pas d' IM**

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Défaillance hépatique

- Pas de traitement favorisant directement la régénération hépatique

- **Intérêt de la NAC +++**

- Pas de transfusion de PFC systématique

- **Place de la dialyse à l'albumine (MARS) ?**

Intravenous N-acetylcysteine improves transplant-free survival in early stage non-acetaminophen acute liver failure
Lee WM et al Gastroenterology. 2009 Sep;137(3):856-64

-Etude prospective ,randomisée , en double aveugle

-multicentrique 24 centres US , entre 1998 et 2006

-173 patients présentant une HF non liée au paracétamol .

Table 1. Study Medication

Application of *N*-acetylcysteine		
	dose	duration
1.	150 mg/kg/h	1 hour
2.	12.5 mg/kg/h	4 hours
3.	6.25 mg/kg/h	67 hours

	NAC (n= 81)	Placebo (n= 92)	
Survie 3 sem	70%	66%	NS
Survie sans TH	40%	27%	P 0,043
Taux de TH	32%	45%	P 0,09 (NS)

Analyse sous groupes : survie à 3 semaines

	NAC	placébo	
Encéphalopathie I et II (n = 114)	52 %	30%	P 0,01
Encéphalopathie III et IV (n=	9 %	22%	

NAC improves transplant-free survival in patients with early stage non-acetaminophen-related acute liver failure. Patients with advanced coma grades do not benefit from NAC and typically require emergency liver transplantation.

Safety and efficacy of N-acetylcysteine in children with non-acetaminophen-induced acute liver failure.

Kortsalioudaki C[1], Taylor RM, Cheeseman P, Bansal S, Mieli-Vergani G, Dhawan A.

Liver Transpl. 2008 Jan;14 :25-30

	G 1 Placebo: 89-94	G2 NAC: 95-04	
-Admission to intensive care	41 (71%)	85 (77%)	NS
-length of intensive care stay	6 (1-58 days)	5 (1-68 days)	NS
-length of hospital stay	25 (1-264 days)	19 (1-201 days)	0,05
-Survival with native liver	13 (22%)	48 (43%)	0,005
-Died without transplant	15 (25%)	21 (19%)	NS
-LT	32 (54%)	42 (38%)	NS
-Death after transplantation	15 (39%)	8 (16%)	0,02

Should N-acetylcysteine be used in treatment of non-acetaminophen pediatric acute liver failure?

Kumar V[1]. Indian Pediatr. 2013 Oct;50(10):972-3

-The writing committee stated that there is increasing evidence for use of NAC infusion in non-acetaminophen causes of ALF

-They recommended routine use ofNAC in the dose of 100 mg/kg/day in all cases of ALF irrespective of the etiology.

-Associated with:

- a shorter length of hospital stay,

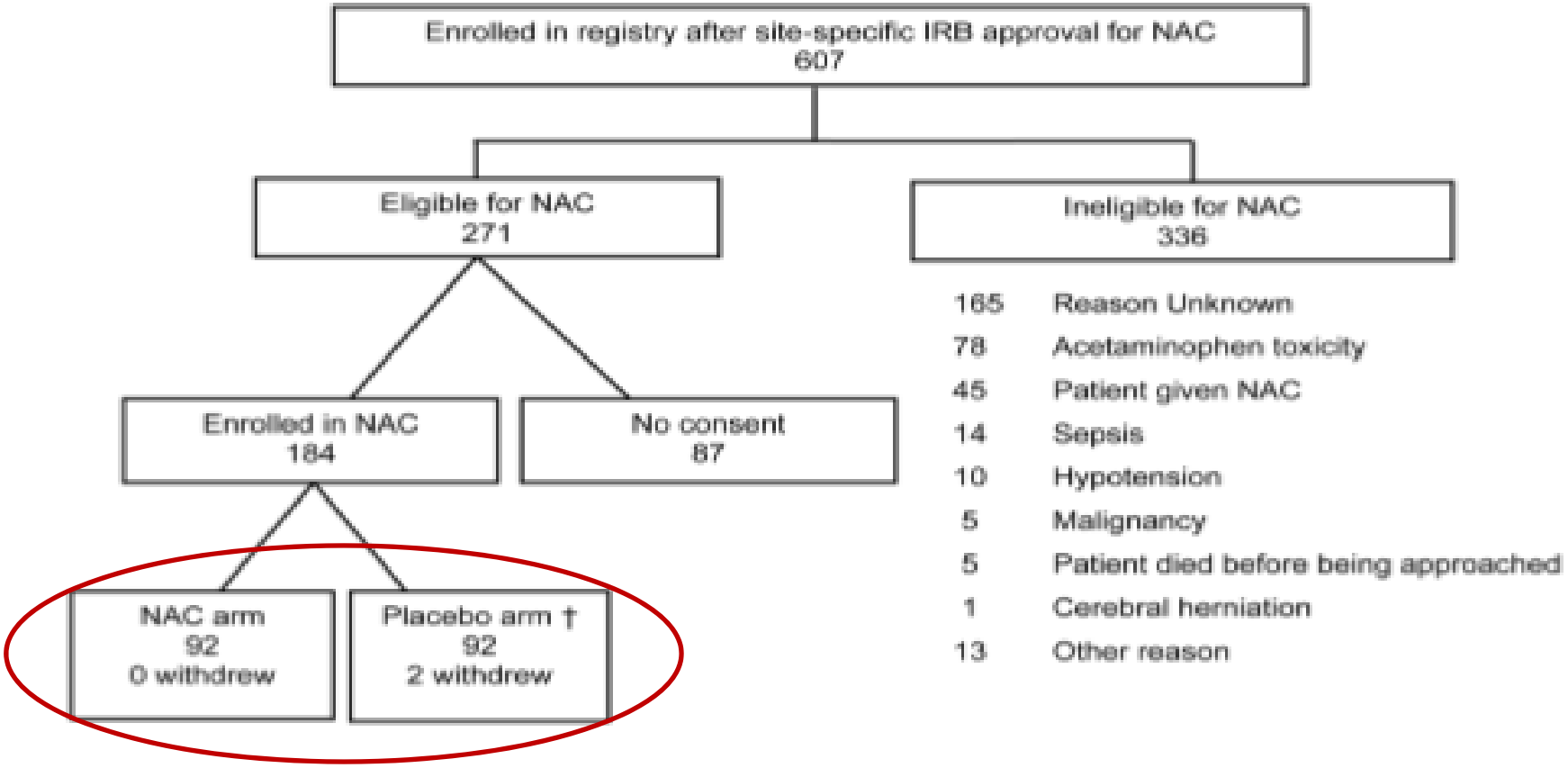
-higher incidence of native liver recovery without transplantation,

-better survival after transplantation.

Intravenous N-acetylcysteine in Pediatric Patients with NonAcetaminophen Acute Liver Failure: A Placebo-Controlled Clinical Trial

Robert H.

Hepatology . 2013 April ; 57(4): 1542–1549



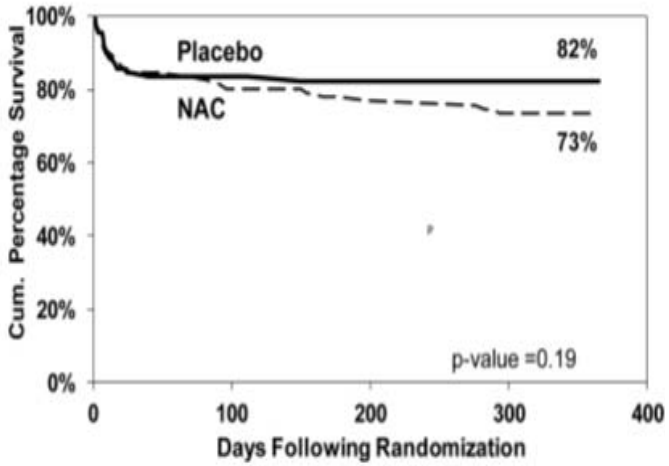


Figure 2. Primary outcome: 1 year survival
Product-limit estimates were used to obtain the cumulative percentages of participant surviving 1 year following randomization. A log-rank test was used to assess statistic significance of the difference in survival curves. The cumulative percentage of childr were alive 1 year following randomization to NAC (dashed line) or placebo (solid lir depicted. The percent surviving 1 year was higher in patients receiving placebo at 82 NAC at 73%, but the differences were not significant with a p-value of 0.19.

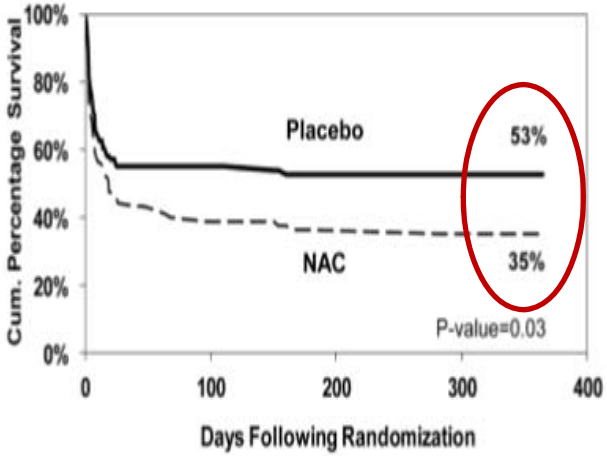
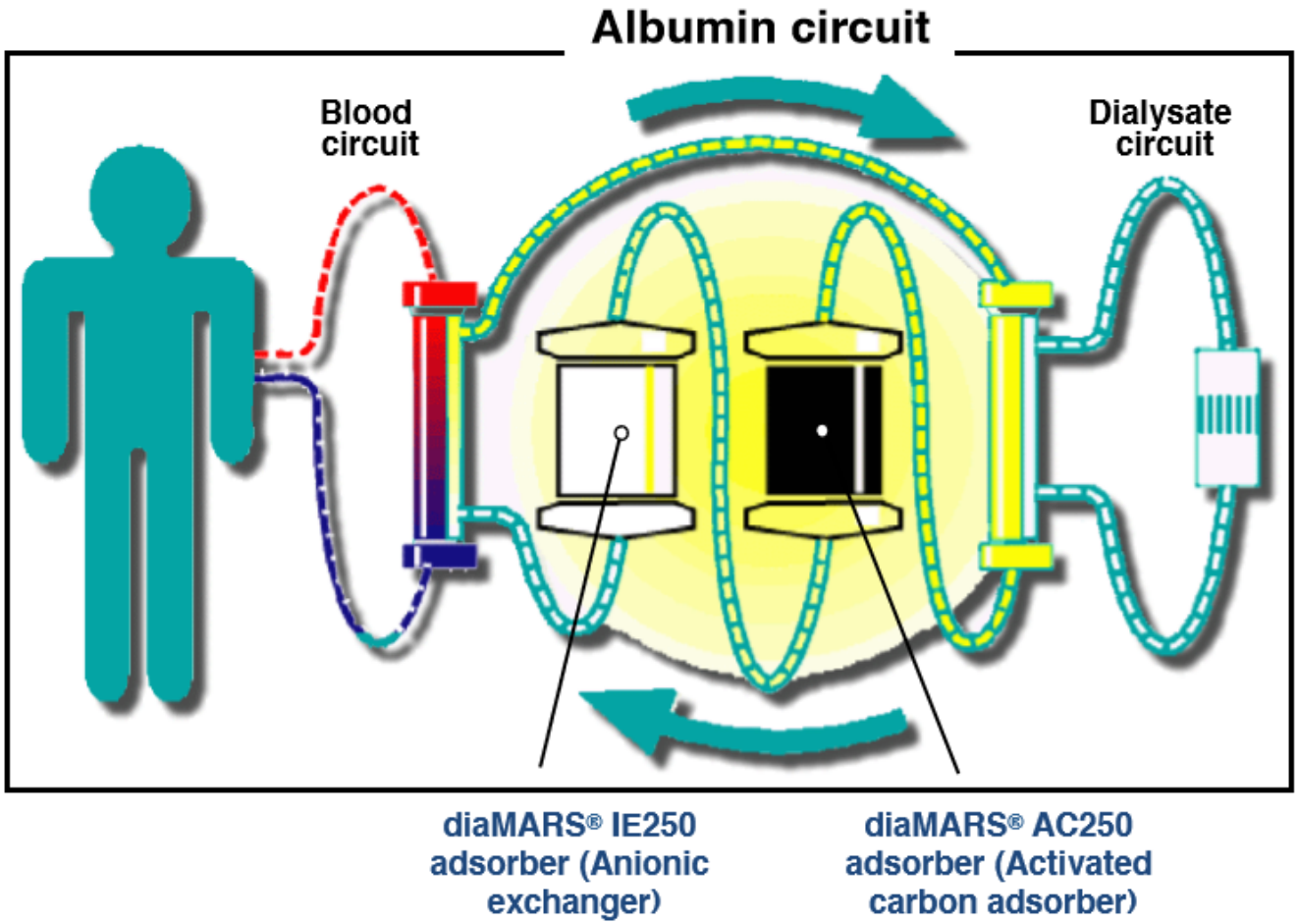


Figure 3.
Product-limit estimates were used to obtain the cumulative percentages of participants with 1 year transplantation free survival. A log-rank test was used to assess statistical significance of the difference in survival curves. The cumulative percentage of children with liver transplantation-free survival 1 year following randomization to NAC (dashed line) or placebo (solid line) is depicted. The cumulative percentage of patients with liver transplantation-free survival was 53% when given placebo vs 35% when given NAC, with a p-value of 0.03.

Conclusion—NAC did not improve 1-year survival in non-APAP PALF. 1-year LTx-free survival was significantly lower with NAC, particularly among those < 2 years old. These results do not support broad use of NAC in non-APAP PALF and emphasizes the importance of conducting controlled pediatric drug trials, regardless of results in adults.

Molecular Adsorbent Recirculating System:MARS®



LIVER TRANSPLANTATION 21:369–380, 2015

ORIGINAL ARTICLE

Experience With Molecular Adsorbent Recirculating System Treatment in 20 Children Listed for High-Urgency Liver Transplantation

Willem S. Lexmond,[1] Carin M. L. Van Dael,[1] René Scheenstra,[2] Joanne F. Goorhuis,[3] Egbert Sieders,[4] Henkjan J. Verkade,[2] Patrick F. Van Rheenen,[2] and Martin Komhoff[1]

[1]Division of Pediatric Nephrology, Beatrix Children's Hospital, [2]Division of Pediatric Gastroenterology and Hepatology, Beatrix Children's Hospital, [3]Division of Pediatric Intensive Care, Beatrix Children's Hospital, and [4]Division of Hepatobiliary Surgery and Liver Transplantation, Department of Surgery, University Medical Center Groningen, Groningen, the Netherlands

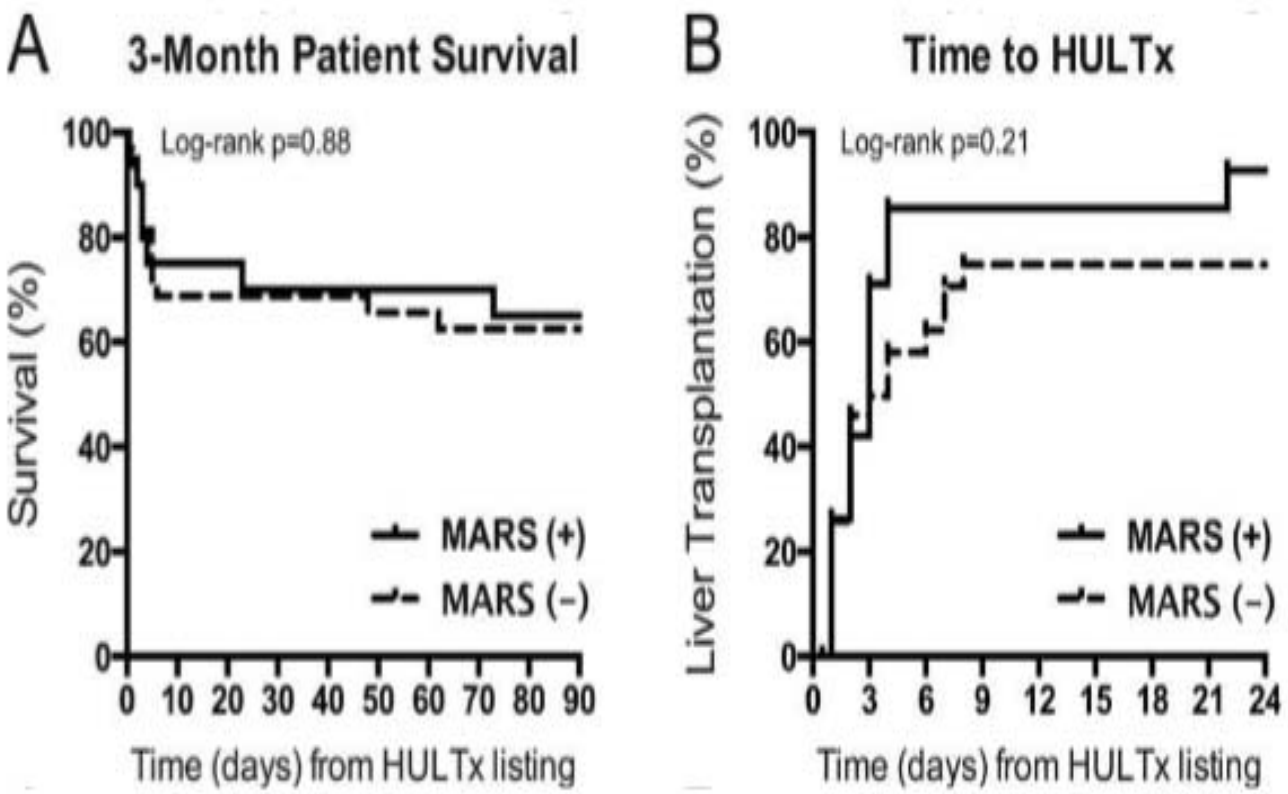


Figure 2. (A) Kaplan-Meier curves depicting 3-month survival from the time of wait listing for patients awaiting HULT who were either MARS-treated (solid curve) or not MARS-treated (dashed curve). (B) Time from listing to HULT for MARS-treated patients (solid curve) and non–MARS-treated patients (dashed curve). Subjects were censored at the time of death before HULT or at the time of removal from the wait list in the case of SR.

Défaillance neurologique

Anaesth Intensive Care 2014; 42: 78-88

Special Article

Preventing cerebral oedema in acute liver failure: the case for quadruple-H therapy

S. J. WARRILLOW*, R. BELLOMO†

Department of Intensive Care, Austin Health, Heidelberg, Victoria

SUMMARY

Severe cerebral oedema is a life-threatening complication of acute liver failure. Hyperammonaemia and cerebral hyperaemia are major contributing factors. A multimodal approach, which incorporates hyperventilation, haemodiafiltration, hypernatraemia and hypothermia (quadruple-H therapy), may prevent or attenuate severe cerebral oedema. This approach is readily administered by critical care clinicians and is likely to be more effective than the use of single therapies. Targeting of $PaCO_2$ in the mild hyperventilation range, as seen in acute liver failure patients before intubation, aims to minimise hyperaemic cerebral oedema. Haemodiafiltration aims to achieve the rapid control of elevated blood ammonia concentrations by its removal and to reduce production via the lowering of core temperature. The administration of concentrated saline increases serum tonicity and further reduces cerebral swelling. In addition, the pathologically increased cerebral blood-flow is further attenuated by therapeutic hypothermia. The combination of all four treatments in a multimodal approach may be a safe and effective means of attenuating or treating the cerebral oedema of acute liver failure and preventing death from neurological complications.

Key Words: cerebral oedema, acute liver failure

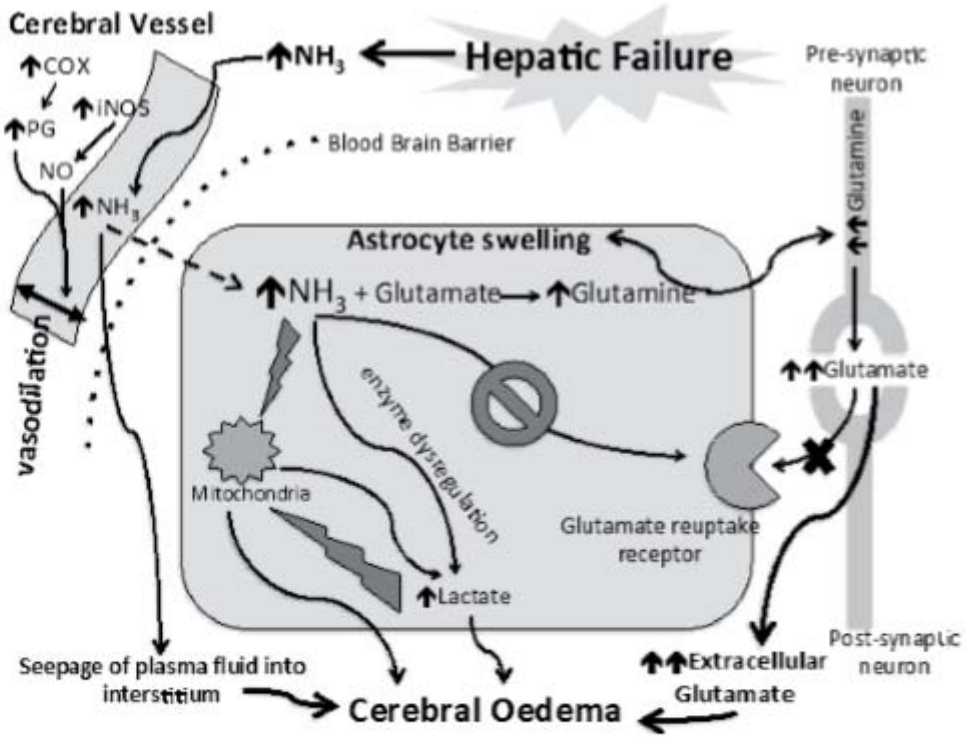


Figure 2: Hyperammonaemia from acute liver failure causes cerebral oedema through cerebral vasodilation, astrocyte swelling and an increase in interstitial fluid volume. The processes involve increases in glutamate concentrations, loss of regulation over crucial enzymes (such as alpha-ketoglutarate and lactate dehydrogenase) and mitochondrial dysfunction. COX=cyclooxygenase, PG=prostaglandins, iNOS=inducible nitric oxide synthase, NO=nitric oxide, NH_3=ammonia.

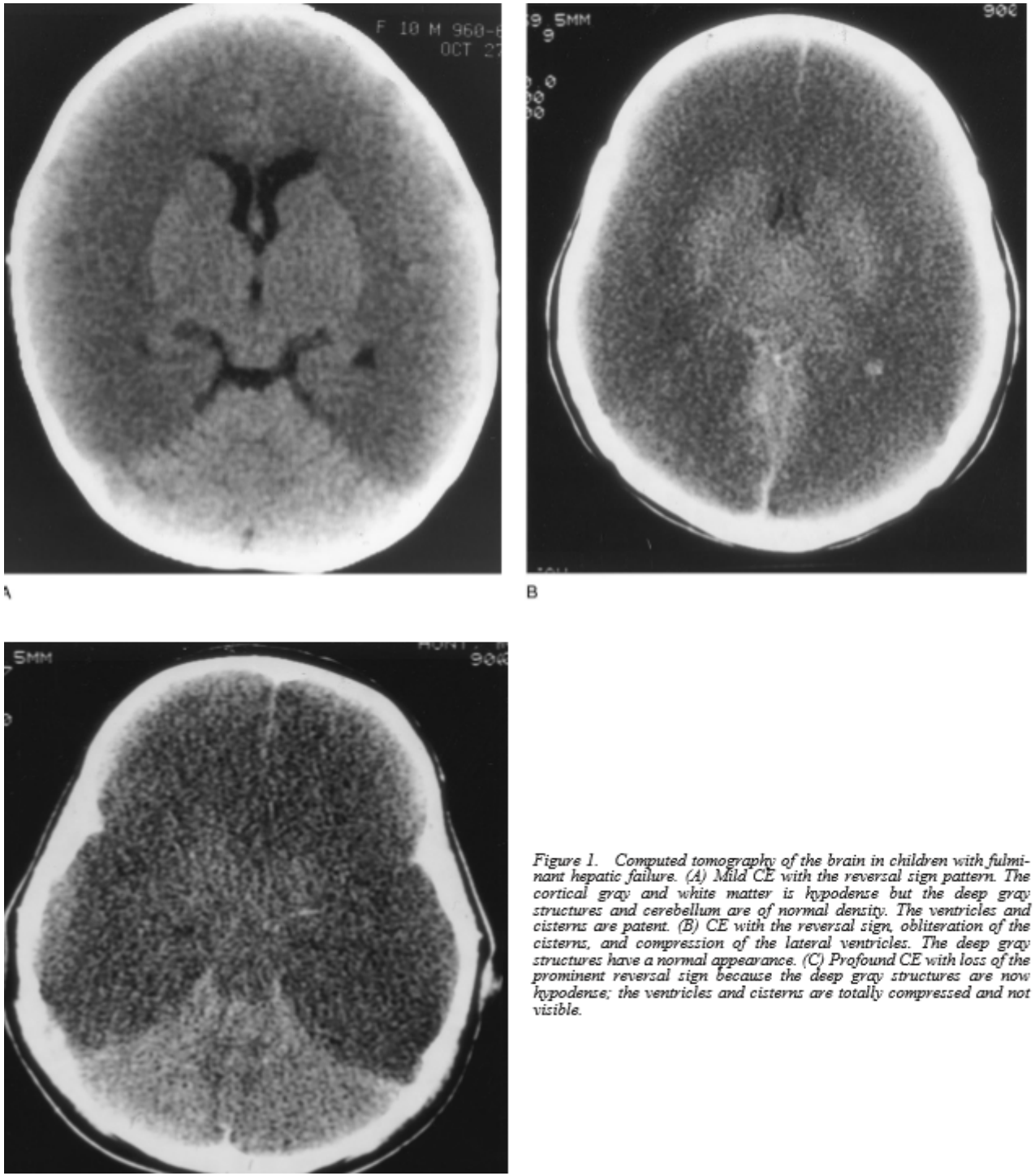


Figure 1. Computed tomography of the brain in children with fulminant hepatic failure. (A) Mild CE with the reversal sign pattern. The cortical gray and white matter is hypodense but the deep gray structures and cerebellum are of normal density. The ventricles and cisterns are patent. (B) CE with the reversal sign, obliteration of the cisterns, and compression of the lateral ventricles. The deep gray structures have a normal appearance. (C) Profound CE with loss of the prominent reversal sign because the deep gray structures are now hypodense; the ventricles and cisterns are totally compressed and not visible.

Summary of quadruple-H interventions, therapeutic targets and mechanisms of benefit

Intervention	Target	Method	Mechanism of benefit
Hyperventilation	The lower of $PaCO_2$=35 mmHg, or that achieved by patient prior to intubation	Adjust mechanical ventilation to achieve necessary minute ventilation	Reduction of cerebral hyperaemia and ICP
Haemodiafiltration	Blood ammonia <60 μmol/l and neutral daily fluid balance	High-exchange continuous haemofiltration incorporating dialysis if required	Ammonia clearance Assists cooling Metabolic, fluid and electrolyte control
Hypernatraemia	Serum sodium 148–152 mmol/l	Continuous infusion of concentrated saline via CVC	Increase serum tonicity causing cerebral dehydration Possible improvements in microcirculatory dynamics Anti-inflammatory effects Expansion of intravascular volume without significant overall positive fluid balance
Hypothermia	Core temperature of 33–35°C	Servo-controlled cooling blanket and RRT circuit	Reduced ammonia production Reduced ammonia uptake in the CNS Reduced CBF Reduced CMR Reduced neuro-excitation Anti-inflammatory effects

ICP=intracranial pressure, CVC=central venous catheter, RRT=renal replacement therapy, CNS=central nervous system, CBF=cerebral blood flow, CMR=cerebral metabolic rate.

Défaillance hémodynamique

-**Vasoplégie intense**

- Objectif : PAM (perfusion cérébrale= PAM-PIC)
- Remplissage +++:Cristalloïdes /colloïdes (albumine)

- Si insuffisant : **Noradrénaline**

Infections

-**Antibioprophylaxie** : encéphalopathie grade II , incidence moindre d'infection , pas de différence en terme de survie

Rolando N et al. Hepatology 1993;17:196—201.

-**Décontamination digestive** : pas de bénéfice

Rolando N Liver Transpl Surg. 1996 Jan;2(1):8-13.

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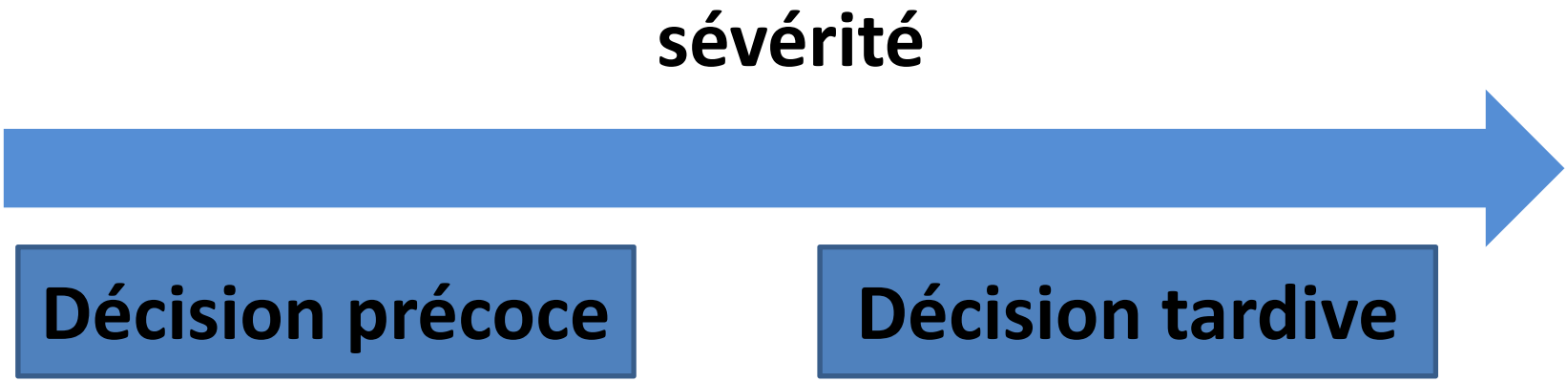
IHA : Indications de TH en urgence



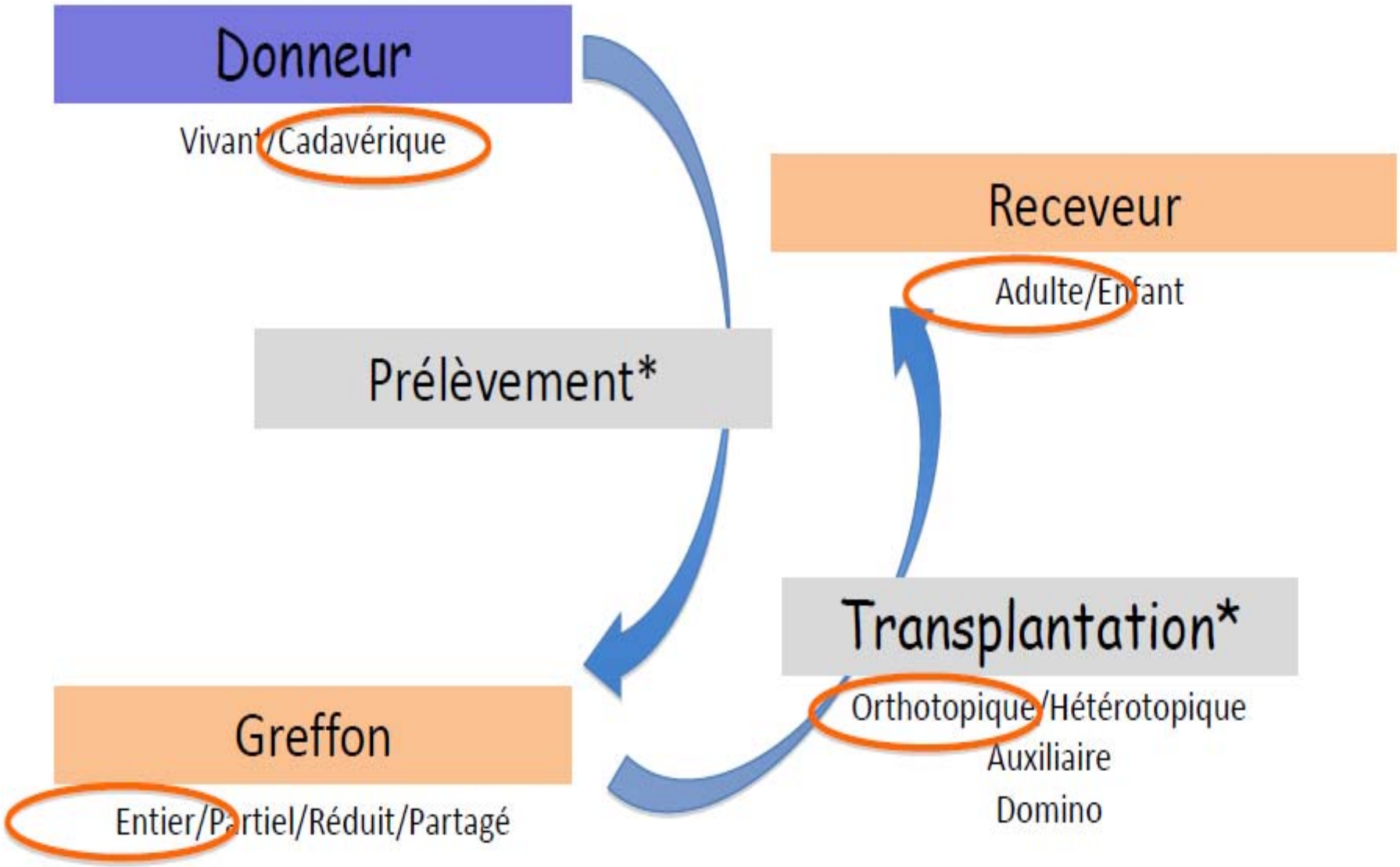
↓mortalité sur la liste ↑ mortalité sur la liste

↓risque opératoire ↑ risque opératoire

↑transplantation en excès ↓ transplantation en excès

Indications de TH en urgence

CRITERES	CONTENU
Critères de Clichy [6]	Confusion ou coma (encéphalopathie grade 3 et 4) Facteur V < 30 % si âge > 30 ans ou Facteur V < 20 % si âge < 30 ans
Critères du Kings College [19]	
Hépatite fulminante liée au paracétamol	pH < 7,3 ou Lactate artériel > 3 (après remplissage vasculaire) Créatinine > 300 µm/l plus INR > 6,5 plus encéphalopathie hépatique > grade 3
Hépatite fulminante non liée au paracétamol	INR > 7 ou au moins trois des critères suivants : INR > 3,5 Bilirubine > 300 _m/l Âge < 10 ans ou > 40 ans, Délai ictère-encéphalopathie > 7 jours


Donneur
Vivant/Cadavérique
Receveur
Adulte/Enfant
Prélèvement*
Transplantation*
Orthotopique/Hétérotopique
Auxiliaire
Domino
Greffon
Entier/Partiel/Réduit/Partagé

Codes de Priorité

Super Urgence Expert

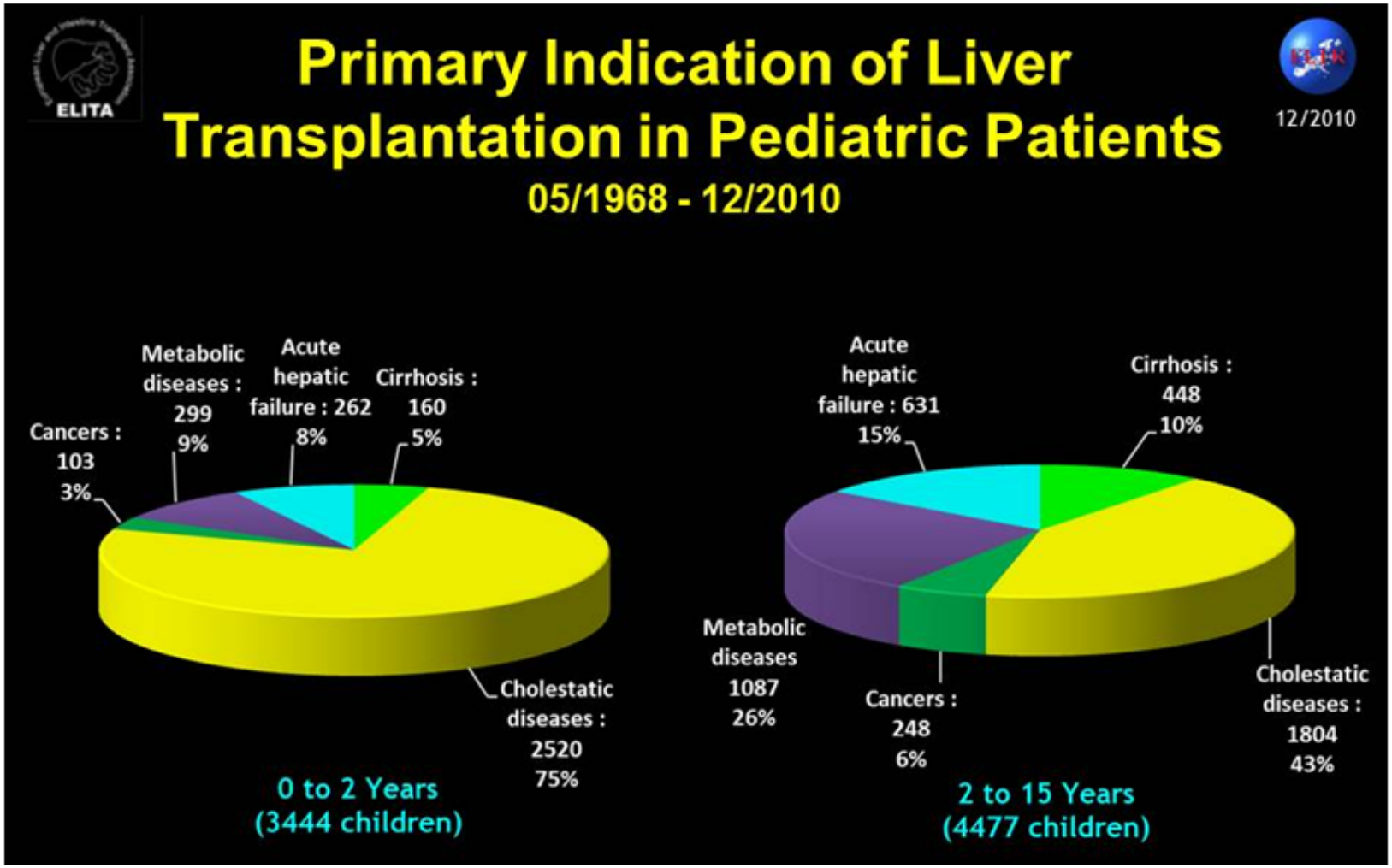
- IHA grave sur foie antérieurement sain
- Retransplantation < 8 jours

Priorité pédiatrique Expert

- Nécrose aiguë sur AVB
- Maladie métabolique révélée par une IHA

Elective normale

- Toutes les autres indications

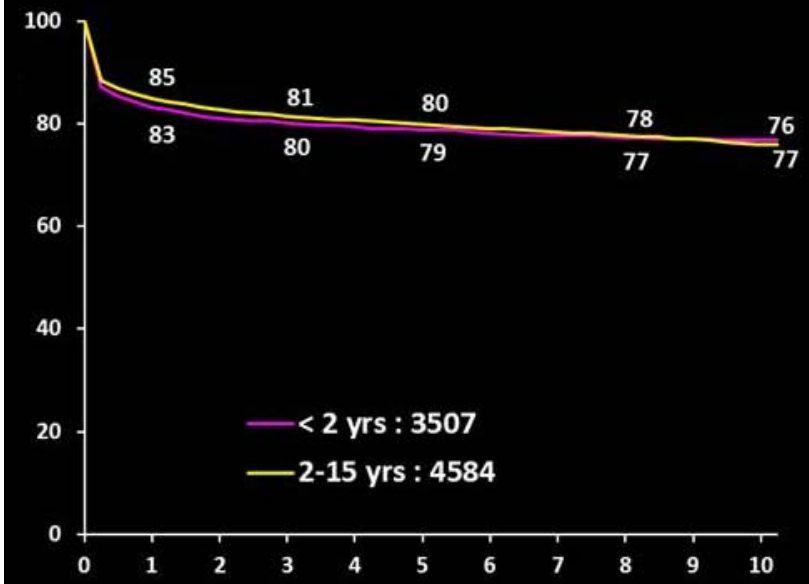

ELITA
Primary Indication of Liver Transplantation in Pediatric Patients
05/1968 - 12/2010
12/2010
Metabolic diseases : 299 9%
Acute hepatic failure : 262 8%
Cirrhosis : 160 5%
Cancers : 103 3%
Cholestatic diseases : 2520 75%
0 to 2 Years (3444 children)
Acute hepatic failure : 631 15%
Cirrhosis : 448 10%
Metabolic diseases 1087 26%
Cancers : 248 6%
Cholestatic diseases : 1804 43%
2 to 15 Years (4477 children)

Survival according to Children Recipient Age

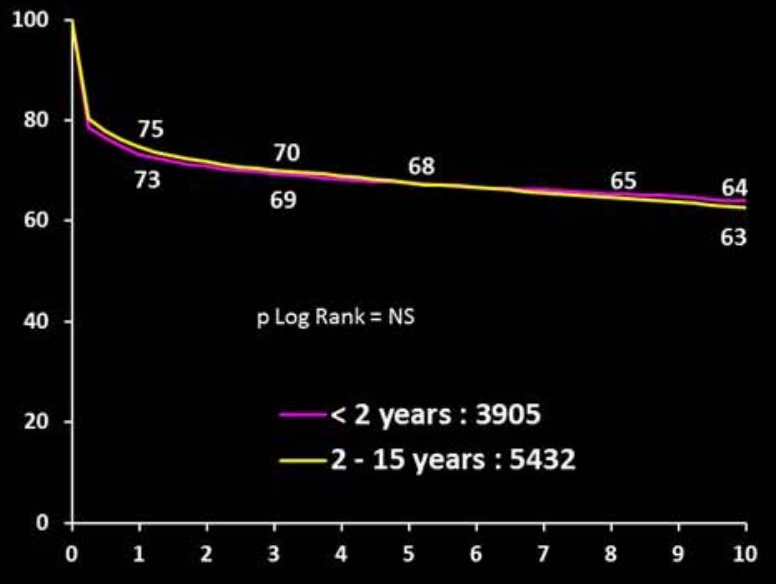
01/1988 - 12/2010

12/2010

Patient



Graft



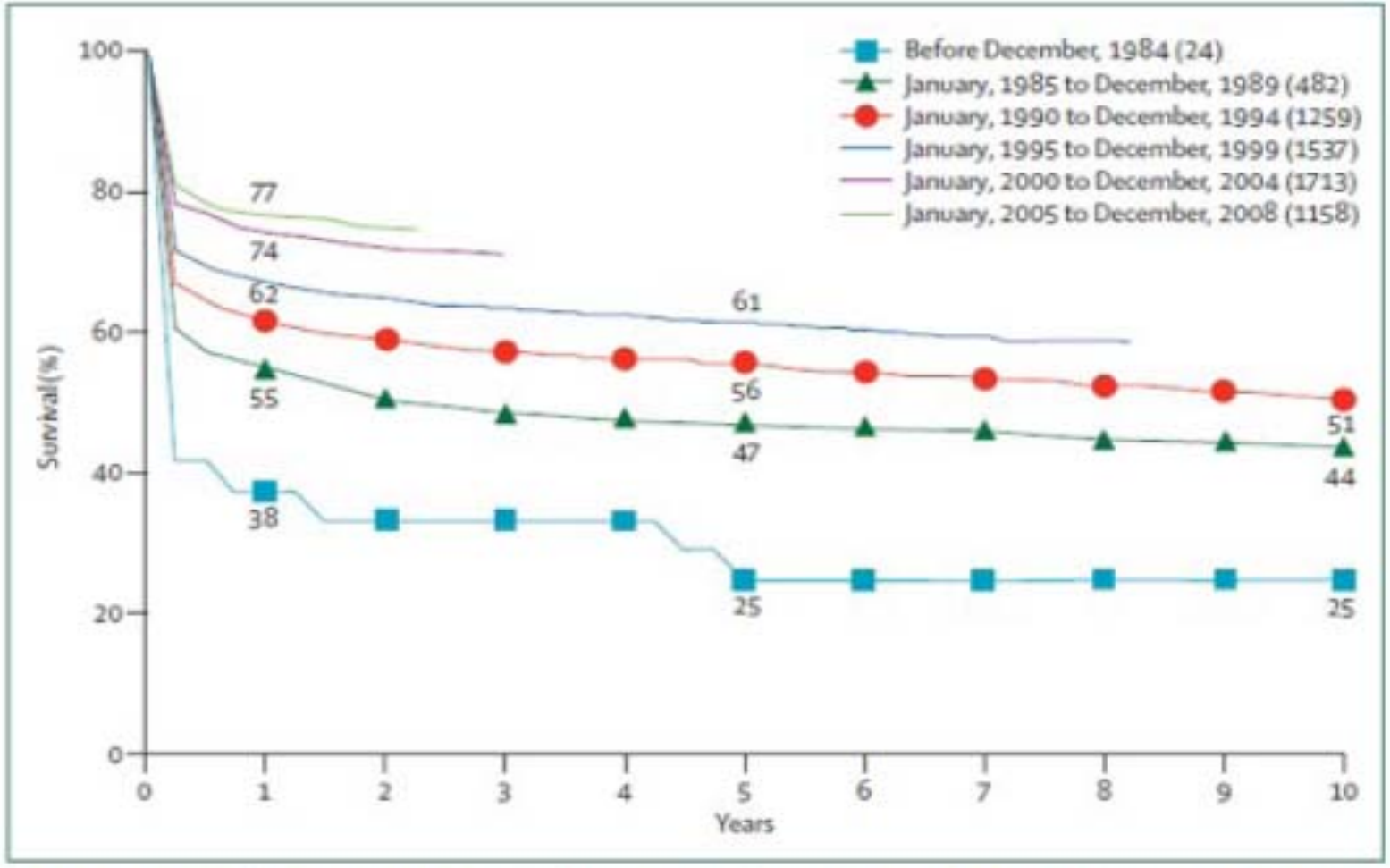


***Figure 3*: Survival after liver transplantation for acute liver failure by date of surgery in Europe, 1984–2008**
Data from the European liver transplant registry.[128] Numbers are completed 1, 5, and 10-year survival rates. Numbers in parentheses are surgeries done in each group.

Expérience
CHU Mohammed VI Marrakech

Pédiatrie

1er: IHA (hépatite virale A) (Foie partiel): **1ère greffe hépatique au Maroc**

2ème: cholestase intra-hépatique familiale progressive. (Foie total)

Adulte

1er: cirrhose (Foie total)

2ème: cirrhose (Foie partiel)

Receveur : Etape préopératoire

-Z. Abdessamai âgé de 10 ans

-Admis aux urgences pédiatriques le **7/2 /2014** (**Vendredi soir**) pour:

-hépatite fulminante, ictérique avec encéphalopathie hépatique **stade 2**.

-Poids=32 kg; Taille=1.48m

-Hépatite virale A (anticorps anti HVA-IgM **positi**f)

Evènements significatifs : l'entrée à l'hôpital -Acte opératoire

-**Samedi 08-02-14**, Directeur CHU averti: Candidat à la greffe hépatique en urgence

-Famille: Accord de la Maman, et hésitation du Mari et des soeurs

-Alerte: donneur cadavérique?

-Contact avec l'équipe de Beaujon: OK

lundi 10-02 AM:

*Réunion comité greffe hépatique

*les risques pour le donneur et receveur: Famille informée

Préparation logistique

Per-opératoire: Jeudi 13-02-14

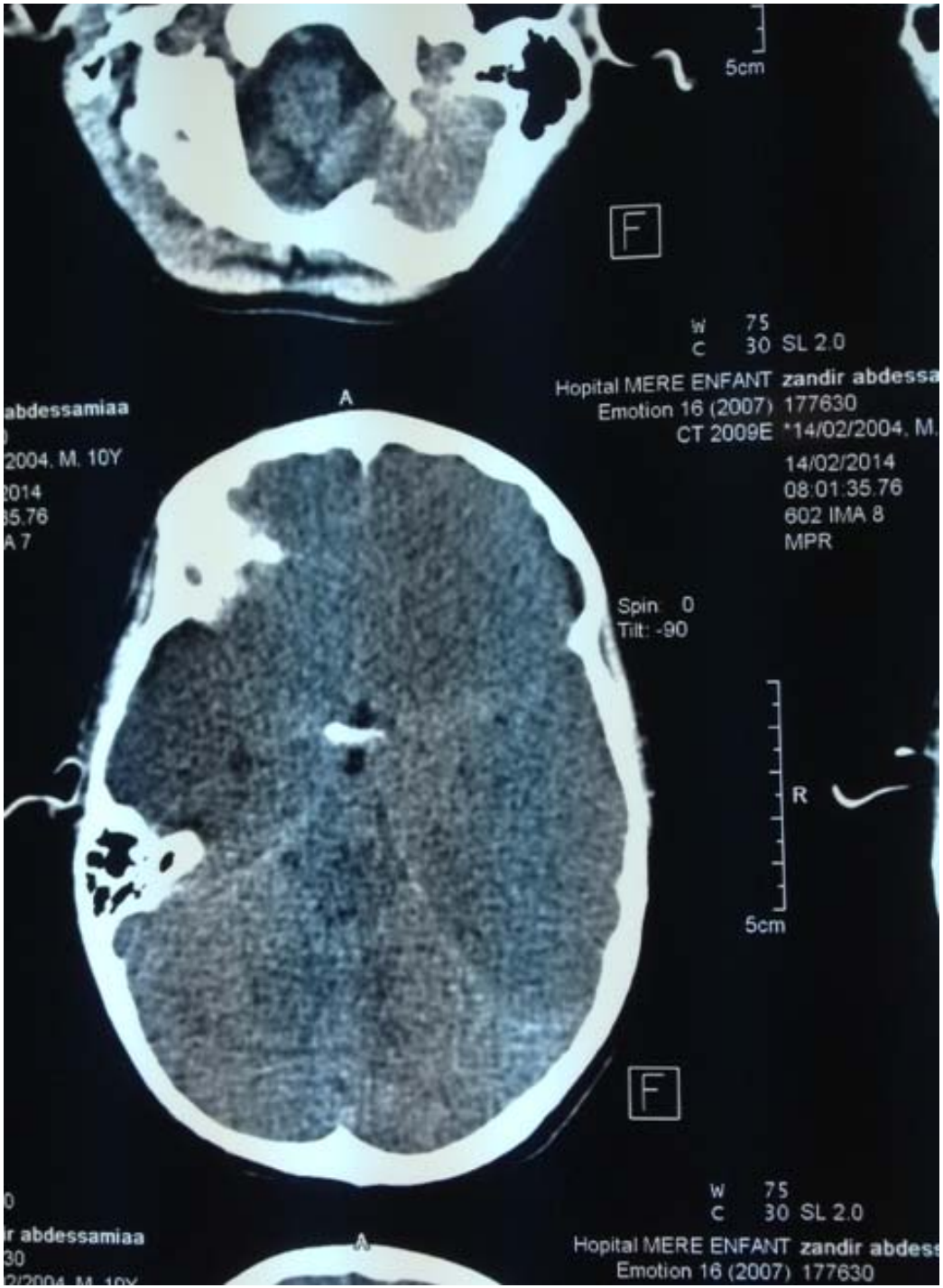
-Entrée au bloc opératoire a était programmée à 11h.

- Devant les **convulsions, la décérébration** et la semi-mydriase, l'enfant a était acheminé en urgence au bloc opératoire à 9h, il a était intubé, ventilé et mis en condition: régression de la mydriase, arrêt des convulsion.

Acte opératoire :

Greffe hépatique par donneur vivant (Donneur papa O+/Receveur fils A+)

Nature de l'intervention : Sauvetage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Hopital MERE ENFANT zandir abdes
Emotion 16 (2007) 177630
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30

-Séjour en réanimation: 2 mois

-Foie fonctionnel

-DCD 11-04-14 par son problème neurologique préalable

2ème Malade

Donneur: Youssef

Donneur potentiel en ME

Après 6 mois d’attente

Hassna la transplantée du foie avec son pédiatre : un sourire… un espoir !!!!

Conclusion

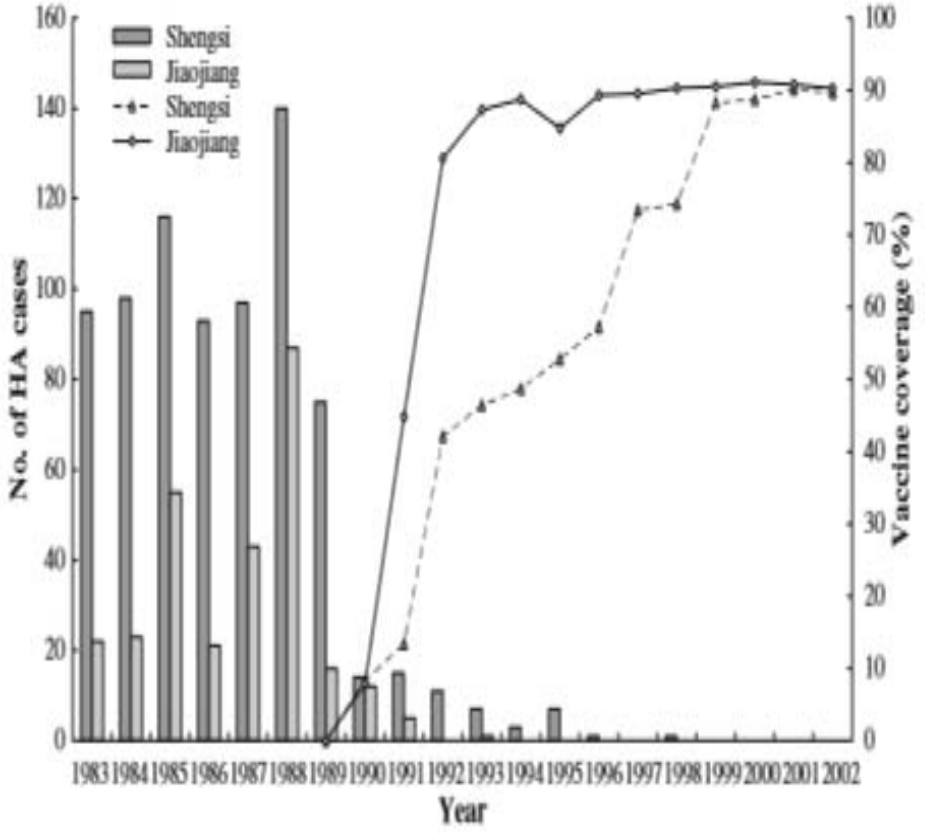


Fig. 7 Hepatitis A (HA) virus vaccine coverage and HA incidence in children aged 1–15 years in Jiaojiang and Shengsi counties, Zhejiang, China.

Leçons de cette expérience

-On a appris à travailler ensemble

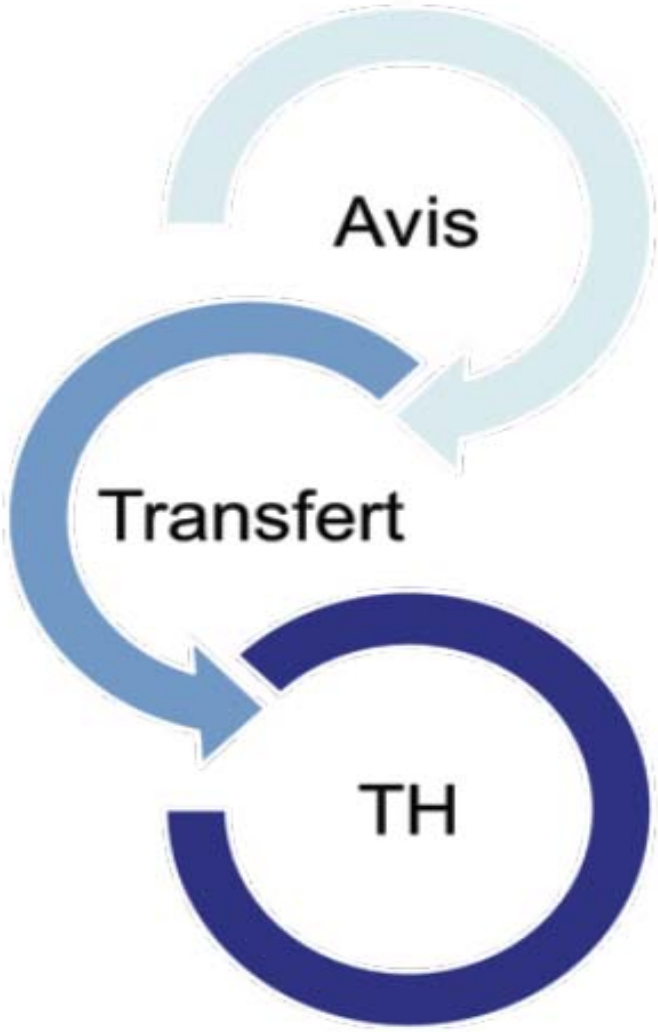
-Amélioration du niveau de soins

-Réussite technique

-C’est faisable

-**Transfert positif/Papa**

-Donneur vivant et cadavérique


Avis
Transfert
TH