Techniques available to support physiological processes in life-threatening states

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Physiologic monitoring of the critically ill patients (1)

- arterial blood pressure
- heart rate (HR)
- hematocrite and haemoglobin concentrations (Hct and Hgb)
- urine output
- electrocardiogram (ECG)
- central venous pressure (CVP)
- arterial blood gases
- pulmonary arterial and precapillary wedge pressure (WP)
- cardiac output (CO) and hemodynamics variables, O2 delivery (DO2) and consumption (VO2)
- Oxygen transport variables
Physiologic monitoring of the critically ill patients (2)

- Tidal volume (TV), respiratory rate (f), minute volume (MV)
- End-tidal CO2, VT/VD
- expiratory oxygen
- transcutaneous oxygen and carbon dioxide

- blood volume, plasma volume

- electroencephalograph (EEG)
- intracranial pressure
Heart rate

• The number of ventricular contractions per minute
Adults

60 – 100
<table>
<thead>
<tr>
<th>Age Group</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonates</td>
<td>120 – 140</td>
</tr>
<tr>
<td>&lt;1 year</td>
<td>110 – 160</td>
</tr>
<tr>
<td>2 – 5 y</td>
<td>95 – 140</td>
</tr>
<tr>
<td>5 – 12 y</td>
<td>80 – 120</td>
</tr>
<tr>
<td>&gt;12 y</td>
<td>60 – 100</td>
</tr>
</tbody>
</table>
tachycardia

- Excessive rapidity of the heart’s action
Bradycardia

• Slowness of the heartbeat
Pulse

- The regularly recurrent palpable wave of distension in an artery due to blood ejected with each cardiac contraction
PULSE OXIMETRY

- Uses spectrophotometry
- Differentiates oxy- from deoxyhemoglobin by the differences in absorption at 660nm and 940nm
- Minimizes tissue interference by separating out the pulsatile signal
- Estimates heart rate by measuring cyclic changes in light transmission
- Measures 4 types of hemoglobin: deoxy, oxy, carboxy, and met
- Estimates functional hemoglobin saturation: oxyhemoglobin/deoxy + oxy
\[ \text{O}_2 \text{ saturation} \]

\[ \text{SaO}_2 = \frac{\text{O}_2\text{Hb}}{\text{RHb} + \text{O}_2\text{Hb}} \]
Limitation of pulsoximetry

- Weak pulsation
- Hypothermia
- Hypotension
- Increased level of carboxyhemoglobin
- Shivering
- anaemia
2.4-4.0 L/min/m² 40-70 mL/m²

Cardiac output = Stroke volume x Heart rate

Determinants of cardiac output:
- Preload
- Afterload
- Myocardial contractility
Preload

- The force applied to ventricular muscle in diastole just before contraction
Afterload

- Pressure that the ventricles must exceed to eject blood
PRELOAD / AFTERLOAD
Common indications for Central Venous Cannulation

- Measurement CVP
- Rapid administration of fluids and blood
- Insertion of pulmonary artery catheter
- Insertion of transvenous pacemaker
- Parenteral alimentation
- Temporary single cannula hemodialysis
- Long-term chemotherapy
- Administration of drugs which cause sclerosis of peripheral veins
Right internal jugular vein cannulation
Right internal jugular vein cannulation
CVP

- Cardiac function
- Adequacy of vascular volume
Contraindications for subclavian and internal jugular central venous catheterisation (1)

A. Absolute and general
   • Conditions of severe bleeding tendency and coagulopathy
   • Persistent shock
   • Obstruction of the superior vena cava, innominate or subclavian vein and previously failed attempts
   • Respiratory distress
   • Injury to the superior vena cava, innominate, or subclavian vein
   • Patient’s refusal
Contraindications for subclavian and internal jugular central venous catheterisation (2)

B. Relative and specific
- in process of CPR
- restless and uncooperative patient
- infection, burn or presence of cancerous nodes in the area
- tracheostomy with copious secretions
- left supraclavicular subclavian puncture in cirrhotic
- infraclavicular puncture when costoclavicular space is very narrow
- in severe hypertension, tortuosity of the arteries and proximity of aneurysm
- when attempt in one side has resulted in a severe complication
- when sterile technique cannot be observed
- lack of experience of the physician and no expert supervision
Complications of central venous catheters from percutaneous insertion and long-term maintenance (1)

A. Subclavian and internal Jugular vein
   - puncture of the pleural dome
   - puncture of lung
   - pneumothorax
   - hemothorax and hemomediastinum
   - puncture of lymphatic ducts, lymphorrhagia, chylomediastinum and chylothorax
   - catheter in pleural cavity and infusion of fluids and blood in the pleural space
Complications of central venous catheters from percutaneous insertion and long-term maintenance (2)

B. Subclavian Veins
   - Injury to brachial plexus, directly by the needle or indirectly by the pressure of an arterial hematoma
   - Injury to the clavicular or first rib periosteum, cartilage or ligaments and osteitis

C. Internal Jugular
   - Injury to the vagus nerve, phrenic nerve, stellate ganglion, and cervical plexus
   - Brain infarction due to injury to the carotid artery
   - Injury to the trachea and esophagus
   - Injury to transverse processes of the cervical vertebrae with consequent reactions and infections of periosteum and bone
Complications of central venous catheters from percutaneous insertion and long-term maintenance (3)

D. Common to all venipuncture techniques and Long-Term catheters

- inadvertent arterial puncture, soft tissue haematoma, false aneurysma, and arteriovenous fistula
- Detour of the catheter into a small peripheral branch
- Catheter tip in opposition to a wall and wall perforation
- Catheter advanced too far
- Catheter short of the desirable length or retraction of catheter tip
- Catheter tip against the blood flow
- Clot and fibrinous sleeve formation
- Thrombosis of the vein and embolism
- Catheter related infections
- Guide wire embolus
- Bleeding accidental removal of catheters
Mechanical complications of central venous catheters
Lewis A. Eisen, MD et al.

- 385 consecutive central venous catheter (CVC) attempts over a 6-month period in critically ill patients
- 67% free of complications
- Excluding placement failure -> 86% free of complications
- The complication rate increased with the number of percutaneous punctures, with a rate of 54% when more than 2 punctures were required.
Complications & Catheter Insertion Site

Table 2. Mechanical Complications by Catheter Insertion Site

<table>
<thead>
<tr>
<th></th>
<th>All Catheters (n = 385)</th>
<th>Subclavian (n = 218)</th>
<th>Internal Jugular (n = 40)</th>
<th>Femoral (n=127)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complications</td>
<td>129 (33.2)</td>
<td>85 (39.0)</td>
<td>13 (32.5)</td>
<td>31 (24.4)</td>
<td>.022</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>5 (1.3)</td>
<td>5 (2.3)</td>
<td>0</td>
<td>N/A</td>
<td>.144</td>
</tr>
<tr>
<td>(n = 258)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arterial puncture</td>
<td>18 (4.7)</td>
<td>7 (3.2)</td>
<td>2 (5.0)</td>
<td>9 (7.1)</td>
<td>.257</td>
</tr>
<tr>
<td>Incorrect position</td>
<td>14 (3.6)</td>
<td>14 (6.4)</td>
<td>0</td>
<td>0</td>
<td>.004</td>
</tr>
<tr>
<td>Hemothorax</td>
<td>1 (0.3)</td>
<td>1 (0.5)</td>
<td>0</td>
<td>N/A</td>
<td>.361</td>
</tr>
<tr>
<td>(n = 258)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subcutaneous hematoma</td>
<td>3 (0.8)</td>
<td>1 (0.5)</td>
<td>0</td>
<td>2 (1.6)</td>
<td>.440</td>
</tr>
<tr>
<td>Death</td>
<td>1 (0.3)</td>
<td>0</td>
<td>1 (2.5)</td>
<td>0</td>
<td>.361</td>
</tr>
<tr>
<td>Failure to place</td>
<td>86 (22.3)</td>
<td>57 (26.1)</td>
<td>10 (25.0)</td>
<td>19 (15.0)</td>
<td>.051</td>
</tr>
</tbody>
</table>
Conclusion from this article

- Failure to place catheter is common, but other complications are unusual.
- Subclavian route carries higher risk of mechanical complications
- Residents and interns could place it safely when supervised properly
- More than 2 percutaneous punctures is related with higher incidence of mechanical complications
Recommendation on mechanical complications

- Recognize risk factors for difficult catheterization
- Seek assistance from an experienced clinician
- Avoid femoral venous catheterization
- Use ultrasound guidance during internal jugular catheterization

The Swan-Ganz catheter

Measurement of cardiac output by thermodilution technique with injection of cold indicator into the right atrium and the definition of the temperature change in the pulmonary artery
<table>
<thead>
<tr>
<th>Pressure</th>
<th>Right atrium</th>
<th>Right ventricle</th>
<th>Pulmonary artery</th>
<th>Pulmonary capillary wedge</th>
</tr>
</thead>
<tbody>
<tr>
<td>30 mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20 mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0 mm Hg</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Flow-directed catheter
Table 16-6. COMPLICATIONS OF 281 PULMONARY ARTERY CATHETERS, JULY, 1982 THROUGH JANUARY, 1986

<table>
<thead>
<tr>
<th>Complications</th>
<th>Number</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infections</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Skin Site</td>
<td>9</td>
<td>3.2</td>
</tr>
<tr>
<td>Catheter</td>
<td>10</td>
<td>3.5</td>
</tr>
<tr>
<td>Subtotal</td>
<td>19</td>
<td>6.7</td>
</tr>
<tr>
<td>Cardiac Arrhythmias</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Transient, requiring no therapy</td>
<td>54</td>
<td>19.2</td>
</tr>
<tr>
<td>Persistent, requiring therapy</td>
<td>6</td>
<td>2.1</td>
</tr>
<tr>
<td>Subtotal</td>
<td>60</td>
<td>21.3</td>
</tr>
<tr>
<td>Pulmonary Infarction</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>Complete A-V Block</td>
<td>2</td>
<td>0.7</td>
</tr>
<tr>
<td>Catheter Knotting</td>
<td>2</td>
<td>0.7</td>
</tr>
<tr>
<td>Failure to Catheterize</td>
<td>5</td>
<td>1.7</td>
</tr>
<tr>
<td>Cardiac Arrest</td>
<td>1</td>
<td>0.3</td>
</tr>
<tr>
<td>TOTAL</td>
<td>90</td>
<td>32.0</td>
</tr>
</tbody>
</table>
### TABLE 34-3. Hemodynamic Variables: Calculations and Normal Values

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>CALCULATION</th>
<th>NORMAL VALUES</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac index (CI)</td>
<td>$\frac{CO}{BSA}$</td>
<td>2.5–4.0 l·min$^{-1}$·$m^{-2}$</td>
</tr>
<tr>
<td>Stroke volume (SV)</td>
<td>$\frac{CO \times 1000}{HR}$</td>
<td>60–90 ml·beat$^{-1}$</td>
</tr>
<tr>
<td>Stroke index (SI)</td>
<td>$\frac{SV}{BSA}$</td>
<td>40–60 ml·beat$^{-1}$·$m^{-2}$</td>
</tr>
<tr>
<td>Mean arterial pressure (MAP)</td>
<td>Diastolic pressure + $\frac{1}{3}$ pulse pressure</td>
<td>80–120 mm Hg</td>
</tr>
<tr>
<td>Systemic vascular resistance (SVR)</td>
<td>$\frac{MAP - CVP}{CO} \times 79.9$</td>
<td>1200–1500 dynes-cm·sec$^{-5}$</td>
</tr>
<tr>
<td>Pulmonary vascular resistance (PVR)</td>
<td>$\frac{PAP - PWP}{CO} \times 79.9$</td>
<td>100–300 dynes-cm·sec$^{-5}$</td>
</tr>
<tr>
<td>Right ventricular stroke work index (RVSWI)</td>
<td>$0.0136 (PAP - CVP) \times SI$</td>
<td>5–9 g·m·beat$^{-1}$·$m^{-2}$</td>
</tr>
<tr>
<td>Left ventricular stroke work index (LVSWI)</td>
<td>$0.0136 (MAP - PWP) \times SI$</td>
<td>45–60 g·m·beat$^{-1}$·$m^{-2}$</td>
</tr>
</tbody>
</table>

$CVP$ = mean central venous pressure; $BSA$ = body surface area; $CO$ = cardiac output; $PAP$ = mean pulmonary artery pressure; $PWP$ = pulmonary wedge pressure; $MAP$ = mean arterial blood pressure; $g$·$m$ = gram meter; sec$^{-5}$ = seconds$^{-5}$. 
<table>
<thead>
<tr>
<th></th>
<th>(kPa)</th>
<th>(mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right atrium</td>
<td>0.6</td>
<td>4</td>
</tr>
<tr>
<td>Right ventricle</td>
<td>3.8/0.6</td>
<td>28/4</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>3.3/1.8</td>
<td>25/12</td>
</tr>
<tr>
<td>Left atrium</td>
<td>1.1</td>
<td>8</td>
</tr>
<tr>
<td>Left ventricle</td>
<td>17.3/1.1</td>
<td>130/8</td>
</tr>
<tr>
<td>Aorta</td>
<td>17.3/9.3</td>
<td>130/70</td>
</tr>
</tbody>
</table>
Arterial pressure

- Systolic – culmination of the ejection phase of aortic contraction
- Diastolic – the nadir of aortic blood pressure, which occurs after aortic valve closure
- Mean – diastolic + 1/3(systolic – diastolic)
Monitoring of arterial pressure

• noninvasive
• invasive
noninvasive

- Korotkoff’s (auscultation)
- Riva-Rocci (palpation)
invasive

- Cannulation site
  - radial artery
  - dorsalis pedis
Hazards of invasive blood pressure monitoring

- vascular compromise
- disconnection
- accidental injection
- infection
- damage to nearby nerves
radial artery

- the vessel is superficial
- the cannulation site is accessible during most types of surgery
- the procedure is reasonably pain-free
- inducing distal vascular ischaemia is quite low
- diameter lumen is relatively small
- is so far distal
- could be frequently occluded by thrombus
dorsalis pedis artery

- when the place on the upper extremities is inaccessible e.g. burns, trauma, previous arterial catheterization
Allen test
Continuous monitoring of blood pressure. A cannula placed percutaneously in an artery is connected to a pressure transducer through a fluid filled non-compliant manometer line incorporating a continuous and intermittent flush device. Adapted from Hinds CJ, Watson D. *Intensive care: a concise textbook*. WB Saunders, 1996.
Ryc. 2 – Dwie metody zerowania układu. Zauważ, że miejsce w którym otwieramy układ do atmosfery zawsze powinno być na wysokości linii pachowej środkowej.
### Table 44-1

Normal Pressures in the Systemic Circulation

<table>
<thead>
<tr>
<th></th>
<th>Mean Value (mmHg)</th>
<th>Range (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic blood pressure*</td>
<td>120</td>
<td>90–140</td>
</tr>
<tr>
<td>Diastolic blood pressure*</td>
<td>80</td>
<td>70–90</td>
</tr>
<tr>
<td>Mean arterial pressure</td>
<td>92</td>
<td>77–97</td>
</tr>
<tr>
<td>Left ventricular end-diastolic pressure</td>
<td>6</td>
<td>0–12</td>
</tr>
<tr>
<td>Left atrium</td>
<td></td>
<td></td>
</tr>
<tr>
<td>a wave</td>
<td>10</td>
<td>4–16</td>
</tr>
<tr>
<td>v wave</td>
<td>13</td>
<td>6–20</td>
</tr>
<tr>
<td>Right atrium</td>
<td></td>
<td></td>
</tr>
<tr>
<td>a wave</td>
<td>5</td>
<td>3–8</td>
</tr>
<tr>
<td>c wave</td>
<td>6</td>
<td>2–10</td>
</tr>
<tr>
<td>v wave</td>
<td>3</td>
<td>0–8</td>
</tr>
</tbody>
</table>

* Measured in radial artery
Shock

Physiologic causes of shock:

- **pump failure** - the heart can be damaged by intrinsic muscular disease or injury, so that it fails to act properly as a pump. It does not generate sufficient energy to move blood through the system.

- **Relative hypovolemia** - the blood vessels constituting the container can dilate so that the blood within them, even though it is of normal volume, is insufficient to fill the system and provide efficient perfusion.

- **Hypovolemia** - blood or plasma can be lost.
Shock

- cardiogenic shock
- distributeshock (relative hypovoleemia)
- hypovolemic shock (oligovolemic)
Advanced classification of shock includes four types

cardiogenic
• -acute myocardial infarction
• -small cardiac output syndrome
• -arrhythmias
• -cardiomyopathia
distribute
• -septic
• -anaphylactic
• -neurogenic
obturate
• -cardiac tamponade
• -massive pulmonary embolism
• -acute pulmonary hypertension
oligovolemic
• -haemorrhagic shock (loss of blood)
• loss of fluids (severe diarrhoea, vomiting, burns)
• **Sepsis**
  • The systemic inflammatory response to infection

• **Severe sepsis**
  • Sepsis associated with organ dysfunction, hypoperfusion or hypotension

• **Septic shock**
  • Defined as sepsis induced hypotension despite adequate fluid resuscitation along with the presence of perfusion abnormalities
Clinical manifestations of hypoperfusion

- pallor
- tachycardia
- diaphoresis
- oliguria
- decreased sensorium/confusion/drowsiness
- cold extremities
- hypotension
- decreased capillary refill
- tachypnea
- metabolic acidosis
Respiratory rate

• adults

• children
VOLUMES

Tidal volume
500 ml

Anatomical dead space
150 ml

Frequency 15 breath/min

Minute volume
7500 ml/min

Alveolar ventilation
5250 ml/min

÷ \approx 1

Alveolar gas
3000 ml

Pulmonary capillary blood
70 ml

Pulmonary blood flow
5000 ml/min
Tidal volume

- Volume of air inspired or expired at each breath
Minute volume

• TV x f
FRC

- The volume of gas remaining in the lungs after a normal expiration
Factors decreasing FRC

- increasing age
- posture – supine
- anaesthesia – intraoperative
- abdominal and thoracic surgery – postoperative
- pulmonary fibrosis
- pulmonary oedema
- obesity
- abdominal swelling – pregnancy, tumour, ascites
- thoracic cage distortion
- reduced muscle tone
Factors increasing FRC

- increased intrathoracic pressure – PEEP, CPAP
- emphysema
- asthma
NORMAL CAPNOGRAM
NORMAL CAPNOGRAM

- Phase I is the beginning of exhalation
- Phase I represents most of the anatomical dead space
- Phase II is where the alveolar gas begins to mix with the dead space gas and the CO$_2$ begins to rapidly rise
- The anatomic dead space can be calculated using Phase I and II
- Alveolar dead space can be calculated on the basis of: $V^D = V_{Danat} + V_{Dalv}$
- Significant increase in the alveolar dead space signifies V/Q mismatch
NORMAL CAPNOGRAM

- Phase III corresponds to the elimination of CO₂ from the alveoli
- Phase III usually has a slight increase in the slope as “slow” alveoli empty
- The “slow” alveoli have a lower V/Q ratio and therefore have higher CO₂ concentrations
- In addition, diffusion of CO₂ into the alveoli is greater during expiration. More pronounced in infants
- ET CO₂ is measured at the maximal point of Phase III.
- Phase IV is the inspirational phase
PaCO$_2$-PetCO$_2$ gradient

- Usually <6mm Hg
- PetCO$_2$ is usually less
- Difference depends on the number of underperfused alveoli
- Tend to mirror each other if the slope of Phase III is horizontal or has a minimal slope
- Decreased cardiac output will increase the gradient
- The gradient can be negative when healthy lungs are ventilated with high TV and low rate
- Decreased FRC also gives a negative gradient by increasing the number of slow alveoli
Blood gas analysis

\[ \text{pH} \quad 7.36 - 7.46 \]
\[ \text{pCO}_2 \quad 36 - 45\text{mmHg (4.8 - 6.0 kPa)} \]
\[ \text{HCO}_3^- \quad 21 - 26 \text{ mmol/l} \]
\[ \text{BE} \quad +/- 2.5 \text{ mmol/l} \]
\[ \text{pO}_2 \quad 65 - 95 \text{ mmHg (8.7 - 12.7 kPa)} \]
Mechanical ventilation vs spontaneous ventilation
Spontaneous Ventilation
Controlled Mechanical Ventilation
<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>RESPONSE</th>
<th>SCORE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eye opening</td>
<td>No response</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Spontaneously</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>To verbal command</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>To pain</td>
<td>1</td>
</tr>
<tr>
<td>Motor response</td>
<td>Obeys verbal command</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Localizes pain</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Flexion-withdrawal</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Decorticate rigidity</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Decerebrate rigidity</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>No response</td>
<td>1</td>
</tr>
<tr>
<td>Verbal response</td>
<td>Oriented and converses</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Disoriented and converses</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>No response</td>
<td>1</td>
</tr>
</tbody>
</table>
Body temperature
monitoring

- Deep body temperature
  - Oesophagus
  - Central veins
  - External acoustic duct
  - rectum

- Superficial body temperature
Hypothermia

- Mild 34 - 36
- Moderate 28 – 33
- Deep 17 - 27
hypothermia

- Decrease consciousness
- Decrease metabolism of fixed drugs
- Delay excretion of drugs and muscle relaxants
- Increase potential for cardiac dysrhythmias
Hypothermia

- Air condition in the operating room
- Vasodilatation induced during anesthesia
- Use of semiopen or semiclosed rebreathing system in anesthesia machine
- Administration of cold fluids intravenously
- Heat lost from exposed body cavities during different surgical techniques
- The inhibition of thalamic regulation of temperature by anesthetic drugs
hypothermia

- Treatment?
Malignant hyperthermia

- A fulminant hypermetabolic crisis triggered by anesthetic agents
- Causative agents: most frequently halothane and succinylocholine
Malignant hyperthermia

- Clinical findings
  - Early: muscle rigidity following succinylcholine, tachycardia, tachypnea, unstable blood pressure, arrhythmias, cyanosis, sweating, rapid temperature increase
  - Late: skeletal muscle swelling; left heart failure; renal failure, DIC
  - Laboratory: respiratory and metabolic acidosis, hypoxemia, increased serum levels of K$^+$, Mg$^{2+}$, mioglobin, CPK, myoglobinuria
Thanks for your attention