BIOCHEMICAL MONITORING IN CRITICALLY ILL

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CRITICALLY ILL PATIENT - DEFINITION

- Decompensation of the status of the patient leading without therapeutic intervention to the multiorganic failure and to death

MAIN CLINICAL DIAGNOSIS OF INTENSIVE MEDICINE

- Traumatological: polytrauma, crush sy, craniocerebral , contusion of the chest, burns
- Shock of various clinical causes, cardiac impairment
- Sepsis 25% mortality
- acute hemorrhagia, pulmonal embolia

HOW TO TREAT CRITICALLY ILL PATIENT?

- To save the basic vital function
- Stabilization of patient
- Support of insufficiency organs , prevention impending organ insufficiency
- Right diagnosis
- adequate therapy

DIAGNOSTIC OF CRITICALLY ILL PATIENTS

- Clinical data, examination /vomitus, heavy smoker, DM, icterus, arytmie, hepatomegalia../
- Monitoring of basic function (blood pressure, heart rate…)
- Imaging methods (X-ray, Computer tomography)
- Bacteriology
- Hematology, Imunology
- Biochemistry

THE POSSIBILITIES OF BIOCHEMICAL MONITORING

- On-line monitoring (cardiosurgery – pH, minerals (K), the electrodes are localized on central cateter, possibility to check parameters on-line.

- bed side monitoring (point of care testing – acidobasis anal.-whole blood, urine /protein, pH, blood../,oximeter O2 saturation, drugs /dg.strips, glycaemia)

- Biochemical analysis (vital indication, statim ,rutine analysis .. severity of pts status how quick and what)

BIOCHEMICAL PARAMETERS – STATIM SET

- Na,K,Cl,Ca,P,Mg, osmolality - blood, urine
- Acidobasis, lactate
- urea, creatinin, creatinin clearence, Nitrogen balance
- bilirubine, ALT, AST, GMT, LD, amylase, lipase
- cholesterol, triglycerides, glucose – blood, urine
- CK, CK-MB, Troponin T,I, myoglobine, CK-MBmass
- Total protein, albumine, prealbumine,CHE
- CRP, procalcitonine
- TSH, b-HCG.
- Basic analysis are made at the first,must be done within 90minutes

THE RANGE AND FREQUENCY OF BIOCHEM.MONITORING- PATIENTŚ STATUS

- Diagnostic examination-a new patient
- Standart examination relat. stabilized patient
- Storm, shock, syncopa, coma..
- Perioperation monitoring

THE INDICATION FOR EXAMINATION

What do I expect ?

- Confirmation of clinical diagnosis – (ac.pancreatitis, AIM, hyperkalemie by ARI).
- Monitoring of therapy (Na, K, Mg, Ca deficit,glykemie,CRP, PCT,bilance).
- Answer for unclear status of patient- (may be I find something/
- Automatism

NA, K, CL, Ca, P, MG - SERUM LEVELS

- Hypernatremia – over 150 mmol/l
- Sec. hyperaldosteronism (rapid decrease of patients condition, rises capillary leak, albumin(water) moves to intersticium, intravascular hypovolemia)renin-angiot-aldost. UNa<20mmol/l..oedema, prerenal insuf.
- Hypothalamic damage
- Hypertonic hyperhydration
- Diabetes insipidus
- Brain death

NA, K, CL, Ca, P, MG - SERUM LEVELS

- Hyponatremia – under 130 mmol/l
- Na in the third space - ascites, hydrothorax
- Cardiac failure – increase of extracellular volume
- Application of solutions without electrolytes
- Hypersecretion of ADH – water retention

OSMOLALITY (BLOOD, URINE)

- All osmotic active particles (measured x counted)
- 2x Na + glykemia + urea
- 380 mosmol/l x 265 mosmol/l
- Quick changes- danger for neurons
- Urine osmo- tubular function, hydratation

NA, K, CL, Ca, P, MG - SERUM LEVELS

- Hyperkalemia – over 5,0 - 5,5 mmol/l – pH dependent /acidosis increases K level pH decreased 0,1…K increased 0,6mmol/l /
- Bigger intake, low output or both
- Acute renal failure
- Cytostatics, tumors- necrosis of cells
- Acute metabolic acidosis
- Infusion with K

NA, K, CL, Ca, P, MG - SERUM LEVELS

- Hypokalemia – under 3,5mmol/l
- Low intake, bigger uptake, or both
- Emesis, diarrhoe / intestinal loss/
- Diuretics
- Chemotherapy, antimycotics /renal tubules failure/
- Anabolic phasis
- Hyperaldosteronism
- Acute metabolic alcalosis

Mentální anorexie, 163cm 34 kg

	12.55	20.47	7.05	7.15
Na	121	128	132	135
K	1,5	1,8	2,3	3,9
Cl	53	74	88	104
Ca	2,3		2.0	1,9
urea	10,1 (krea 77)		6,5	3,9 (krea 43)
AST	0,6			
Alb	45		30	25
CB	77.9		61	53
glykémie	8.9		4,7	4,3
pH		7,555	7,467	
pCO2		6,0	7,0	
BE		15,4	12,8	
chol	8,9		6.8	
TG	3,1		0,8	
U-Cl		< 15		108

NA, K, CL, CA, P, MG - SERUM LEVELS

- Ca-total-binding protein 40%,12% binding phosphates,hydrogencarbonate,ionizate 48%.
- Ioniz.Ca the only one biological active form /influeces contractility of muscle,hemocoag../.
- Hypoalb. -10g /-0,2mmol/l, pH +0,1/ +0,03 mmol/l binding protein-decrease ionizate form iCa-tetanie
- Cl corig.Cl diagnosis of hypo chloremic alcalosis, hyperchloremic acidosis
-

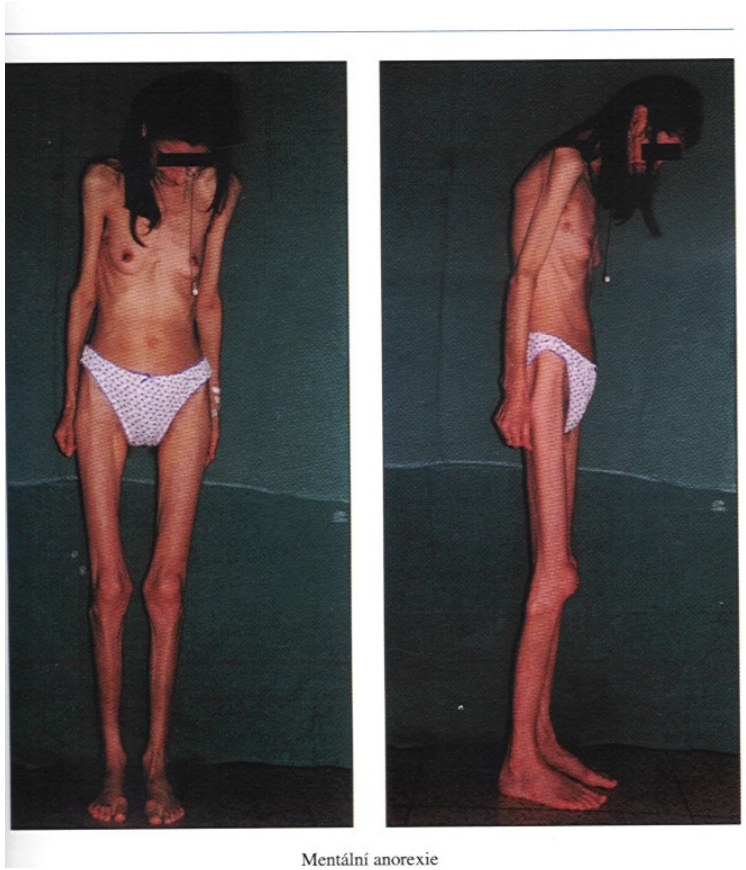
NA, K, CL, CA, P, MG – SERUM LEVELS

- Mg – together with potassium
- Hypomagnesaemia – under 0,6 mmol/l / in renal failure cause impaire of renal tubular cells , low intake../
- Preventive application during cardiac surgery (metabolic resuscitation of myocardial cells – K, P, Mg, glucose

NA, K, CL, CA, P, MG – SERUM LEVELS

- Hypophosphataemia – under 0,6 mmol/l
- Acute wastage of energy after succesfully resuscitation, overfeeding sy, anabolism (energetic substrates without K,Mg,P)
- Hyperphosphataemia – over 1,9 mmol/l
- Renal failure
- Cell damage

ANOREXIA NERVOSA



Mentální anorexie

OVERFEEDING SYNDROM (chron. malnutrition, high doses of glucose without minerals K,Mg,P

	21.3. 9,30	21.3. 15,30	22.3. 9,30
Na	136	137	138
K	3,7	2,4	4,1
P		0,15	0,9
Mg		0,3	0,8
glykémie	5,4	12,6	5,1
laktát	1,1	2,8	1,3
urea	0,6	4,6	2,0

NA, K, CL, CA, P, MG – URINE LEVELS

- Hypernatriuria – over 200 mmol/l
- High input of Na (food, drugs)
- Na mobilisation from the 3rd space during anabolic phase
- Hyponatriuria – under 20 mmol/l
- Hyperaldosteronism, Hypovolemia
- Diferential diagnosis between prerenal and renal failure

NA, K, CL, CA, P, MG – URINE LEVELS

- Hyperkaliuria – hyperaldosteronism Na – K change
- Hypokaliuria
- Hypocorticoidism
- The risk of hypokalemia

- Metabolic balance of electrolytes/24 hours – monitoring of parenteral nutrition
- Clearence Na,K, fraction

ACIDOBASIS, LACTATE

- We evaluate several aspects,
- Clinical aspects anamnesis-emesis, diarhoea, smoker , renal failure…
- Electrolytes, total protein, albumine, Hemoglobine, respiration functions, hepatic and renal function, hydration,

- Metabolic acidosis – cardiopulmonal resuscitation, Diabetes mellitus, renal insuficiency, hypoxia, hypoperfusis
- Metabolic alcalosis – blood transfusions, hyperaldosteronism, stomach output

ACIDOBASIS, LACTATE

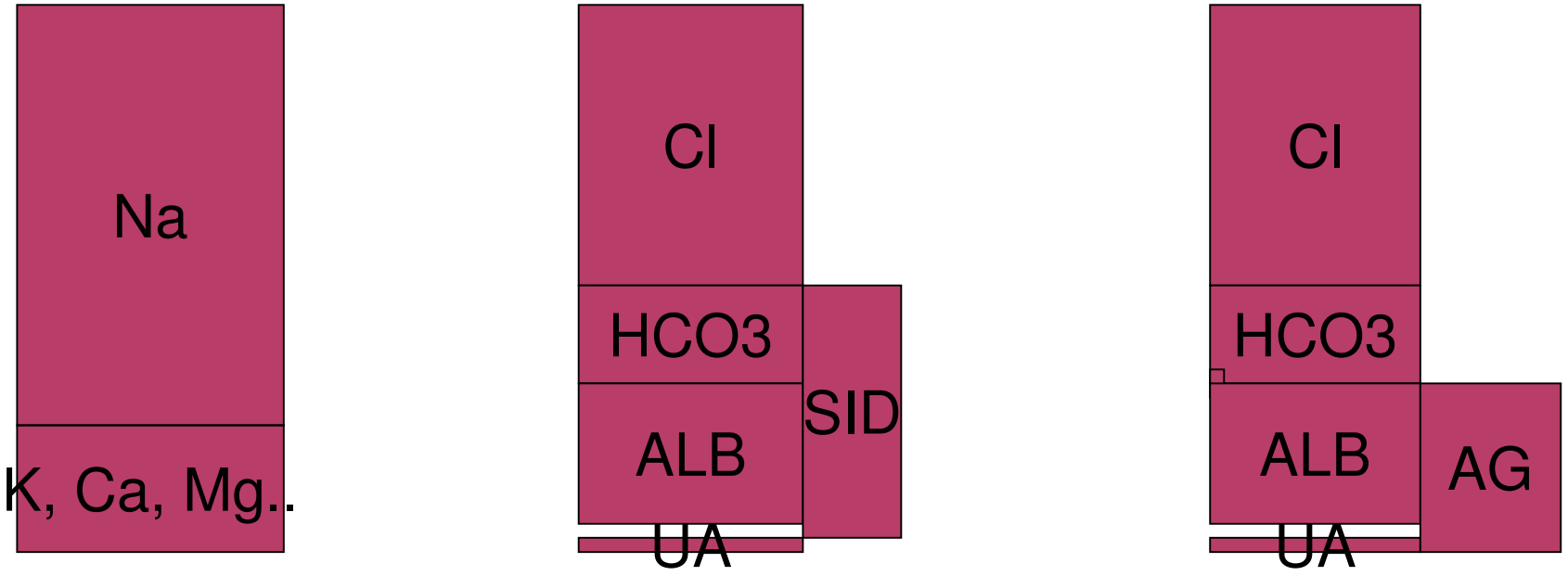
- Respiratory acidosis – Chronic obstructive bronchopulmonary disease, hydrothorax, malnutrition, high intake of glucose in parenteral nutrition
- Respiratory alcalosis – hypoxia, microembolisation into pulmonary arteria, shock, craniocerebral trauma

NEW ASPECTS I EVALUTION ABB - STEWARD-FENCL

- Decomposition of finding on examination ABB into separate failures- possibility causally therapy /90% mixed failures ABB/
- pH, pCO2, HCO3,BE….
- SID,Cl corig.,UA,P, Alb.,.AG corig.
- Alb 44, P 0.9-1.1, Na 140

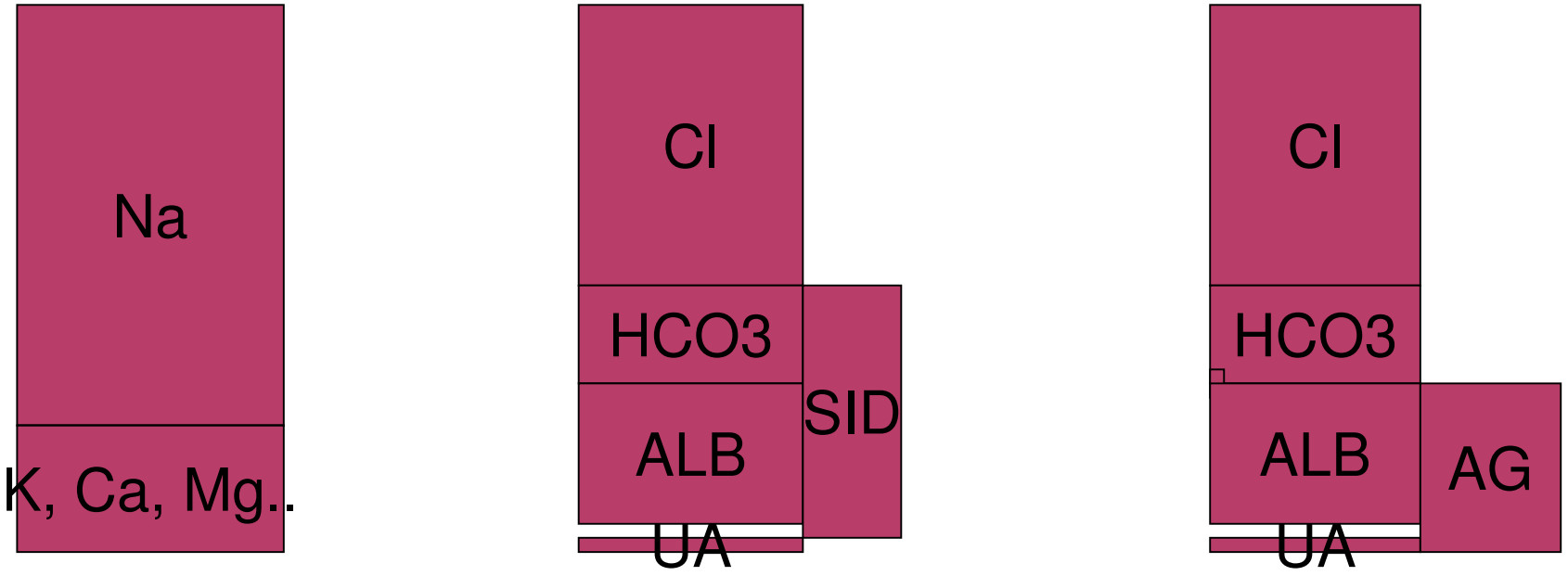
NEW MARKERS

- SID = Na(sum)-Cl+UA/ 38-40 mmol/l...HCO3+Alb.+P
- Cl corig.= Cl akt. x Na akt./Na norm. 104-108 mmol/l
- AG= Na + K - /Cl +HCO3/..RA
- AGcorig for act.albumin
- UA=/Na+ K+ Ca+Mg/ -/Cl+ SID/
- UAcorig for act.Na



INTERPRETATION OF NEW MARKERS

- Changes of SID –decrease MAC /increase Cl, UA,water dilution../
- Increase SID- MAL /decrease Cl, water loss../
- UA inrease- MAC /lactate,hydroxybutyrate,sulfate../
- AG increase MAC- UA,hyperfosfatémia, decrease- MAL hypoproteinémia



PARAMETERS NEED FOR CALCULATION

- Na, K, Cl, P, Ca, /Mg/
- Albumin
- pH, pCO2,akt.HCO3, BE, pO2

CAUSAL TREATMENT

- Improve perfusion, hydratation,saturation of O2/
- Correction ionts dysbalance Na,Cl,K
- Compensation of renal failure, DM, hepatic functions
- Correction hypoproteinémia
- Correction lactic acid MAC
- Correction respiratory component

ABR, LACTATE

- Hyperlactataemia – over 2,2 mmol/l /1,0/

- Bigger offer- move the balance of lactate-pyruvate to the part of lactate
- Hypoxia – cardiopulmonal resuscitation
- low level of B1 /koenzyme pyruvatedehodrogenaze complex..Krebs cycle/
- Metabolic failure – diabetic coma, intoxication,
- Low output
- hepatopathy, leucaemia …

UREA, CREATININE, CREATININE CLEARENCE, N-BALANCE, URATIC ACID

- The level of urea – together with nutritional status, hydration
- High level of urea – high intake of N, increase catabolism /polytrauma- 500gr of musceles per day loss/ GIT bleeding, dehydration
- low output- renal failure,
- Low level – malnutrition,serious hepatic failure- ureosyntetic cycle and gluconeogenesis dysfunction, pregnancy- increase ECF

UREA, CREATININE, CREATININE CLEARENCE, N-BALANCE, URATIC ACID

- Urea in urine
- Increase – catabolism, prerenal failure
- Decrease – chronic malnutrition, acute renal failure

UREA, KREATININ, CLEARENCE KREATININU, N-BALANCE, URATIC ACID

- Serum levels of creatinine evaluation together with muscle mass, age, gender
- Increase – bigger offer- destruction of muscle mass, low output-renal failure
- Decrease- low offer-low muscle mass-seniores, malnutrition
- Creatinine clearence, excretion fraction -renal function
- N-balance – catabolism – the need of nitrogen
- Uratic acid – cell damage, arthritis uratica

NGAL NEUTROPHIL GELATINASE-ASSOCIATED LIPOCALIN

- in renal tubules- increase during ischemia and nefrotox.demage./elevation after 2 houres/
- Blood and urine
- Early,sensitive and non invasive parameter of acute renal demage
- Elevace above 350-400ng/ml positive prediction ARI /90%/

RENAL FUNCTION IN SEPSIS DEVELOPMENT

	23.11.	22.11.	21.11.	Ref.rozmezí
K	6,4	6,8	5,3	3,8- 5,0 mmol/l
P	3,2	2,6	1,8	0,65-1,61ug/l
urea	30,2	24,3	6,7	2,0-6,7 mmol/l
krea	125	93	65	44-104umol/l
glykémie	14,0	12,4	14,9	3,9-5,6mmol/l
TG	5,0	5,1	4,3	0,68-1,6mmol/l
CRP	230	130	92	<7 mg/l
PCT	48	16	5,3	<0,5 ng/l
U-Na	N	3	26	
U-K	N	119	80	-

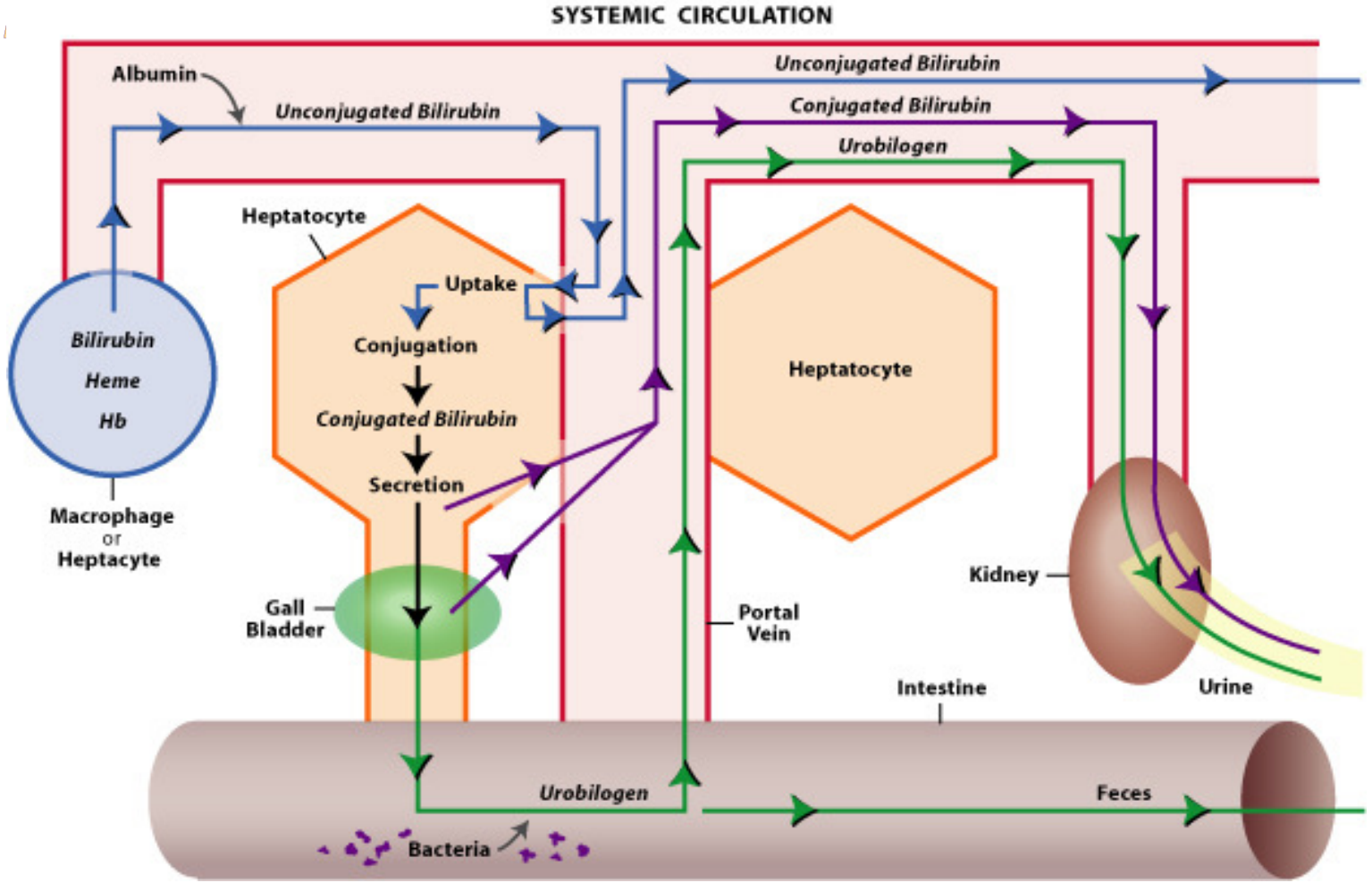
RENAL FUNCTION AFTER ATACS OF SEPSIS (2 MONTH IN ICU)

	3.11.	1.11.	11.09.	Ref.rozmezí
K	6,4	5,1	4,3	3,8- 5,0 mmol/l
P	2,6	1,4	1,8	0,65-1,61ug/l
urea	24	2,3	3,7	2,0-6,7 mmol/l
krea	91	25	65	44-104umol/l
glykémie	12,0	4,5	6,5	3,9-5,6mmol/l
TG	2,5	1,5	1,3	0,68-1,6mmol/l
CRP	230	13	122	<7 mg/l
PCT	18	0,2	0,3	<0,5 ng/l
U-Na	N	113	116	
CCrea	0,8	2,5	1,9	1,58-2,67

BILIRUBIN, ALT, AST, GMT, ALP, LD, AMONIUM, AMYLASE, LIPASE

- Bilirubine direct, indirect
- High level – neonatal hyperbilirubinaemia, massive blood transfusion, hepatocellular damage /drugs, hepatitis, hypoperfusion, necrosis, obstruction – extra-, intrahepatic

METABOLISMUS OF BILIRUBIN



BILIRUBINE, ALT, AST, GMT, ALP, LD, AMONIUM, AMYLASE, LIPASE

- ALT – high level – hepatopathia, steatosis, hepatitis, cell damage,
- AST – high level – hypoperfusion, hepatitis, cell necrosis, muscles damage
- both aminotransferases increase during damage of hepatic cells during inf.hepatitis.

BILIRUBINE, ALT, AST, GMT, ALP, LD, AMONIUM, AMYLASE, LIPASE

- ALP – increase – extrahepatal obstruction, isoenzymes – intestinal, bone, placental
- GGT – increase – obstruction intra, extrahepatal, intoxication – ethanol, amanita..
- Both enzymes increase during parenteral nutrition. Impaire enterohepatic cycle of bile acids

BILIRUBIN, ALT, AST, GMT, LD, AMONIUM, AMYLASE, LIPASE

- amoniac-increase during hepatic insufficiency /decompenzation of cirrhosis, premature /
- amylasa-serum, or plasma and urine, drain
- pancreatic izoenzyme
- Increase- irritation of pancreas, hypoperfusion, treatment with opiates,inflamation-pancreatitis, tumor
- lipasa- specific for pancreas

CHOLESTEROL, TG, GLUCOSA

- TG-increase- during sepsis, mainly on the begining,monitorate during parenteral nutrition with lipids more than 5 mmol/l contraindication to take lipid emulsion

- Glycemia, serum, urine,
- Hypoglycemia below 2,5mmol/l-vital danger
- hyperglycemia- insulin.rezistence, recomendation level of glycemia 4,5-8,2 /2006/ better survive in ICU patient

BIOCHEMICAL MARKERS OF NUTRITION STATUS:

- Plasmatic proteins with short biologic half-life

- Albumin
  - -syntetizate in liver, half-life time is 21 days
  - Fyziolog.value35-45g/l.Decrease of alb.level is typical for malnutrition kwashiorkor./chronical maln./Trends of changes alb.levels during realimentation are criterium of succesfull terapy.
  - Acute decrease during inflammatory- increase of capilary leak,escape fluid from IVspace to intersticium.Hypalbuminemiae-marker of systemic responce.

BIOCHEMICAL MARKERS OF NUTRITION STATUS:

- Transferin-syntetizate in liver,biolog.half-life is 8days.Fysiolog.value2-4g/l,

- Prealbumin-syntetizate in liver,biolog.half-life is 1,5 days.Fysiolog. Value 0,15-0,4g/l.Decrease in failure of proteosyntesis-indicator of acute protein malnutrition.

- RBP - biolog.half-life time is 12hod.,fysiolog.value 0,03-0,006g/l.

BIOCHEMICAL MARKERS OF NUTRITION STATUS:

- PINI / prognostic inflanatory nutrition index /

- Acute infect /tonsilitis/CRP-83,AGP-2.4,prealb.0,35,alb.42 PINI=10
- Chron.malnutrition patient CRP 40,AGP 2,0,prealb.0.1,alb.27,PINI=29
- Polytrauma,sepse CRP 240,AGP 2,5,prealb 0,07,alb 25,PINI=600.

PROTEINS OF ACUTE PHASE (PAF) IN ICU PATIENTS

	21.3.after ATB treatment	2.3.development of sepsis	1.3. admission
CB	44	65	80
albumin	15	24	40
prealbumin	0,09	0,18	0,25
CRP	120	240	110
PCT	2,3	12,6	5,1

CARDIOMARKERS : CK, ,TROPONIN T, I, MYOGLOBIN, CK-MB MASS, BNP, PRO BNP

- Myoglobin-2-12 hours, nonspecific for myocard,increase injury muscels mass, renal insufficiency
- CK-MB mass-3-36 hours-better specific than myoglobin, less than troponin
- Troponin T, I-4h-7days,absolut specifity for myocard

- dg.ACS combination 3 markers
- CK and CRP for monitoring dynamic and range of damage

- BNP, proBNP diagnosis heart failure, exclude noncardiac dyspnoe.

RABDOMYOLYSIS

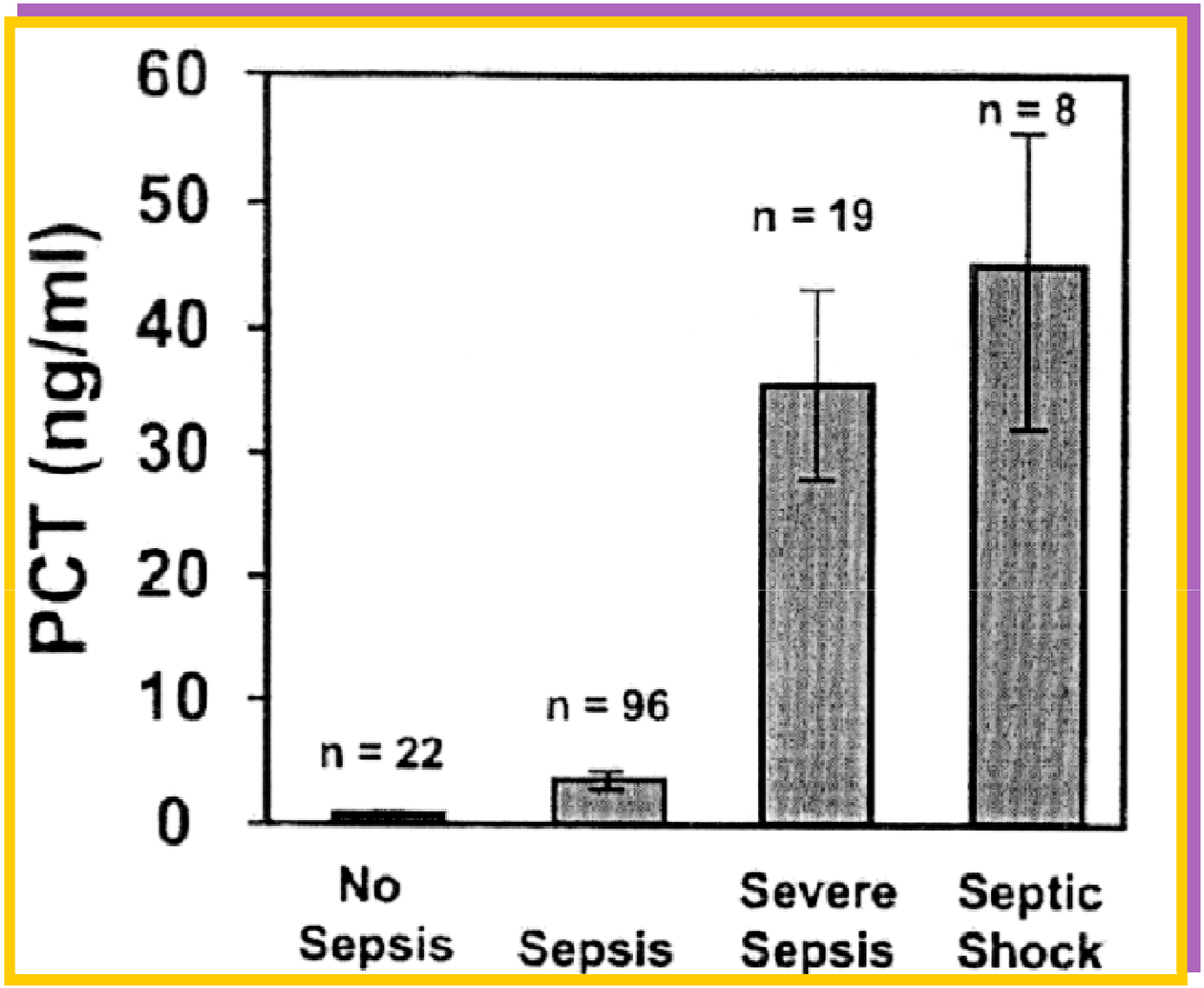
	25.11.	26.11.	1.12.	Ref.rozmezí
bili	11,8	12,6	12,1	2-17 umol/l
ALT	7,4	7,6	3,0	0,1-0,78 ukat/l
AST	30,2	24,3	1,7	0,1-0,72 ukat/l
GGT	0,2	0,3	0,1	0,1-0,7 ukat/l
CK	1641	915	14,9	19-92 ug/l
CK-MB	12,8	8,1	0,3	0,0-0,42 ukat/l
Myoglobin	5946	1304	92	0,2-2,6 ukat/l
Troponin	0,02	0,01	-	<0,04
CRP	12	13	2	<7mg/l
Blood in urine	+	+	-	-

INFLAMMATORY MARKERS : CRP, PROCALCITONIN

- CRP /C-reactive protein/ 6-12 hours to increase,nespec.increase-viral inf.,tumors,monitoring of ATB therapy.
- Normal value is less 7 mg/l / bact.inf.more than 100 mg/l

- PCT /procalcitonin/-more sensitive and earlier marker for systemic bacterial inflammation depends on degree of MODS and stages of sepsis

- Combination both PCT and CRP



(*Zeni et al. Clin.Intensive Care 1994, Suppl. 2, s. 89-98, cit. Meisner 2002)*

LOCAL BACTERIAL INFECT- ABDOMINAL ABSCES

Den	1	2.	3.	4.	5.	6.	7.	
PCT	1,5	1,8	1,3	0,9	0,3	0,1	0,3	
CRP	220	245	280	230	150	85	75	

WEGWNERS GRANULOMATOSIS, LOB.PNEUMONIE,SEPSIS

	Na	urea	ALT	Alb	pA	CRP	PCT	chol
3.10	150	17	1,4	25	0,07	458	23	2,6
4.10	155	26,7	1,5	20	0,07	718	71	2,6

CMV	bili	ALT	AST	GGT	ALP	LD	pA	CRP	PCT
14.5.	23	6,0	3,7	1,3	2,5	15	0,20	40	0,05
30.5	19	2,5	0,6	0,8	2,2	9,0	0,25	20	0,05