Monitoraggio intraoperatorio in chirurgia ortopedica ad alto rischio emorragico.

Variazioni emodinamiche, perfusione d'organo
e ripercussioni sul microcircolo.

FINALITA' DEL MONITORAGGIO

GESTIONE PIU' APPROPRIATA PER MIGLIOR OUTCOME: mantenere ottimale

o ottimizzare

la perfusione tessutale.

RISCHIO PZ-CH

- BASSO PROFILO DI RISCHIO: REACTIVE MANAGEMENT

- **ALTO PROFILO DI RISCHIO: PRO-ACTIVE MANAGEMENT**

FASE PRE-OP

- GESTIONE Fattori di rischio Pz: CRITICITA' ELIMINABILI/MODIFICABILI?
- PIANIFICAZIONE:
    - PBM
    - DISPONIBILITA'/APPROVVIGIONAMENTO EMODERIVATI
    - Monitoraggio, Presidi gestione emodinamica
    - CONOSCENZA DELLA PROCEDURA CHIRURGICA
    - STRATEGIA PROATTIVA

FASE INTRA-OP

- TIPO DI ANESTESIA
- POSIZIONE PAZIENTE
- ESPOSIZIONE E INVASIVITA' CHIRURGICA
- STRATEGIE BLOOD SPARING
- COAGULAZIONE: STRATEGIE EMOSTATICHE TOPICHE E SISTEMICHE
- STRATEGIE GESTIONE SANGUE
- DA STRATEGIA PROATTIVA A REATTIVA: DAMAGE CONTROL

FASE POST-OP

- STABILITA' PZ: CONSOLIDAMENTO O RAGGIUNGIMENTO
- SVEZZAMENTO SUPPORTI
- P.E. POST-OP

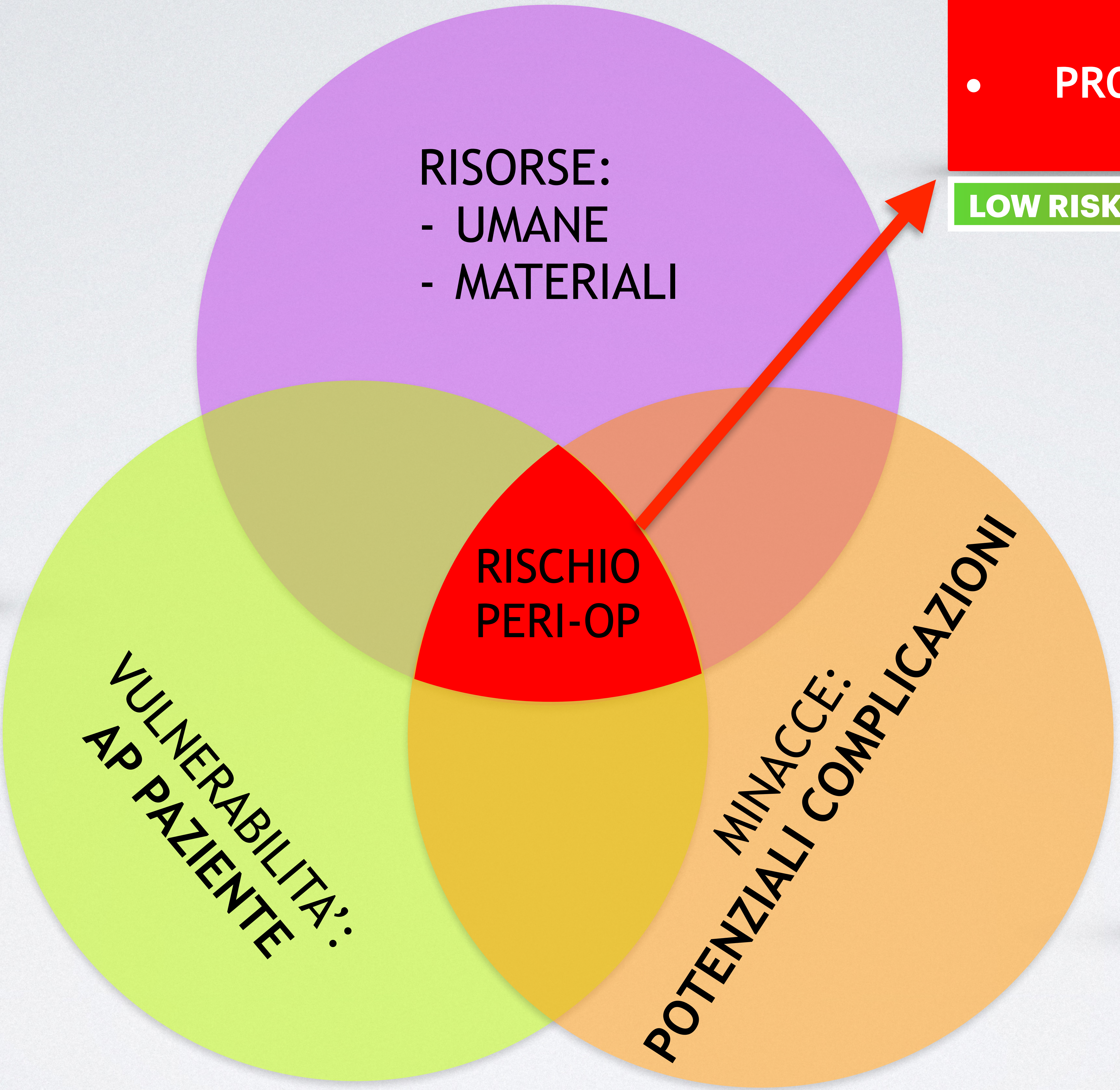
ANALISI DEL RISCHIO

- Anestesista
- Chirurgo
- Nurse
- Strumentista
- OSS

- Esperienza
- Capacità
- Tecniche

- Devices dedicati
- Sistemi di Monitoraggio
- Strumentazione
- Impiantabili

- Patologie croniche:
  - Lievi
  - Moderate
  - Severe
- Patologie croniche:
  - Stabili
  - Instabili
- Stati morbosi acquisiti dalla patologia acuta o dalle terapie
- Riserva Funzionale



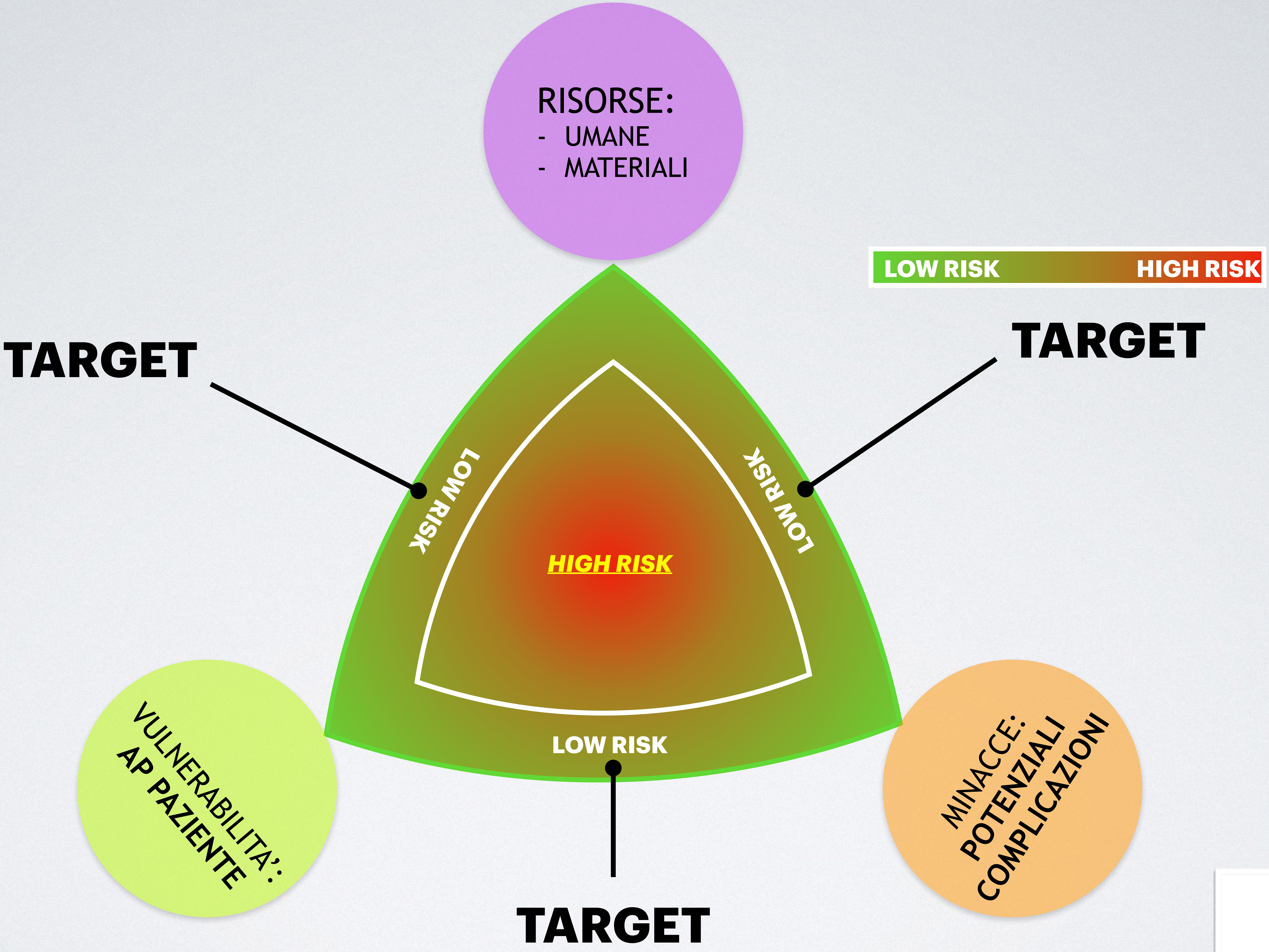
- APPROCCIO PROATTIVO PER ELIMINAZIONE/MODULAZIONE DELLE CRITICITÀ
- PROGRAMMI/PROCEDURE DI SICUREZZA

LOW RISK — HIGH RISK

- Complicanze anestesiologiche
- Complicanze mediche
- Complicanze chirurgiche
- Malfunzionamenti devices

Orthopea. Anestesia e Ortopedia

ANALISI



Dott. Tiziano CRESPI
Anestesia&Rianimazione. Ospedale Galeazzi - Sant'Ambrogio

Orthopea®
Anestesia e Ortopedia

DOMANDA / OFFERTA

QUANTO CE N'E'?

- **EBV**:
  - **Adulto: 7% peso ideale o 70 ml/kg$_{\mathbf{id}}$** (riduzione in età avanzata): ~ **<u>5000 ml</u>**
  - Pediatrici: 8-9% peso
  - Neonati: 9-10% peso

- **P.E. CONSENTITE?**

QUANTO NE PERDO?

Injury, Int. J. Care Injured 45S (2014) S35–S38

Contents lists available at ScienceDirect

Injury

journal homepage: www.elsevier.com/locate/injury

The ATLS® classification of hypovolaemic shock: A well established teaching tool on the edge?

M. Mutschler [a,*], T. Paffrath [a], C. Wölfl [b], C. Probst [a], U. Nienaber [c], I.B. Schipper [d], B. Bouillon [a], M. Maegele [a]

[a] Department of Orthopedics, Trauma and Sportsmedicine, Cologne-Merheim Medical Center (CMMC), Private University Witten-Herdecke, Cologne, Germany
[b] Department of Trauma and Orthopedic Surgery, BG Hospital Ludwigshafen, Ludwigshafen, Germany
[c] Academy for Trauma Surgery (AUC), Berlin, Germany
[d] Department of Trauma Surgery, Leiden University Medical Center, Leiden, The Netherlands

EMORRAGIA MASSIVA:

– Perdita dell'**intero EBV** in un periodo di **24h**
– Perdita del **50%** di EBV in un periodo di **3h**

Table 1
The ATLS® classification of hypovolaemic shock [1].

	Class I	Class II	Class III	Class IV
Blood loss in %	<15	15–30	30–40	>40
Pulse rate	<100	100–120	120–140	>140
Blood pressure	Normal	Normal	Decreased	Greatly decreased
Pulse pressure	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate	14–20	20–30	30–40	>35
Mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic
Urine output (mL/hr)	>30	20–30	5–15	Minimal

QUANTO TOLLERO?

70 KG ~ **5000 ML**

- **CLASSE I: < 750 ML**
- **CLASSE II: 750 - 1500 ML**
- **CLASSE III: 1500 - 2000 ML**
- **CLASSE IV: > 2000 ML**

50 KG ~ **3500 ML**

- **CLASSE I: < ~ 500 ML**
- **CLASSE II: ~ 500 - 1000 ML**
- **CLASSE III: ~ 1000 - ~ 1400 ML**
- **CLASSE IV: > ~ 1400 ML**

QUANTO NE PERDO?

Injury, Int. J. Care Injured 45S (2014) S35–S38

ELSEVIER

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The ATLS® classification of hypovolaemic shock: A well established teaching tool on the edge?

CrossMark

M. Mutschler [a,*], T. Paffrath [a], C. Wölfl [b], C. Probst [a], U. Nienaber [c], I.B. Schipper [d], B. Bouillon [a], M. Maegele [a]

[a] Department of Orthopedics, Trauma and Sportsmedicine, Cologne-Merheim Medical Center (CMMC), Private University Witten-Herdecke, Cologne, Germany
[b] Department of Trauma and Orthopedic Surgery, BG Hospital Ludwigshafen, Ludwigshafen, Germany
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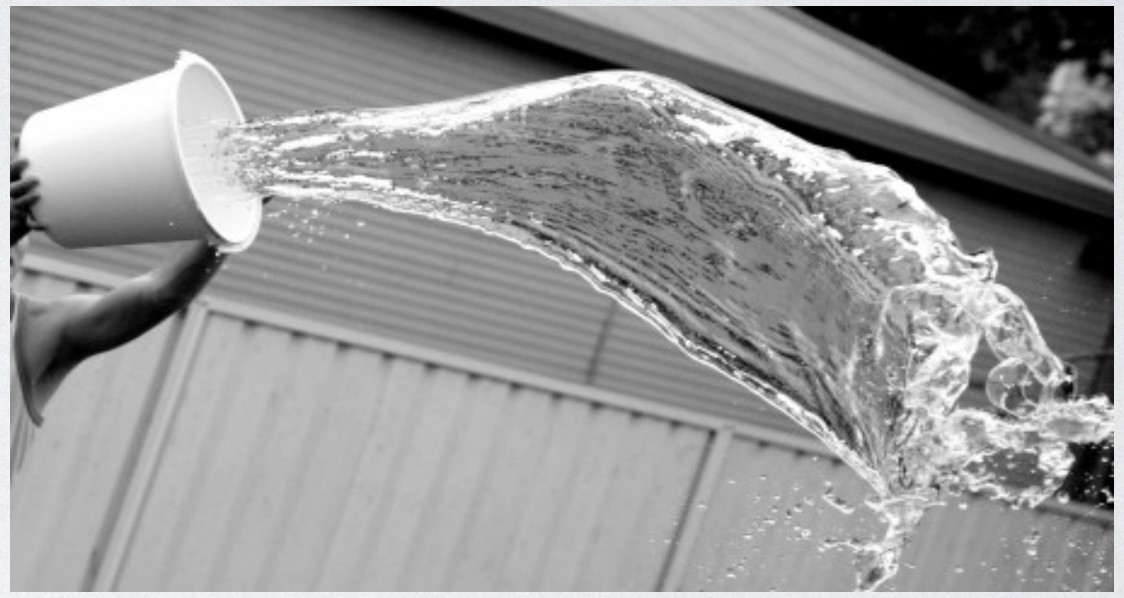
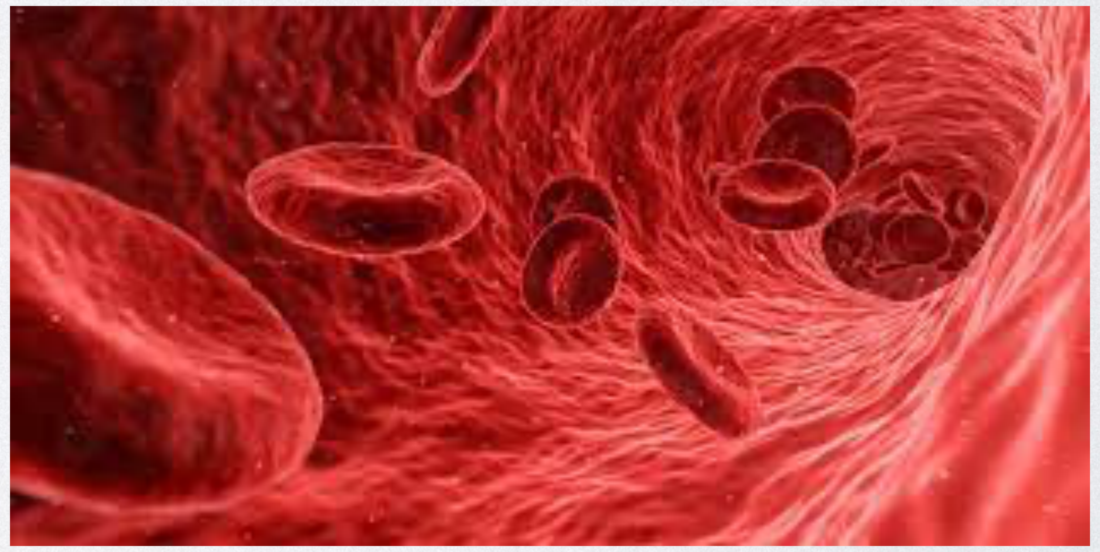
EMORRAGIA → IPOVOLEMIA E ANEMIA

EMORRAGIA

↓ VOLUME EMATICO CIRCOLANTE → ↓ PRELOAD



↓ Hb → IMPATTO SU DO_2



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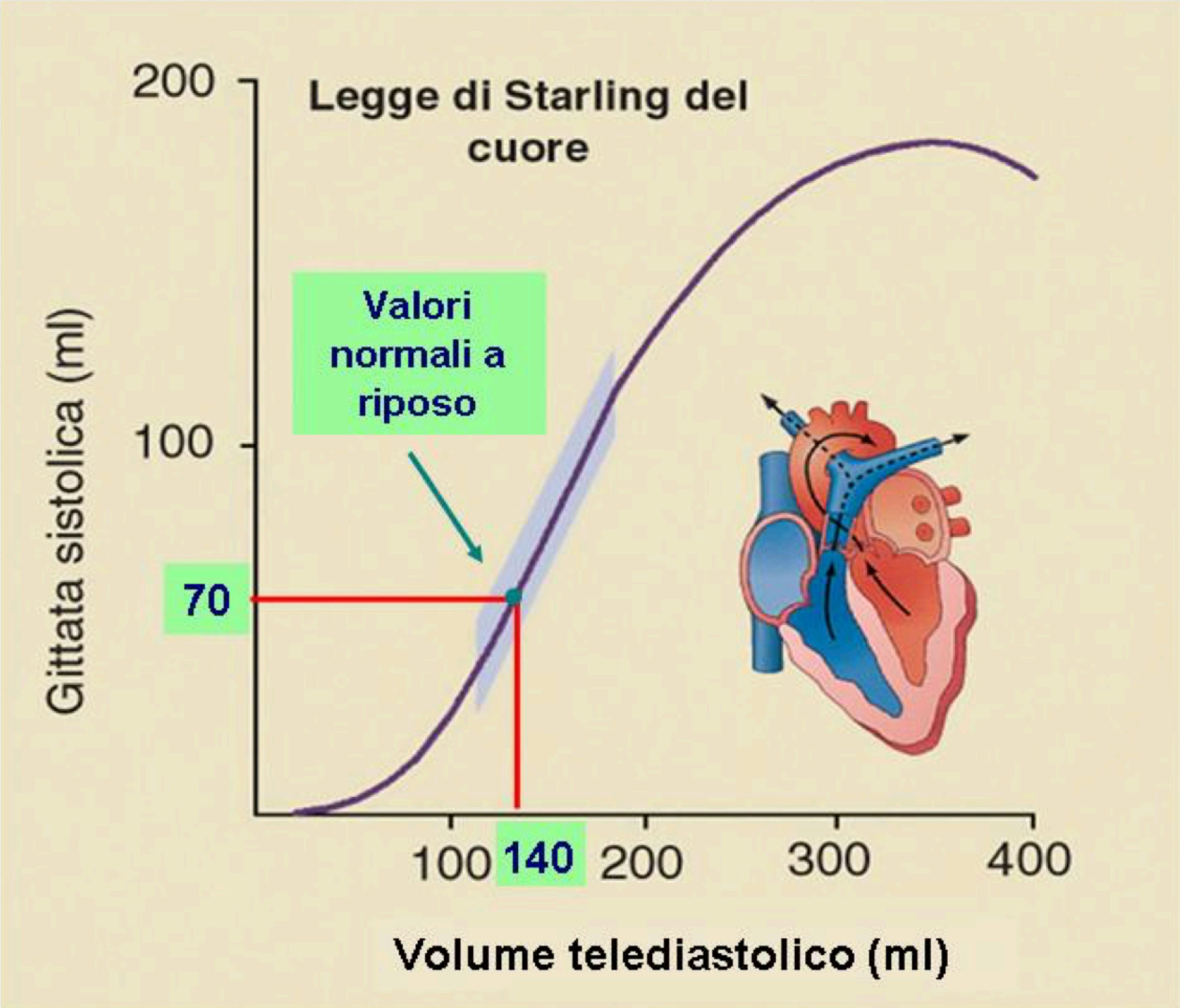
Anestesia&Rianimazione. Ospedale Galeazzi - Sant’Ambrogio

RELAZIONE PRELOAD / SV



RELAZIONE SV / HB / O2

- **DO2I** = [1.39 × **Hb** × SaO_2 + (0.003 × PaO_2)] × CI
- CI = CO/m^2 (m^2= sup. corporea dipendente da cm e kg)
- CO = **SV** x FC

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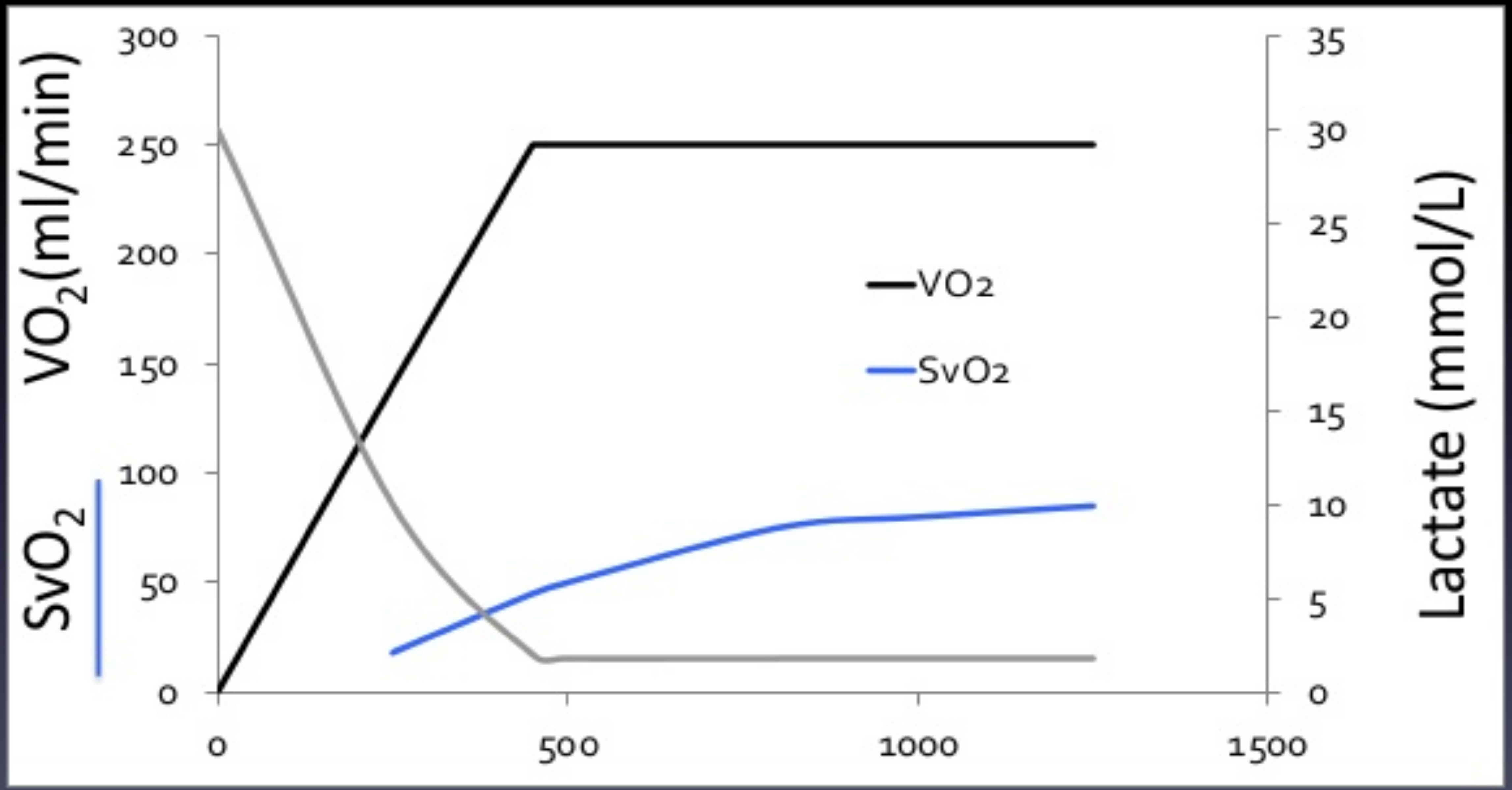
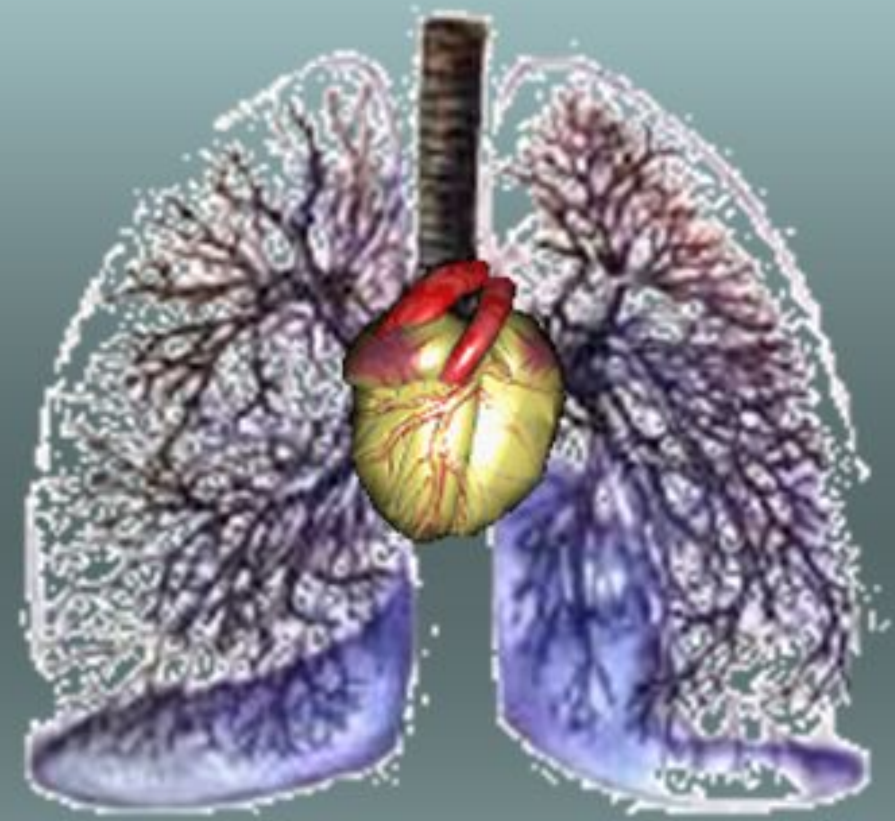
Anestesia&Rianimazione. Ospedale
Galeazzi - Sant'Ambrogio

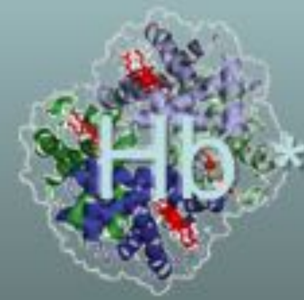
VO_2 DO_2 Relationship:



DO_2(ml/min)

Ossigenazione dei tessuti





Hb * 1,34 * SO_2

*

Gittata Cardiaca

Hb	SO_2	Ct arterioso O_2
15	98 %	20 ml
15	90 %	18.1 ml
10	98 %	13.4 ml
8	90 %	10 ml

MECCANISMI DI COMPENSO

- Aumento della FE
- Aumento della FC

Aumento estrazione tissutale di O_2 (↓ SvO_2)

DO_2I

400 - 650

ml/min/m²

ok

VO_2I

125 - 175

ml/min/m²

ok

↓O2 ER:

- ↑ CO
- Riduz Att Muscolare
- SHUNT periferico
- Ipotermia
- ↑ Hb
- ↑ PaO2
- Tox

O2 ER
20 - 25%
oK

↑O2 ER:

- ↓CO
- ↑VO2
  - Attività Muscolare
  - Att Critica Cerebrale
  - Brivido
  - Ipertermia
- Anemia
- ↓ PaO2

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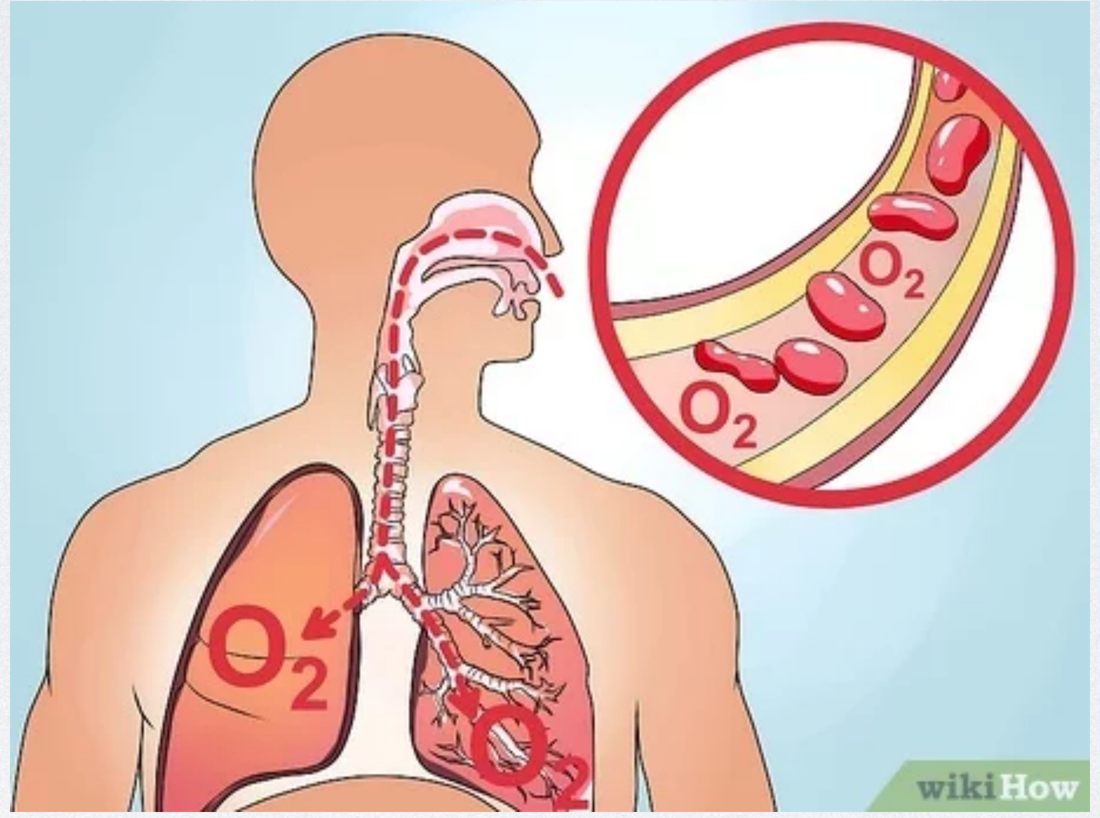
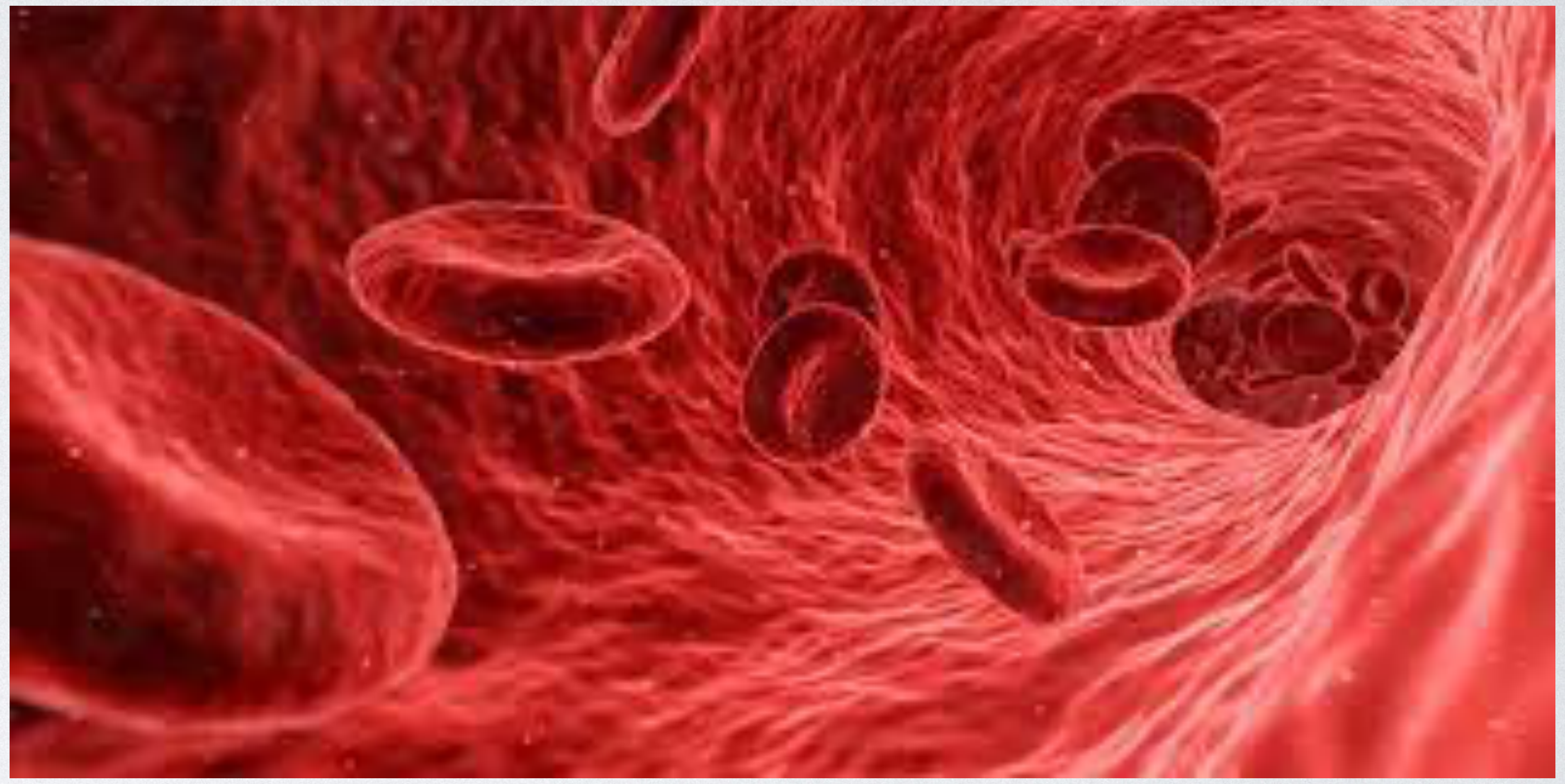
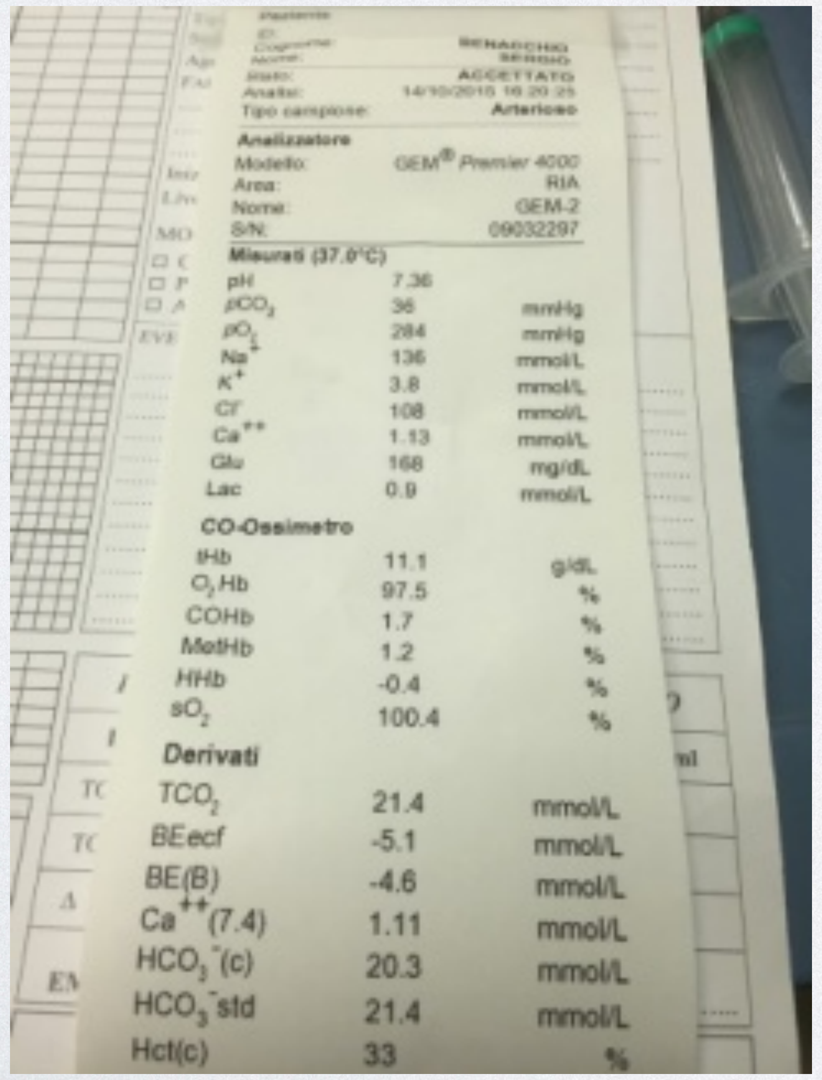
Anestesia&Rianimazione. Ospedale Galeazzi - Sant'Ambrogio

MONITOR + POC + CLINICA + CONTESTO







Stato: ACCETTATO
Analisi: 14/10/2015 16 20 25
Tipo campione: Arterioso

Analizzatore

Modello:	GEM® Premier 4000
Area:	RIA
Nome:	GEM-2
S/N:	09032297

Misurati (37.0°C)

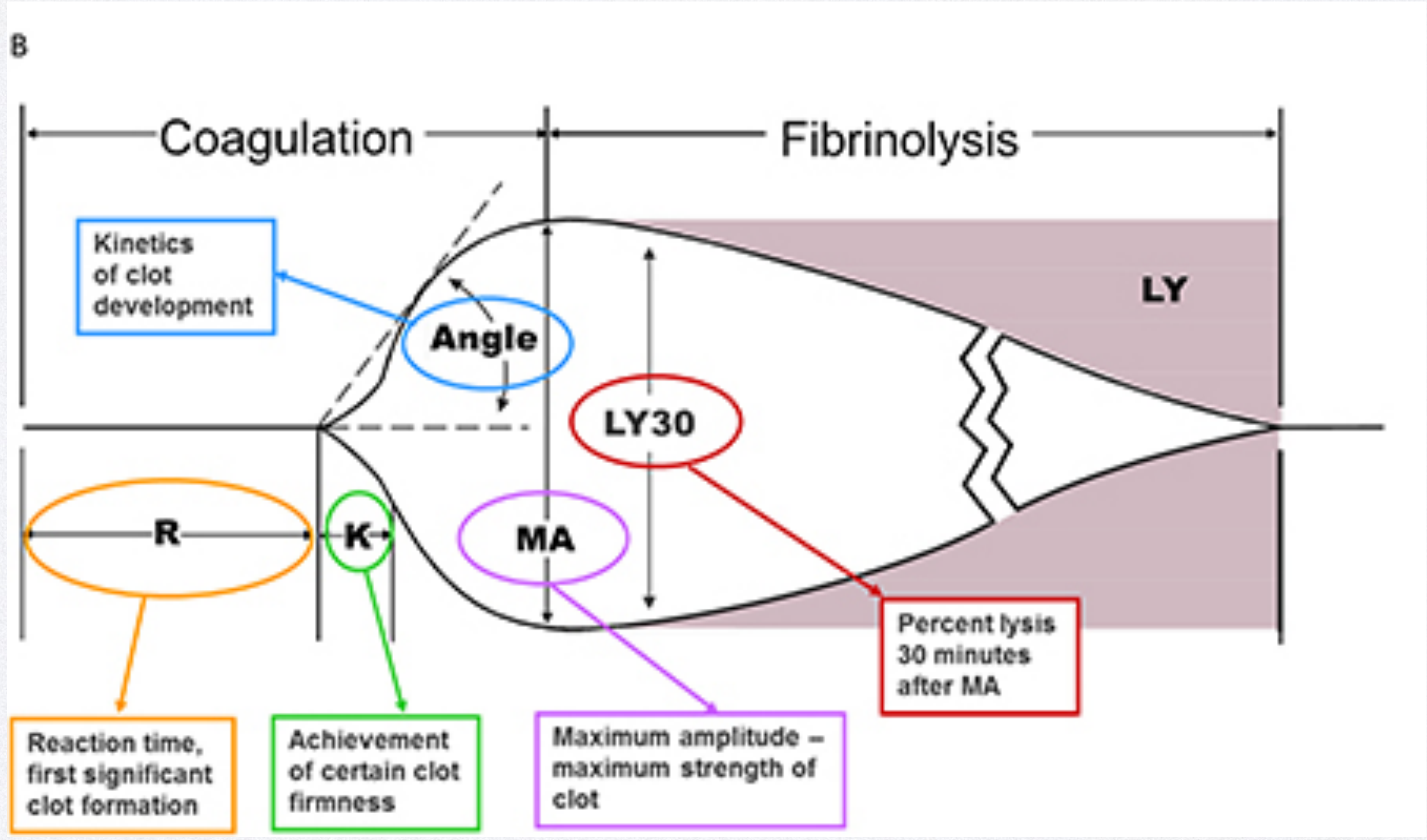
pH	7.36	
pCO_2	36	mmHg
pO_2	284	mmHg
Na^+	136	mmol/L
K^+	3.8	mmol/L
Cl^-	108	mmol/L
Ca^{++}	1.13	mmol/L
Glu	168	mg/dL
Lac	0.9	mmol/L

CO-Ossimetro

tHb	11.1	g/dL
O_2Hb	97.5	%
COHb	1.7	%
MetHb	1.2	%
HHb	-0.4	%
sO_2	100.4	%

Derivati

TCO_2	21.4	mmol/L
BEecf	-5.1	mmol/L
BE(B)	-4.6	mmol/L
$Ca^{++}(7.4)$	1.11	mmol/L
$HCO_3^-(c)$	20.3	mmol/L
HCO_3^-std	21.4	mmol/L
Hct(c)	33	%



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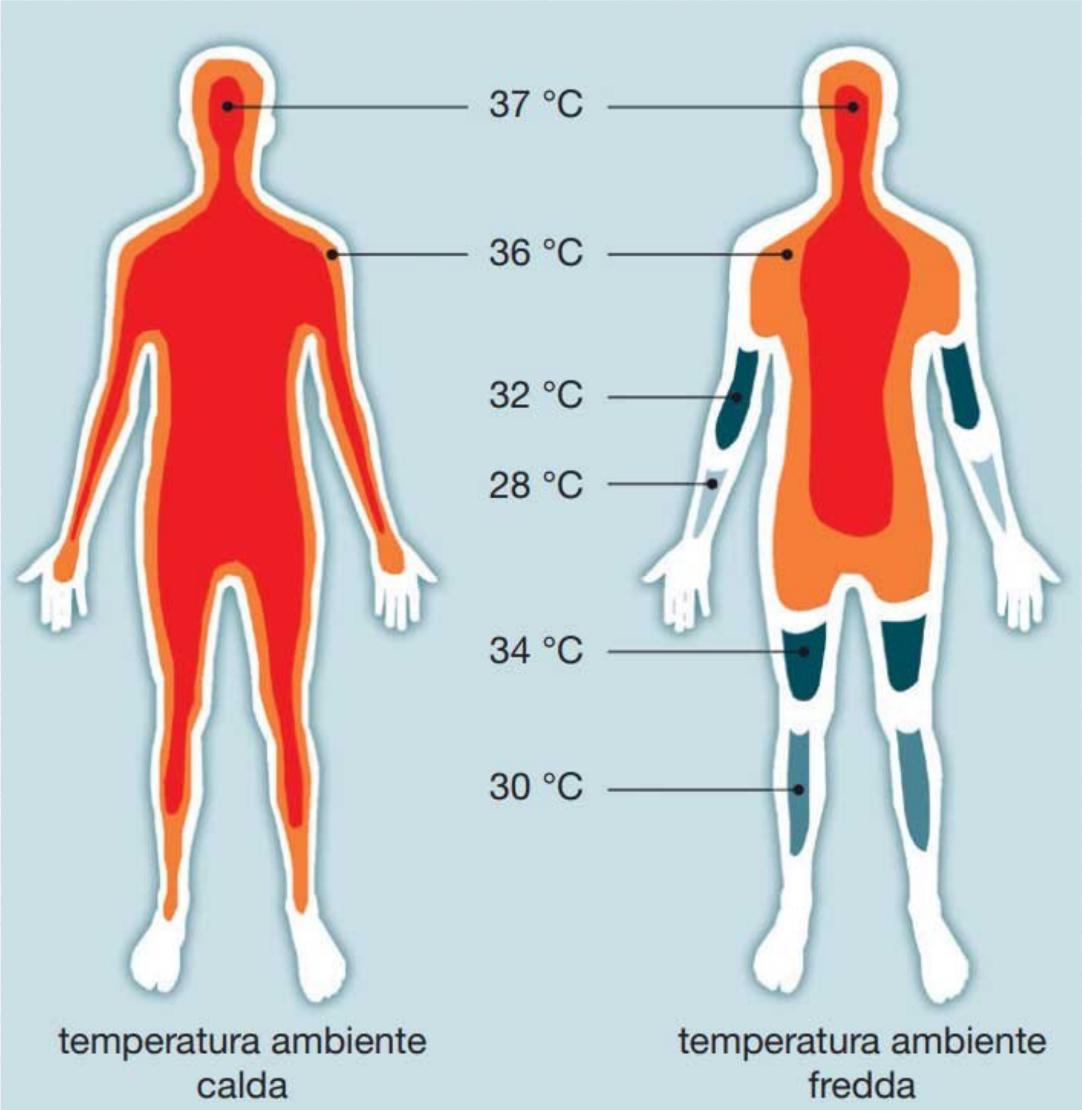
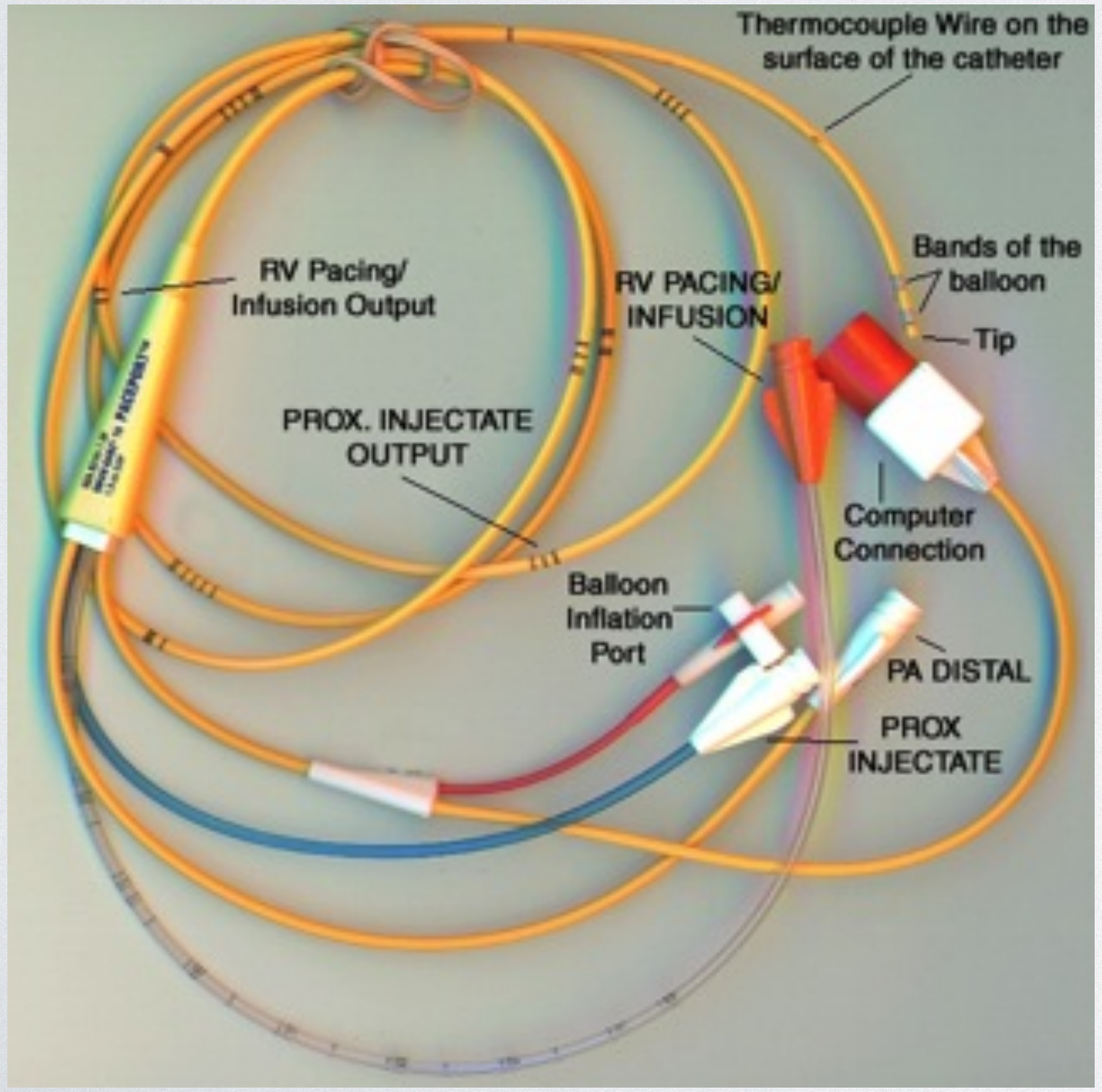
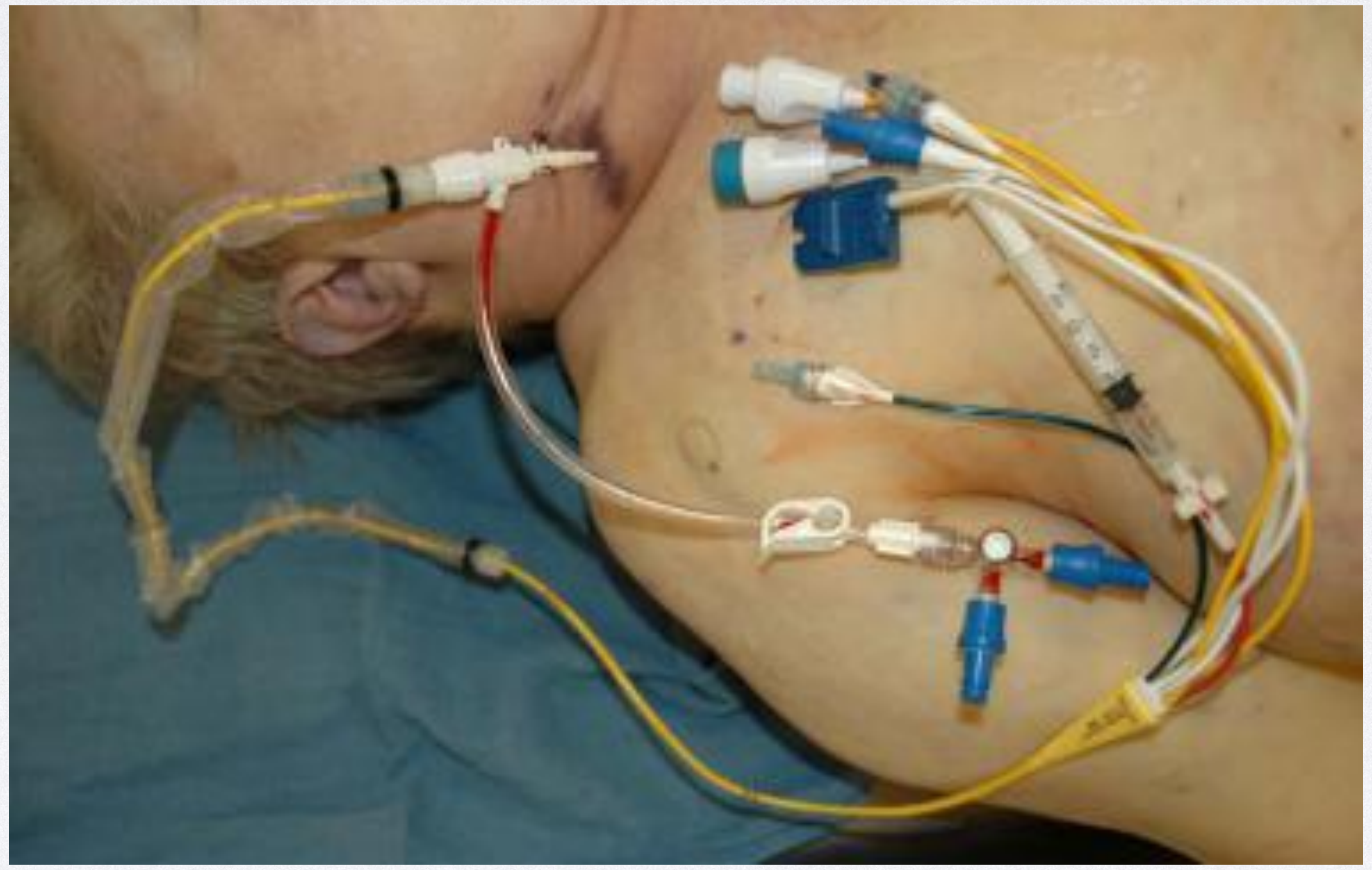
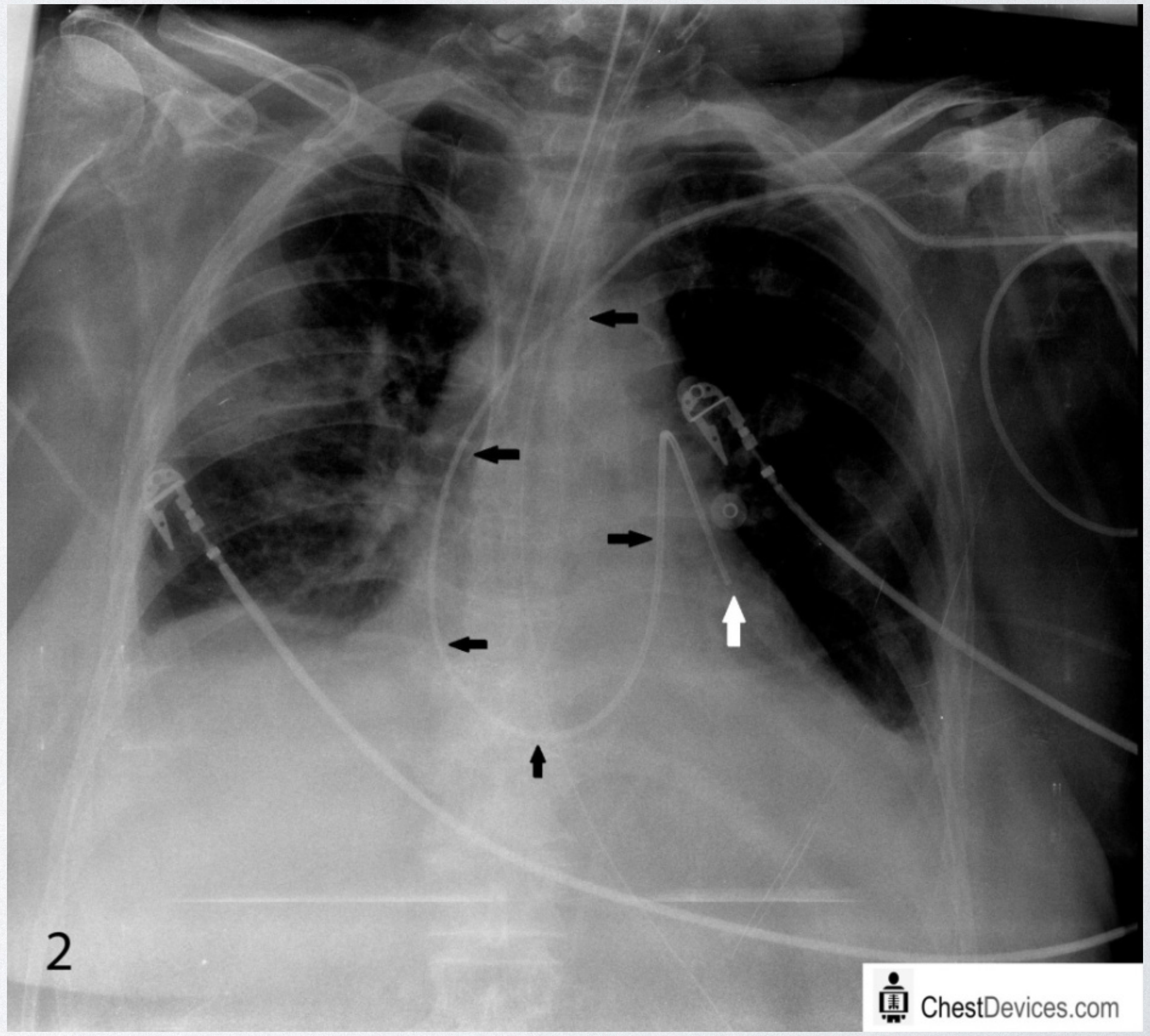
Anestesia&Rianimazione. Ospedale Galeazzi - Sant'Ambrogio



ALTA INVASIVITA’



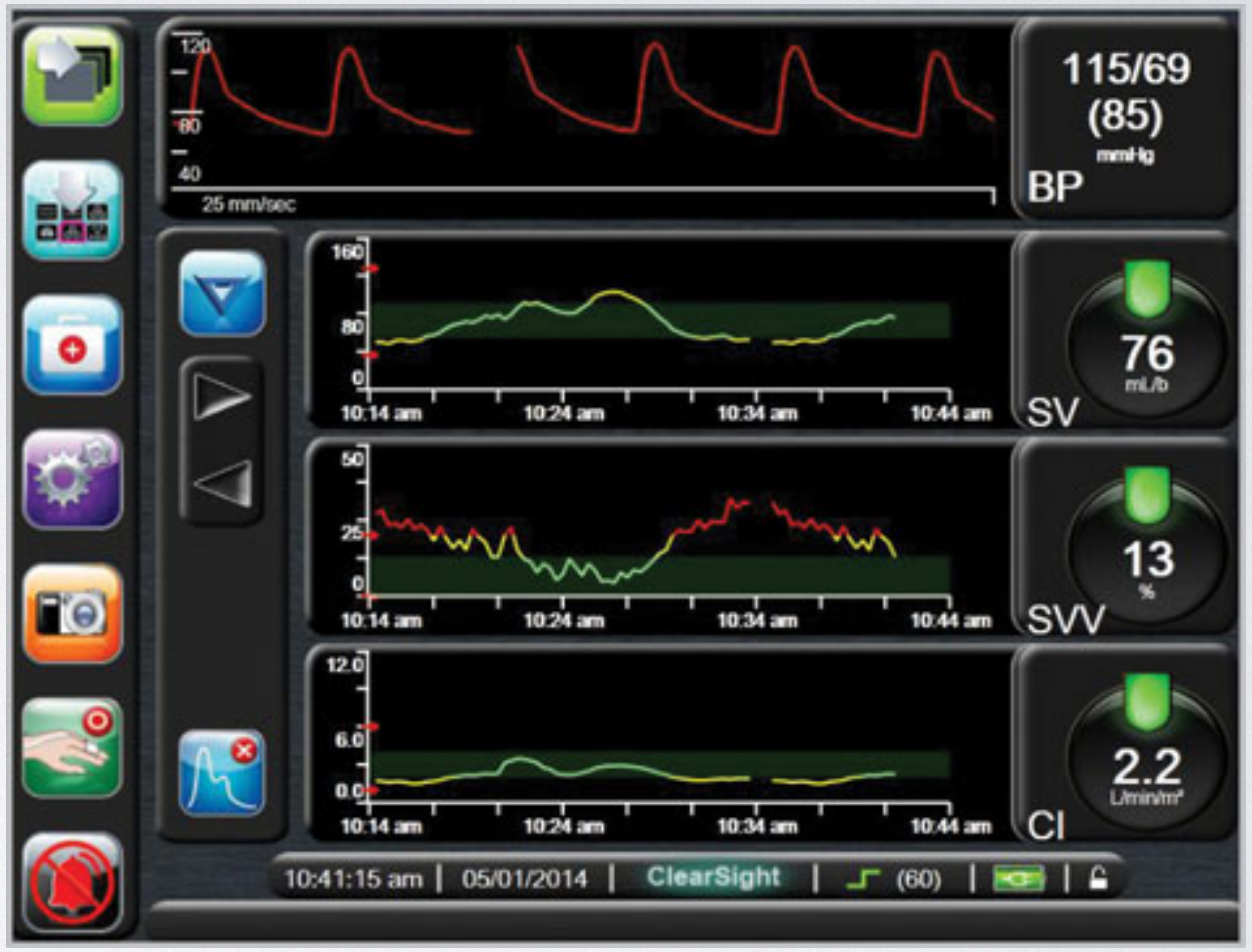




MEDIA EVASIVITÀ’

Dott. Tiziano CRESPI
Anestesia&Rianimazione. Ospedale
Galeazzi - Sant’Ambrogio

BASSA EVASIVITÀ'



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Anestesia&Rianimazione. Ospedale
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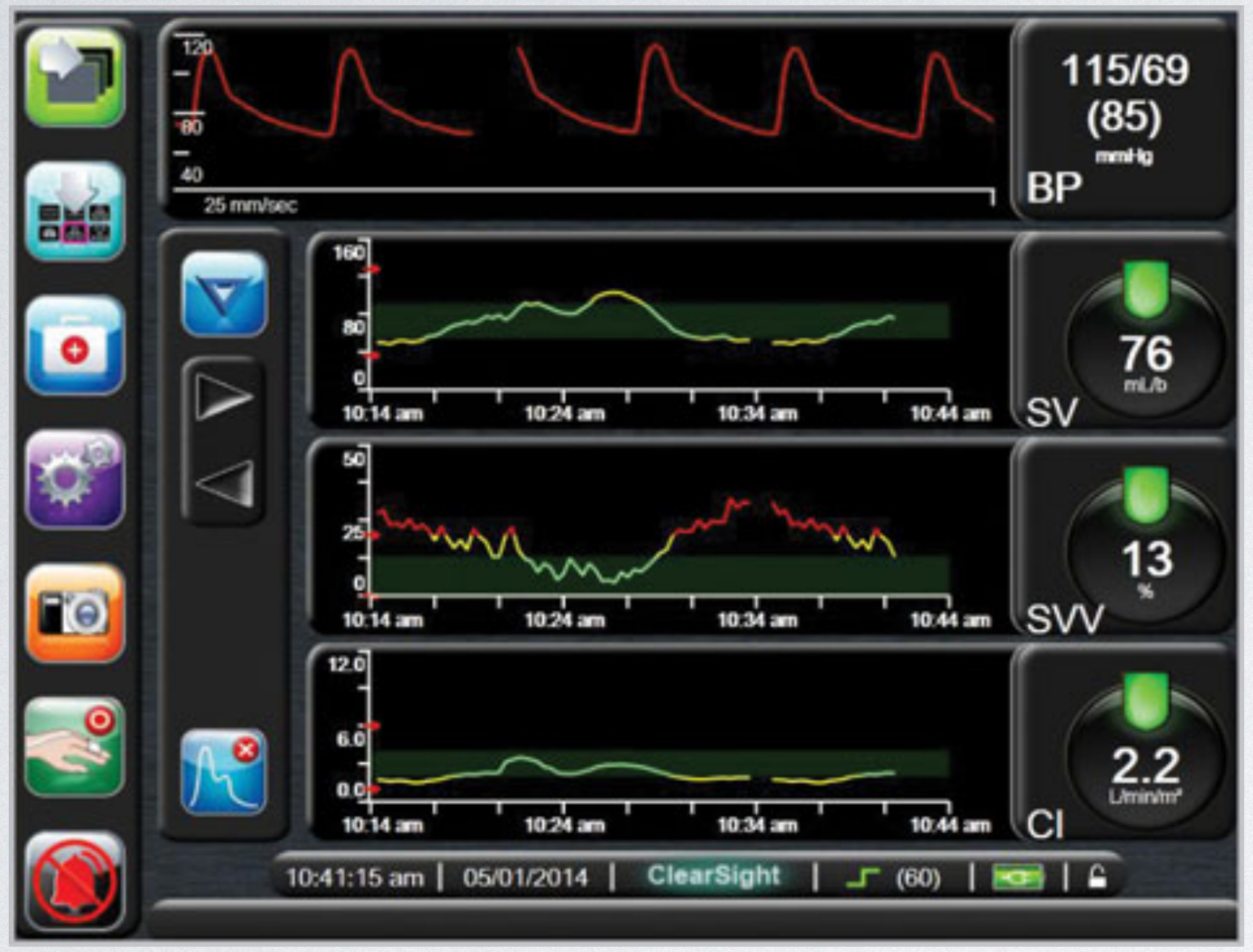
NON INVASIVO



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Anestesia&Rianimazione. Ospedale
Galeazzi - Sant’Ambrogio

PVC?

Conclusions: There are no data to support the widespread practice of using central venous pressure to guide fluid therapy. This approach to fluid resuscitation should be abandoned. (*Crit Care Med* 2013; 41:1774–1781)

Review Articles

Does the Central Venous Pressure Predict Fluid Responsiveness? An Updated Meta-Analysis and a Plea for Some Common Sense*

Paul E. Marik, MD, FCCM[1]; Rodrigo Cavallazzi, MD[2]

Background: Despite a previous meta-analysis that concluded that central venous pressure should not be used to make clinical decisions regarding fluid management, central venous pressure continues to be recommended for this purpose.
Aim: To perform an updated meta-analysis incorporating recent studies that investigated indices predictive of fluid responsiveness. A priori subgroup analysis was planned according to the location where the study was performed (ICU or operating room).
Data Sources: MEDLINE, EMBASE, Cochrane Register of Controlled Trials, and citation review of relevant primary and review articles.
Study Selection: Clinical trials that reported the correlation coefficient or area under the receiver operating characteristic

0.56 (95% CI, 0.54–0.58) for those done in the operating room. The summary correlation coefficient between the baseline central venous pressure and change in stroke volume index/cardiac index was 0.18 (95% CI, 0.1–0.25), being 0.28 (95% CI, 0.16–0.40) in the ICU patients, and 0.11 (95% CI, 0.02–0.21) in the operating room patients.
Conclusions: There are no data to support the widespread practice of using central venous pressure to guide fluid therapy. This approach to fluid resuscitation should be abandoned. (*Crit Care Med* 2013; 41:1774–1781)
Key Words: central venous pressure; fluid challenge; hemodynamic monitoring; meta-analysis; volume responsive

INDICI DINAMICI

Dynamic changes in arterial waveform derived variables and fluid responsiveness in mechanically ventilated patients: A systematic review of the literature*

Paul E. Marik, MD, FCCM; Rodrigo Cavallazzi, MD; Taj

Conclusions: Dynamic changes of arterial waveform-derived variables during mechanical ventilation are highly accurate in predicting volume responsiveness in critically ill patients with an accuracy greater than that of traditional static indices of volume responsiveness. This technique, however, is limited to patients who receive controlled ventilation and who are not breathing spontaneously. (Crit Care Med 2009; 37:2642–2647)

Objectives: A systematic review of the literature to determine the ability of dynamic changes in arterial waveform-derived variables to predict fluid responsiveness and compare these with static indices of fluid responsiveness. The assessment of a patient's intravascular volume is one of the most difficult tasks in critical care medicine. Conventional static hemodynamic variables have proven unreliable as predictors of volume responsiveness. Dynamic changes in systolic pressure, pulse pressure, and stroke volume in patients undergoing mechanical ventilation have emerged as useful techniques to assess volume responsiveness.

Data Sources: MEDLINE, EMBASE, Cochrane Register of Controlled Trials and citation review of relevant primary and review articles.

Study Selection: Clinical studies that evaluated the association between stroke volume variation, pulse pressure variation, and/or stroke volume variation and the change in stroke volume/cardiac index after a fluid or positive end-expiratory pressure challenge.

Data Extraction and Synthesis: Data were abstracted on study design, study size, study setting, patient population, and the correlation coefficient and/or receiver operating characteristic between the baseline systolic pressure variation, stroke volume variation, and/or pulse pressure variation and the change in stroke index/cardiac index after a fluid challenge. When reported, the receiver operating characteristic of the central venous pressure, global end-diastolic volume index, and left ventricular end-diastolic area index were also recorded. Meta-analytic techniques

0.84, and 0.86, respectively, compared with 0.55 for the central venous pressure, 0.56 for the global end-diastolic volume index, and 0.64 for the left ventricular end-diastolic area index. The mean threshold values were 12.5 ± 1.6% for the pulse pressure variation and 11.6 ± 1.9% for the stroke volume variation. The sensitivity, specificity, and diagnostic odds ratio were 0.89, 0.88, and 59.86 for the pulse pressure variation and 0.82, 0.86, and 27.34 for the stroke volume variation, respectively.

Conclusions: Dynamic changes of arterial waveform-derived variables during mechanical ventilation are highly accurate in predicting volume responsiveness in critically ill patients with an accuracy greater than that of traditional static indices of volume responsiveness. This technique, however, is limited to patients who receive controlled ventilation and who are not breathing spontaneously. (Crit Care Med 2009; 37:2642–2647)

KEY WORDS: arterial waveform; pulse pressure variation; stroke volume variation; pulse contour analysis; heart-lung interactions; fluid responsiveness; preload; stroke volume; fluid therapy; hemodynamic monitoring; critical care; systematic review; meta-analysis

British Journal of Anaesthesia **101 (2):** 200–6 (2008)
doi:10.1093/bja/aen133 Advance Access publication June 2, 2008

BJA

Pleth variability index to monitor the respiratory variations in the pulse oximeter plethysmographic waveform amplitude and predict fluid responsiveness in the operating theatre

M. Cannesson[1*†], O. Desebbe[1], P. Rosamel[1], B. Delannoy[1], J. Robin[2], O. Bastien[1] and J.-J. Lehot[1]

[1]*Department of Anaesthesiology and Intensive Care and* [2]*Department of Cardiac Surgery, Hospices Civils de Lyon, Louis Pradel Hospital, Claude Bernard Lyon 1 University, ERI 22 Lyon, France*

**Corresponding author: Service d'Anesthésie Réanimation, Hôpital Cardiologique Louis Pradel, 200 Avenue du Doyen Lépine, 69500 Bron, France. E-mail: maxime_cannesson@hotmail.com*

Table 3 Areas under the ROC curves and cutoff values of various parameters for the prediction of fluid responsiveness. ΔPP, respiratory variations in arterial pulse pressure; ΔPOP, respiratory variations in plethysmographic waveform amplitude; PPV, automated pulse pressure variations; PVI, pleth variability index; CVP, central venous pressure; PCWP, pulmonary capillary wedge pressure; CI, cardiac index; PI, perfusion index

	Area under the curve	Standard error	Asymptomatic 95% confidence interval		*P*-value	Cutoff	Sensitivity (%)	Specificity (%)
			Lower bound	Upper bound				
ΔPP	0.938	0.046	0.847	1.028	<0.001	12.5%	87	89
ΔPOP	0.944	0.042	0.861	1.028	<0.001	12%	87	89
PPV	0.941	0.044	0.854	1.028	<0.001	10.5%	87	89
PVI	0.927	0.051	0.828	1.026	<0.001	14%	81	100
CVP	0.417	0.120	0.182	0.651	0.497	12.5 mm Hg	44	78
PCWP	0.396	0.120	0.161	0.631	0.396	14.5 mm Hg	50	67
CI	0.556	0.118	0.324	0.787	0.651	2.8 litre min^{-1} m^{-2}	44	89
PI	0.438	0.131	0.181	0.694	0.610	1.43%	94	22

$\Delta PP\ (PPV)\ \% =$

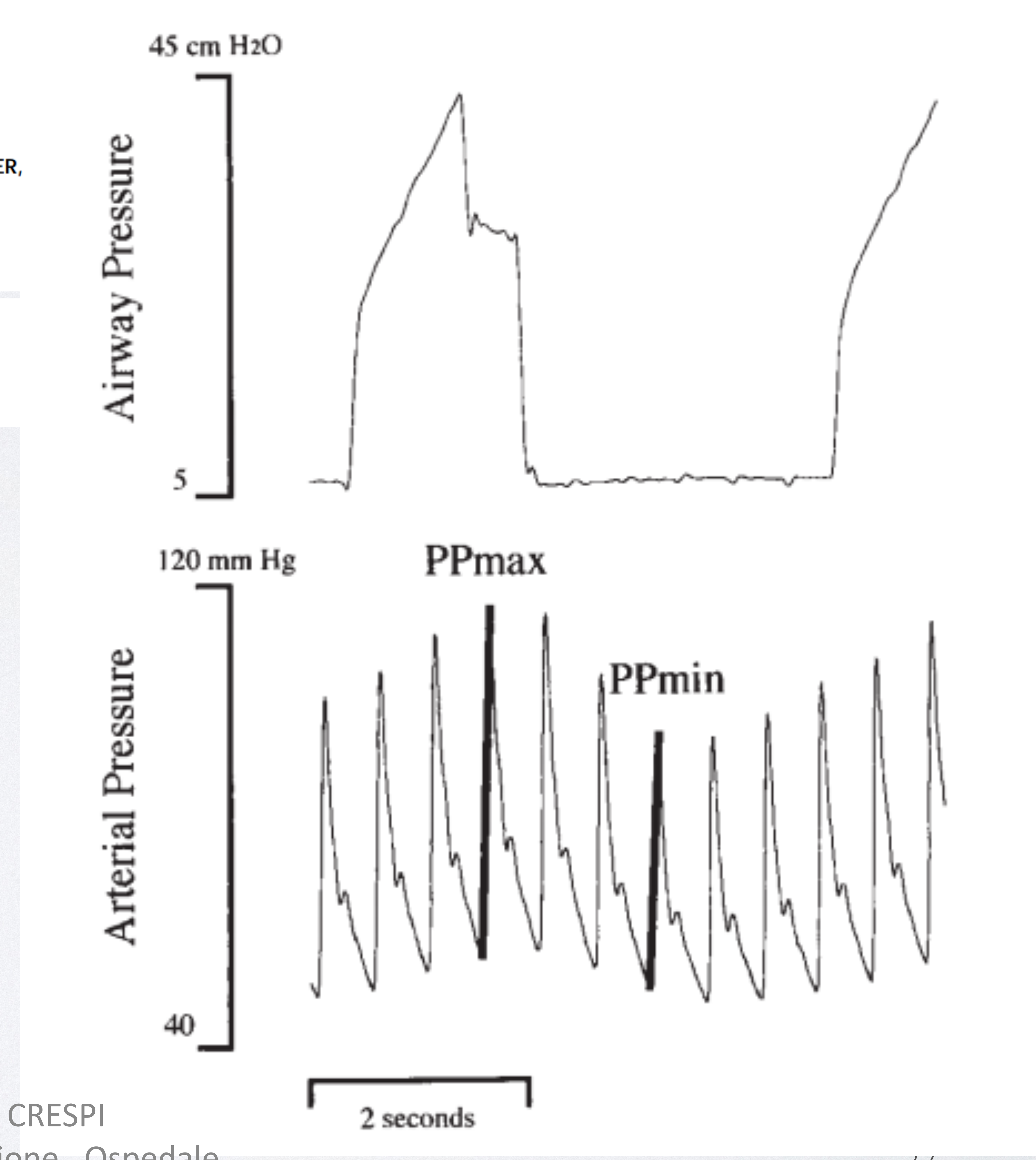
$$= 100 \times (Pp_{max} - Pp_{min}) / [(Pp_{max} + Pp_{min})/2]$$

Relation between Respiratory Changes in Arterial Pulse Pressure and Fluid Responsiveness in Septic Patients with Acute Circulatory Failure

FRÉDÉRIC MICHARD, SANDRINE BOUSSAT, DENIS CHEMLA, NADIA ANGUEL, ALAIN MERCAT, YVES LECARPENTIER, CHRISTIAN RICHARD, MICHAEL R. PINSKY, and JEAN-LOUIS TEBOUL

Service de Réanimation Médicale et Service de Physiologie Cardio-Respiratoire, Centre Hospitalo-Universitaire de Bicêtre, Assistance Publique-Hopitaux de Paris, Le Kremlin Bicêtre, Université Paris XI, Paris, France; INSERM U451-LOA-ENSTA-Ecole Polytechnique, Palaiseau, France; and Division of Critical Care Medicine, University of Pittsburgh, Pittsburgh, Pennsylvania

Am J Respir Crit Care Med Vol 162. pp 134–138, 2000
Internet address: www.atsjournals.org



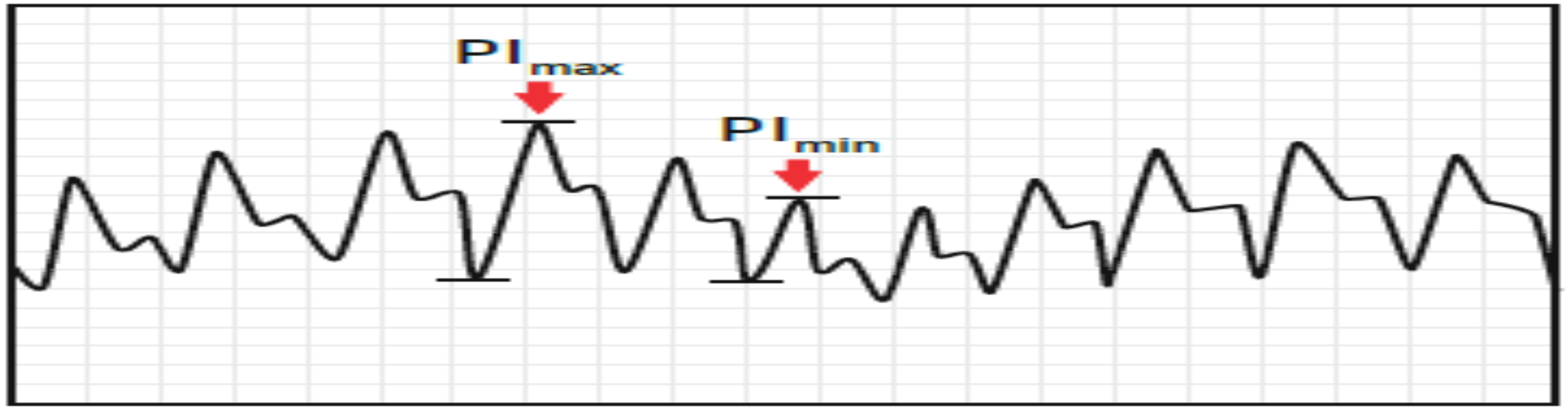
$\Delta PP > 10.5$ = Pz FLUID RESPONDER
PPV > 12.5 = Pz FLUID RESPONDER

CALCOLO DEL PVI: COME FUNZIONA

L'indice di perfusione (IP) è il rapporto tra flusso di sangue pulsante e non pulsante attraverso il letto capillare periferico. Il PVI è una misura automatica della variazione dinamica di IP che si verifica durante il ciclo respiratorio.

$$PVI = \frac{PI_{max} - PI_{min}}{PI_{max}} \times 100$$

La probabilità che il paziente risponda alla somministrazione di fluidi è proporzionale al PVI.



- Un valore del PVI > 14% prima dell'espansione volemica è predittivo di una risposta del paziente alla somministrazione di fluidi (sensibilità, 81%)
- Un valore del PVI < 14% prima dell'espansione volemica è predittivo di una mancata risposta del paziente alla somministrazione di fluidi (specificità, 100%)

INDICI DINAMICI MA…(1)

- Ritmo Sinusale
- Ventilazione meccanica controllata
- TV 8-10 ml/kg (peso ideale)

INDICI DINAMICI MA… (2)

- Aritmia
- Cuore polmonare o insufficienza cardiaca destra
- Ipertensione addominale
- Toracotomia
- Ridotta compliance polmonare
- Repentine variazioni di PA
- Dolore (> PVI)
- Ipotermia (per PVI)
- Vasocostrizione (per PVI)

ATTENZIONE (1): PRONE POSITION

- PRECARICO & INOTROPISMO
  - **PPV** >10.5 % (**≥15% prone position**) = RESPONDER TO FLUID CHALLANGE in MECHANICAL VENTILATION
  - **SVV** > 10% (**≥ 14% prone position**) = RESPONDER TO FLUID CHALLANGE in MECHANICAL VENTILATION

MICROCIRCOLO I

- Disfunzione microcircolo e mitocondriale:
  sepsi ≈ HIGH RISK SURGERY

MICROCIRCOLO II

- PRIMARY GOAL: ottimizzazione perfusione tessutale;
- Ha impatto su outcome;
- Ne si assume l'adeguatezza attraverso parametri sistemici (PA, CI, ScVO2…);
- Alterazione Densità capillare ed eterogeneità perf. cap.

MICROCIRCOLO III

- CLINICA: insuff

- LATTATI: discreto surrogato. Risp.ipox., produz. Aerobica da mediatori flogistici, da risp adrenergica, da ridur clearance;

MICROCIRCOLO IV

- TECNICHE STRUMENTALI
  - **Laserdoppler**: misurazione cutanea di flusso aggregato, no valore assoluto ma variazioni risp basale —> no MALE/BENE ma MEGLIO O PEGGIO.
  - **Videomicroscopia**: illuminazione tessutale profonda a lunghezza d'onda 530 nm, applicabile solo a tessuti rivestiti da sottile strato epiteliale (mucose, encefalo, fegato). Perde gli strati più profondi.
  - **Misurazione PO2 con elettrodi Clarke**: sensibilità per massimi valori O2, perde l'eterogeneità.
  - ***<u>Misurazione Sat O2 NIRS</u>***: saturazione Hb venosa regionale, perde l'eterogeneità.
  - **Test di occlusione con Laserdoppler o NIRS**: velocità recupero flusso/SO2 determinata da reattività endoteliale e reclutabilità arteriolare e capillare.
  - **Misurazione CO2 tessutale e venosa**: PCO2 GAP (=PCO2-PaCO2); PCO2 misurata con elettrodi, fotometria o metodiche transcutanee. Produzione e flusso di CO2 .PCO2 GAP inversamente proporzionale a perfusione capillare.

MICROCIRCOLO V

- Regionalità del distretto indagato
- Impiego non routinario
- No monitoraggio continuo
- Nessun ENDPOINT definito e validato

MONITORARE

- Rilevazione periodica e sistematica DEI PARAMETRI allo scopo di controllare la situazione o l'andamento di un sistema complesso.

ATTENZIONE (2)

- Non esiste "IL PARAMETRO"
- Esistono DIVERSI SINGOLI PARAMETRI, NON TUTTI SONO RIPORTATI SU UN MONITOR, TUTTI VANNO CONTESTUALIZZATI.
- **PROSPETTIVA e TOLLERANZA: Ciò che è ottimale adesso può aver ripercussioni dopo.**

FINE

Prof. Andrea DE GASPERI