QUELQUES CONTROVERSES EN ANESTHESIE-REANIMATION DU GRAND BRULE

CHU de Charleroi
Service d’Anesthésie

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Burn Survival vs Burn Size and Age

Survival (%)

100 90 80 70 60 50 40 30 20 10

Burn Size (% TBSA)

5 15 25 35 45 55 65 75

Curve | Age (Yrs)
--- | ---
1 | 0-2
2 | 3-20
3 | 21-40
4 | 41-50
5 | 51-60
6 | 61-70
7 | 71+

Insert:
A group of people standing around, with some individuals having burns on their arms and shoulders.
# Burn Mortality

(TBSA associated with LD50)

<table>
<thead>
<tr>
<th>Age Groups (years)</th>
<th>0-14</th>
<th>15-44</th>
<th>45-64</th>
<th>&gt;65</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bull &amp; Fisher (1942-52) 2807 Patients</td>
<td>49 n = 1366</td>
<td>46 n = 967</td>
<td>27 n = 330</td>
<td>10 n = 144</td>
</tr>
<tr>
<td>Bull 1967-70 1917 Patients</td>
<td>64 n = 962</td>
<td>56 n = 565</td>
<td>40 n = 246</td>
<td>17 n = 144</td>
</tr>
<tr>
<td>Curreri &amp; Abston 1975-79 1508 Patients</td>
<td>77 n = 803</td>
<td>63 n = 413</td>
<td>38 n = 178</td>
<td>23 n = 114</td>
</tr>
<tr>
<td>SBI/UTMB 1980-89 2164 Patients</td>
<td>98 n = 1524</td>
<td>70 n = 450</td>
<td>46 n = 127</td>
<td>19 n = 63</td>
</tr>
<tr>
<td>SBI/UTMB 1989-2005 Patients 1722</td>
<td>98 n=1083</td>
<td>82 n=420</td>
<td>78 n=152</td>
<td>35 n=67</td>
</tr>
</tbody>
</table>
Decreased Mortality From Major Thermal Injury Has Been Due To Advances In:

- Resuscitation
- Support of the Hypermetabolic Response To Trauma
- Early Closure of the Burn Wound
- Control of Infection
Integrins and Tissue Pressure

- Integrins: a group of adhesion molecules that attach to collagen fibers and fibroblast cells—ties putting a restrain on the gel in the collagen matrix
- Unleashing/cutting of ties volume → expansion possible → tissue pressure negative → fluid sucked into the wound → edema will bring Pf to normal

History of Fluid Resuscitation in Burns

- Before WW II, most burn victims with more than 20% TBSA were at mortal risk of acute renal failure.
- Fluid resuscitation used but ill defined until early 1950's (Evans et al 1952).
- This culminated in the derivation of the Parkland (or Baxter, 1968) formula.

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**Fluid and Electrolyte Balance In Burns**

Reiss E., Stirman J.A., Artz C.P., Davis J.H.
*JAMA* 1953; 152: 1309-1313

**Fluid Volume and Electrolyte Changes Of the Early Postburn Period**

C.R. Baxter
*Clin Plast Surg* 1974; 1: 643-709
### Table 1

**Formulae Used for Estimating Adult Burn Patient Resuscitation Fluid Needs**

**First Twenty-Four Hours**

<table>
<thead>
<tr>
<th>FORMULA:</th>
<th>ELECTROLYTE</th>
<th>COLLOID</th>
<th>GLUCOSE IN WATER</th>
</tr>
</thead>
<tbody>
<tr>
<td>Burn Budget of F. D. Moore</td>
<td>Lactated Ringer's 1000-4000 ml, 0.5 N saline 1200 ml</td>
<td>7.5% of body weight</td>
<td>1500-5000 ml</td>
</tr>
<tr>
<td>Evans</td>
<td>Normal Saline 1.0 ml/Kg, 1.0 ml/Kg/%burn</td>
<td>1.0 ml/Kg/%burn</td>
<td>2000 ml</td>
</tr>
<tr>
<td>Brooke</td>
<td>Lactated Ringer's 1.5 ml/Kg/%burn</td>
<td>0.5 ml/Kg/%burn</td>
<td>2000 ml</td>
</tr>
<tr>
<td>Parkland</td>
<td>Lactated Ringer's 4 ml/Kg/%burn</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertonic Sodium Solution</td>
<td>Volume to maintain urine output at 30 ml/hr, (Fluid contains 250 mEq Na/L)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Modified Brooke</td>
<td>Lactated Ringer's 2 ml/Kg/%burn</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

![Fig. 8.1](image.png)  
*Physiological curve of fluid requirements compared to Parkland formula, emphasizing that formulas are only guidelines for fluid therapy during burn shock (from Warden GD. World J Surg 1992; 16: 21-23).*
Fluid volume really given?

- We know that more is needed:
  1. In severe inhalation: about 5.7 ml / kg /
     %
  2. If initial fluid resuscitation is late (> 2 Hr PB)!
  3. If the burn is deep (mainly 3rd)
- Fluid administration in 7 Burn units the 1st 24 h: 40-80% more than the Baxter formula

Keymalyan et al. Annual Meeting of ABA, 1996
**Fluid Creep**


Fifty patients age 14-82 with 17-91% TBSA burns

<table>
<thead>
<tr>
<th>First Twenty Four Hours</th>
<th>All Patients</th>
<th>Patients with Inhalation Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluid Administered</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (ml/hr.)</td>
<td>5.2</td>
<td>6.1</td>
</tr>
<tr>
<td>&gt; 4.3 ml/kg/% TBSA</td>
<td>58%</td>
<td>69%</td>
</tr>
<tr>
<td>Urine Output</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 70 ml/hr.</td>
<td>66%</td>
<td>81%</td>
</tr>
<tr>
<td>&gt; 1.0 ml/kg/hr.</td>
<td>64%</td>
<td>75%</td>
</tr>
</tbody>
</table>
Reanimation Endpoints

Invasive Hemodynamic Monitoring

- The majority of studies using invasive hemodynamic monitoring (PA, TPID) to guide fluid resuscitation in burn, reported significant higher fluid requirements than Baxter or other empirical formulae: 4,5-9,5 ml/kg.BSA/24 h


Urinary Output

- Acceptable urinary output does not always guarantee or reflect adequate resuscitation after burn injury

Kaups et al. J Burn Care Rehab 1998; 19: 346-8
Holm et al. Burns 2000; 26: 25-33
AACS

• Definition: high intra-abdominal pressure without trauma/surgery to abdomen
• No consensus definition: constellation of clinical abnormalities including respiratory failure and oliguria with an acute elevation of IAP to more than 25 mm Hg
• Oliguria at IAP = 15-20 mm Hg
• Anuria above 30 mm Hg
• Causes: ↓ CO and renal parenchyma compression

AION & PION

• AION
  – Anterior portion of ON
  – optic disc and scleral canal portion of op
  – posterior ciliary arteries (< ophthalmic art.)
  – incompetent anastomotic ring (circle of Zinn & Hallen)
    = watershed zone

• PION
  – Posterior portion of ON
  – Centripetal pial vessels easily compressible
  – Branches of ophthalmic art. and central retinal art.
AION & PION

• Cause = lack of oxygen carriage of sufficient duration at a critical place for anatomical reason
  – visual field loss
  – loss of visual acuity
  – painless
  – visual field loss

• Mean time to diagnosis: 36 days after resusc.
  Cullinane et al. J Trauma 2000; 44: 381-7

• Association with bleeding and shock
  Johnson et al. Ophtalmology 1987; 94: 1577-84
  Hayreh. Ophtalmology 1987; 94: 1488-502

• Association with large resusc.
  Volume & with ACCS:
  350 patients with > 20 L resusc. Fluid: 2,6 % AION
  Cullinane et al. J Trauma 2000; 44: 381-7
Fig. 1. Resuscitation fluid volume (0–24 h) and %TBSA in individual. (□): Patients without ACS; (■): patients with ACS.
Reduction of Resuscitation Fluid Volume with Ascorbic Acid


• High-dose Vit C: 66 mg/kg per Hr during first 24-Hr

• 37 patients with more than 30% TBS randomized

• Conclusions
  – reduction of fluid requirements (3,0 ±1,7 vs 5,5 ± 3,1 ml/kg%), weight gain and edema
  – reduction of respiratory dysfunction
Fluid Resuscitation in Burn

- Lactate-Ringer or Hartmann (or other balanced crystalloid)
- No colloids during the first hours

HES (Voluven®) maintain plasma volume in a porcine model of septic shock with capillary leakage


- Intravascular persistency of artificial colloids in the presence of albumin leakage
Albumin & Burns: The Cochrane-group meta-analysis and its 3 studies in burn patients

- Greenhalgh DG. J Trauma; 39: 67-73, 1995
  patients >20% TBSA, 1-18 years, 6 weeks follow-up, randomly allocated to:
  G1 (n=34) albumin substitution to get 2.5-3.5 g/dl
  G2 (n=36) albumin substitution to get > 1.5 g/dl
  No differences in terms of survival, morbidity, length of stay or transfusions

  patients >50% TBSA, 28 years, 7 days follow-up, randomly allocated to:
  G1 (n=34) RL after the 1st day post-burn
  G2 (n=36) RL + albumin 2.5% after the 1st day post-burn
  Better CI in G2 during day 2, no significant differences later. Trend toward more lung water in G2 from day 2 to 7.

  patients >40-50% TBSA, 96 hours follow-up, randomly allocated to:
  G1 RL
  G2 hypertonic saline solution
  G3 hypertonic saline solution + Albumin 1.25%
  Faster hemodynamic normalization in G3, similar respiratory outcome

- Cochrane study group conclusions :
  relative risk to die = 2.47 (95% CI : .69 - 8.79)
  No advantage , rather disadvantageous to utilize albumin in burn patients
A quantitative and qualitative analysis of protein loss in human burn wounds

M Lehnhardt et al. Burns 31 (2005) 159-67

- Patients with 2nd degree burns (18-68%) studied from admission up to 48h: Burn wound enclosed in vinyl wound chambers covering 2.25 cm². Wound fluid analyzed for total protein content, albumin and immunoglobulin A, E, G, M

- Average loss of 17 g proteins on a wound area of 10% accumulating in 8 h, with a peak value of 21 g in the first 8 h of the day 2nd.
- For a 20% burn, the total of serum protein is lost to wound surface every 24 h
- Capillary leakage still important for albumin on day 2nd
- Role of loss of IG on susceptibility to infection?
Resuscitation

1. Volume

- Formulae are 50 years old
- 4 ml/kg/% or 2 ml/kg/%
- Pharmacological manipulations?
- Avoid overfilling (< 300 ml/kg/24H)

2. Which fluid

- No colloid the first hours?
- Artificial colloids
- Albumin
Decreased Mortality From Major Thermal Injury Has Been Due To Advances In:

- Resuscitation
- Support of the Hypermetabolic Response
- Early Closure of the Burn Wound
- Control of Infection

The Three Last are Related
Effects of Disease or Trauma on BMR

- Fasting-denutrition -10 à -30%
- Surgery +10%
- Polytrauma +30%
- Sepsis +50%
- Burn > 50% TBSA +100%

The body aims to deliver the optimal level of energy and substrate to the burn wound at the expense of other tissues and to keep a high body temperature.

<table>
<thead>
<tr>
<th>Formula</th>
<th>Age</th>
<th>Formula for Requirements</th>
</tr>
</thead>
<tbody>
<tr>
<td>Galveston Infant</td>
<td>0-1 year</td>
<td>$2100 \text{ kcal/m}^2 + 1000 \text{ kcal/m}^2$ burned/day</td>
</tr>
<tr>
<td>Galveston Revised</td>
<td>1-11 years</td>
<td>$1800 \text{ kcal/m}^2 + 1300 \text{ kcal/m}^2$ burned/day</td>
</tr>
<tr>
<td>Galveston</td>
<td>12 years</td>
<td>$1500 \text{ kcal/m}^2 + 1500 \text{ kcal/m}^2$ burned/day</td>
</tr>
<tr>
<td>Adolescent</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Curreri Formula</td>
<td>16-60 years</td>
<td>$25 \text{ kcal/kg/day}$ PLUS $40 \text{ kcal/%TBSA}$ burned/day</td>
</tr>
<tr>
<td>Adult</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Curreri Formula</td>
<td>&gt; 60 years</td>
<td>$25 \text{ kcal/kg/day}$ PLUS $65 \text{ kcal/%TBSA}$ burned/day</td>
</tr>
<tr>
<td>Seniors</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Metabolic Rates Elevation

- Actual metabolic rate depends on the TBSA
  - Elevated but near normal for burns up to 25% TBSA
  - For burns over 40%, MR are 1.4-2 times normal
- Resting metabolic rates remain at 180% of RMR during acute hospitalization, dropping to 150% when injuries are fully healed and stay higher than normal up to one year later.

Effect of Ambient Temperature on Metabolic Rate: We Need Warm Rooms

Association of Metabolic Rate and Protein Catabolism

Complications of Catabolism

- Consequences associated with erosion of body mass\(^1\)

<table>
<thead>
<tr>
<th>% Lost</th>
<th>Altered Physiology</th>
<th>% Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>10%</td>
<td>Impaired immune function</td>
<td>10%</td>
</tr>
<tr>
<td>20%</td>
<td>Decreased wound healing</td>
<td>30%</td>
</tr>
<tr>
<td>30%</td>
<td>Pneumonia, pressure sores</td>
<td>50%</td>
</tr>
<tr>
<td>40%</td>
<td>Death (pneumonia)</td>
<td>100%</td>
</tr>
</tbody>
</table>

\(^1\)Chang, DeSanti, Demling. SHOCK. 1998
Persistence of Muscle Catabolism after Severe Burn

**Postabsorptive Net Balance**

N=25  N=23  N=21  N=21

*μmol PHE/min/100 ml leg*

-0.045  -0.030  -0.015  0.000  0.015

Time After Burn

6 mo  9 mo  12 mo

Normal
Young Adults

Data presented as mean±SEM
*p<0.05 vs. 6 and 9 months*

**Change in Lean Mass from Discharge**

N=16  N=16

Grams

-2000  -1500  -1000  -500  0  500  1000

Time After Injury

6 months  9 months  12 months

Data presented as mean±SEM
*p<0.05 vs Discharge
†p<0.01 vs 9 months*

6 Carbon 3 Carbon Flow

LIVER
- Glycogen
- Protein
- Urea
- ± Insulin
- ++++ Catecholamines
- ++++ Glucagon
- ++++ Glucocorticoids

GLUCOSE

Heat

SKELETAL MUSCLE
- Glycogen
- Protein
- ± Insulin
- ++++ Catecholamines

WOUND
- Anaerobic Metabolism
- Heat

3-C

Triglycerides

FAT
- ± Insulin
- ++++ Catecholamines
- Fatty Acids

Lactate
**Effects of Burn Injury on the Liver**

Liver Weight Per Body Weight for Normal vs Burned Patients (2 mo. to 15 yrs. of age)

<table>
<thead>
<tr>
<th>Full-Thickness Burn (%)</th>
<th>Liver wgt/BW (gm/kg)</th>
<th>Weight Increase (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal (n=14)</td>
<td>0</td>
<td>34.3 ± 1.1</td>
</tr>
<tr>
<td>Burn (n=14)</td>
<td>76 ± 5</td>
<td>75.6 ± 6.0*</td>
</tr>
</tbody>
</table>

Burn Size and Liver Weight Ratios Are Means ± SE, BW = Body Weight

* Significant Difference at p<.001
Metabolic Manipulations

- Warm the patient
- Early vs. delayed treatment
- Dietary composition
- Insulin
- Oxandrolone
- Propranolol
- Growth Hormone
- Insulin-like Growth Factor-1
- Itraconazole
A New Concept
In the Early Excision and Immediate Grafting of Burns

J Trauma 1970; 10: 1103-1108
Early Excision and Closure of the Burn Wound

**Early Excision Mortality vs Conservative Therapy**

<table>
<thead>
<tr>
<th>Treatment Group</th>
<th>n</th>
<th>Age (yrs)</th>
<th>Mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early Excision</td>
<td>22</td>
<td>17-30</td>
<td>9</td>
</tr>
<tr>
<td>Conservative</td>
<td>11</td>
<td></td>
<td>45*</td>
</tr>
<tr>
<td>Early Excision</td>
<td>7</td>
<td>31-55</td>
<td>57</td>
</tr>
<tr>
<td>Conservative</td>
<td>16</td>
<td></td>
<td>75</td>
</tr>
</tbody>
</table>

*Significant difference at p<.03

**Length of Hospital Stay; Serial Debridement vs Early Excision**

<table>
<thead>
<tr>
<th></th>
<th>Days Postburn Final Coverage</th>
<th>Length of Hospital Stay</th>
</tr>
</thead>
<tbody>
<tr>
<td>1977-1981 Serial Debridement n = 25</td>
<td>67 ± 6</td>
<td>97 ± 8</td>
</tr>
<tr>
<td>1981-1984 Early Excision n = 25</td>
<td>44 ± 4*</td>
<td>57 ± 5*</td>
</tr>
</tbody>
</table>

Means ± SE
*Significant Difference between groups, p<.01

Effect of Delay to Excision and Grafting on Protein Catabolism

Changes in % Lean Body Mass and % Fat Mass

Diet Study of Fat and Carbohydrates

Plasma Insulin Level

Data presented as mean±SEM
*p<0.05

Muscle Protein Net Balance with Dietary Manipulation

- High Fat → High Carb, N=6
- High Carb → High Fat, N=7
Insulin

## Insulin - Donor Site Healing Time (Days)

<table>
<thead>
<tr>
<th>Patients</th>
<th>Placebo</th>
<th>Insulin</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>3</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>8</td>
<td>5</td>
</tr>
<tr>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>6</td>
<td>7</td>
<td>4</td>
</tr>
<tr>
<td><strong>Mean ± SD</strong></td>
<td><strong>6.51 ± 0.95</strong></td>
<td><strong>4.71 ± 2.3</strong>*</td>
</tr>
</tbody>
</table>

* p<.05 placebo versus insulin period (paired t-test)

Oxandrolone

• Anabolic agents ameliorate catabolism
• Oxandrolone
  – Synthetic testosterone analogue
  – Oral
  – Inexpensive
  – Safe
• Preliminary studies in burn patients
• Adults: 10 mg/12 Hr PO
• FDA: 23 Oct 1991
Protein Synthetic Efficiency

Fraction of Available Amino Acids Accreted into Muscle

0.0 0.1 0.2 0.3 0.4 0.5
Baseline Control

7 Time Control Subjects

Fractional Synthetic Rate of Muscle Protein Synthesis

% per hour

0.0 0.1 0.2 0.3 0.4 0.5
Baseline Oxandrolone

7 Oxandrolone Subjects

Data presented as mean ± SEM
†p<0.05 vs. Baseline
*p<0.05 vs. Time Control

Twenty subjects randomized to receive either 0.1 mg/kg Oxandrolone BID, or placebo diluent.
Percent change in the number of patients above the 25\textsuperscript{th} percentile for Height compared to discharge

![Bar chart showing percent change in height over time for Placebo (n=25) and Oxandrolone (n=24) with significant differences marked by * (p<0.05).]

Percent change in the number of patients above the 25\textsuperscript{th} percentile for Weight compared to discharge

![Bar chart showing percent change in weight over time for Placebo (n=25) and Oxandrolone (n=24) with significant differences marked by * (p<0.05).]
Propanolol

*N Engl J Med 2001; 345: 1223-9*

– PO 0.33 mg/kg by nasogastric tube every 4 hours
– Increase progressively to 1 mg/kg/ 4 hours
– Aim at 20% reduction in heart rate
Propranolol

Δ in % Fat Free Mass
4 Week Treatment Course

Data presented as mean±SEM
*p<0.01

% Lean Body Mass
at Discharge

Data presented as mean±SEM
*p=0.01

Muscle Protein Net Balance

Dual-Image X-Ray Absorptiometry

Bars are Means ± SEM for % change.

* Significant difference between Placebo and Propranolol at p<.002, unpaired t-test.

Trunk Fat


One Dimensional Doppler Measurements of Liver

Bars are means ± SD for % change.

* Significant difference between placebo and propranolol at p < .02, unpaired t-test.

<table>
<thead>
<tr>
<th>Past</th>
<th>Present</th>
<th>Future</th>
</tr>
</thead>
<tbody>
<tr>
<td>Starvation with loss of body mass</td>
<td>Hypermetabolism reduced; preservation of body mass</td>
<td>Hormonal manipulation</td>
</tr>
<tr>
<td>Superior mesenteric artery syndrome</td>
<td>Early physical therapy</td>
<td>Improved pain control</td>
</tr>
<tr>
<td>Prolonged convalescence</td>
<td>Hepatic steatosis</td>
<td>Organ specific diets</td>
</tr>
<tr>
<td></td>
<td>Fluid overload</td>
<td></td>
</tr>
</tbody>
</table>
Decreased Mortality From Major Thermal Injury Has Been Due To Advances In:

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- Control of Infection

The Three Last are Related
Deaths from Sepsis

[Graph showing the number of deaths from sepsis over different years.]
Invasive Burn Wound Infections  
1991-2004

Admissions: 3,876

Patients with Invasive Wound Infections

Bacterial:
  Gram negative bacilli: 20
  Gram positive cocci: 5

Fungal:
  Aspergillus sp.: 47
  Mucor sp.: 16
  Candida sp.: 2

Incidence 2.3%
Mortality 55/90 (61%)
Mortality associated with Fungal Infection in Burns

- 30% of Burn Patients Become Colonized With *Candida Sp.* At Some Time During Their Acute Hospital Stay.
Results (2)

- Characteristics of *P. Aeruginosa* colonized patients
  - Most of them were colonized in the unit, so the main factor was the length of stay (LOS)
  - Out of 441 patients, 70 patients (16%) colonized,
  - 12 (17%) at admission, 58 (83%) later (nosocomially)
  - Colonization versus hospitalization length, age and TBSA
366 Ps. Aeruginosa isolates (incl. 45 environmental): 48 genotypes

48 AFLP patterns and dendrogram of the Ps. Aeruginosa genotypes
DNA genotyping: Results (1)

• 48 different AFLP genotypes - N patient = 70 (100%)
  • 21 exclusively from environment
  • 15 from only one patient
  • 12 from several patients (N = 57), of which 2 in env.

Conclusion:

• No ongoing *Ps. Aeruginosa* reservoir in environment
• But, 57 events of cross-acquisition

• Concentrating on the genotypes found in n patients

• 27% of the patients colonized by 2 to 4 strains
• And, 2 genotypes were found in 60% of the patients
  – AFLP 35: 131 isolates, 29 patients
  – AFLP 8: 76 isolates, 19 patients
Time course of AFLP 35 and AFLP 8 colonization
Poster-Abstract / BSM / 24-11-2006:

Bacteriophages: “To Claim”, “Not to Claim”, “What to Claim”?

At the Burn Centre of the Military Hospital Brussels, as all over the world, we are confronted with antibiotic-resistant bacteria who are infecting patients. We feel the need for alternatives to antibiotic treatments in general. The Burn Unit in Brussels cares for 1350 patients per year (10,000 consultations/year). We are convinced that Phage-Therapy has to be (re-) introduced in “Western” Europe. For decades, the therapeutic use of Phages has been documented in e.g. “Eastern” Europe and Russia. Safety has been proven. Bacteriohages are viruses that only attack bacteria. Genetic material of Bacteriophages was never found back in Eukaryotic cells. Phage therapy never penetrated to the (Western) European or the U.S. market partially because the Pharma Industry preferred to realise the marketing of easily patentable and easily marketable antibiotics. Antibiotics could be produced for broad-spectrum use. That was not the case for Phages. Phages act against specific bacteria. Diagnostic tools for exact identification of the infecting bacteria did not exist. Now phage-cocktails can be prepared an rapid identification of bacteria is possible.

The Phage-cocktails that will be used are now produced in collaboration with the University Hospital of Ghent (Dept. Microbiology), the Tbilisi institute of microbiology and the Moscow Institute of microbiology. Part of these phages were isolated at the Brussels Burn Unit. Containing Phages present in (e.g.) the wound beds of the hospitalized patients. They were up-scaled ‘in vitro’ and will be given back to the patients.
Le chirurgien voudrait que vous lui fassiez un autographe pour son fils avant l'opération au cas où l'anesthésie se passerait mal.

Ça fait toujours plaisir!