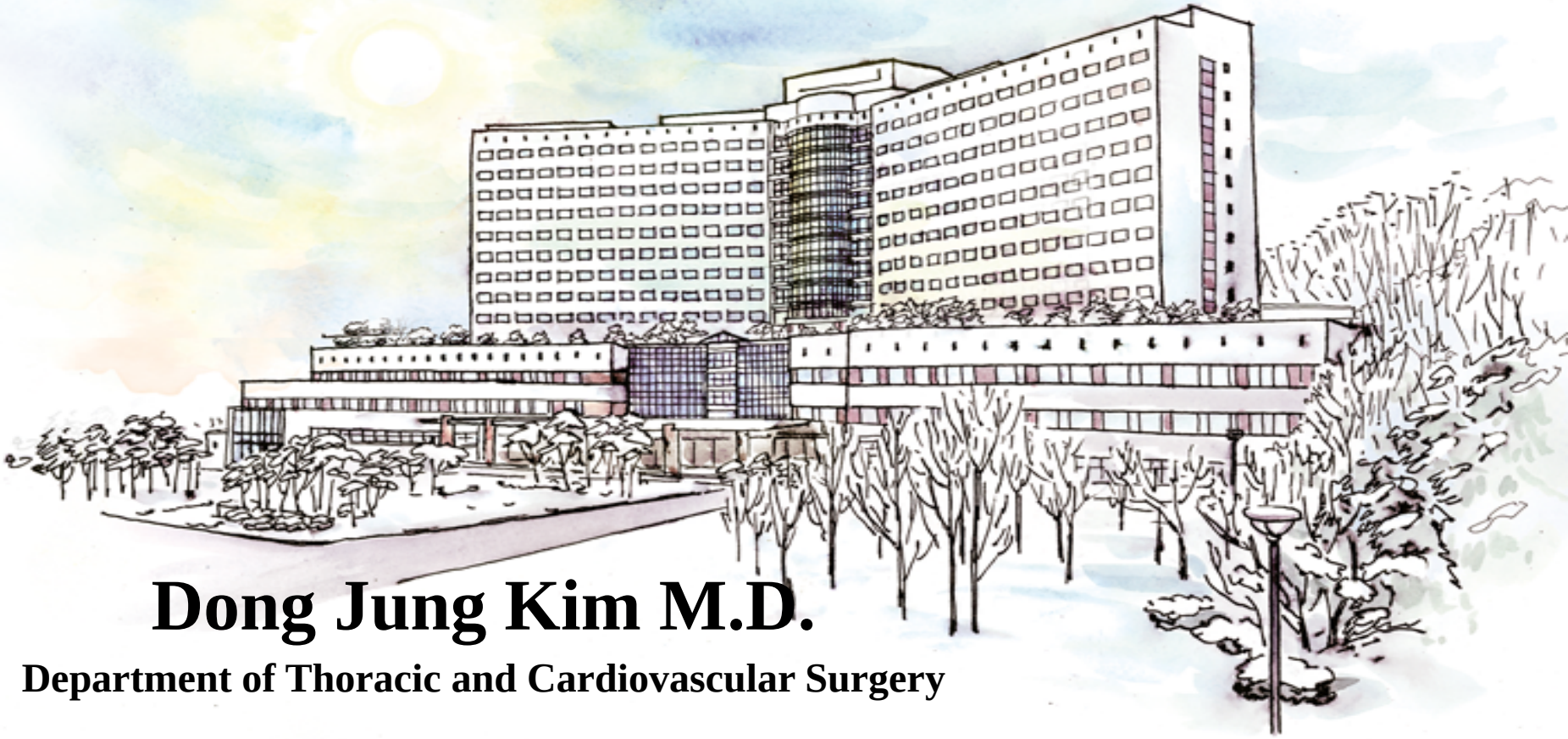
흉부외과 전공의 연수강좌

Fluid, renal and metabolic problem management



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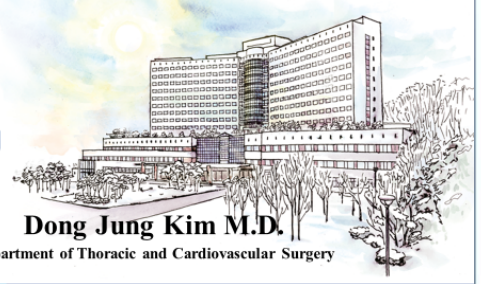
Fluid, renal and metabolic problem management



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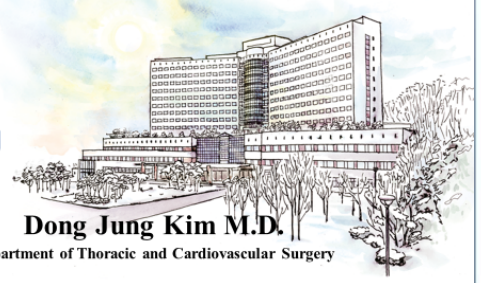
Introduction

Contents

- Fluid management
- Acute kidney injury
  - CRRT
- Hyperkalemia
- Metabolic acidosis

Fluid, renal and metabolic problem management



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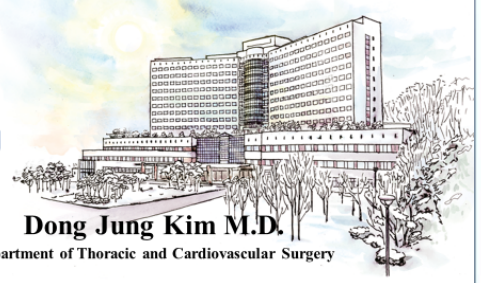
Fluid

Postoperative volume status

- Hemodilution on CPB
  - total body Na^+ and water overload
  - ↑ body weight by about 5%
- Cardiac filling pressures
  - do not reflect state of volume overload
  - capillary leak from systemic inflammatory response
    - ↓ plasma osmotic pressure
  - impaired myocardial relaxation (diastolic dysfunction)
    - from ischemia/reperfusion after cardioplegic arrest
  - vasodilation

Fluid, renal and metabolic problem management



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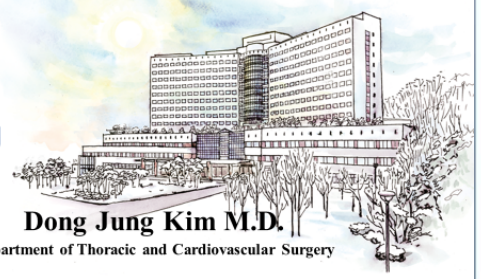
Fluid

Postoperative fluid management

- Low filling pressures with hypovolemia
  - despite presence of body water overload
  - additional fluid administration
    - to maintain satisfactory hemodynamics
- High filling pressures with hypovolemia
  - especially in patients with diastolic dysfunction
  - additional fluid administration
    - may be necessary in that situation as well

Fluid, renal and metabolic problem management



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Fluid

Routine fluid management

- Fluid must invariably be administered
  - to maintain intravascular volume and hemodynamics
  - at the expense of expansion of interstitial space
- 1st 4~6 hours after surgery
  - cardiac output is often depressed
  - hemodynamics is dependent on both preload and inotropic support

Fluid, renal and metabolic problem management

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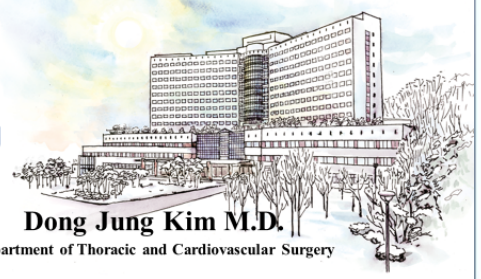
Fluid

Types of fluid

- Any fluid will expand interstitial space
  - during a period of altered capillary membrane integrity
- Preferable fluid
  - more effectively expand intravascular space
  - minimize expansion of interstitial space

Fluid, renal and metabolic problem management



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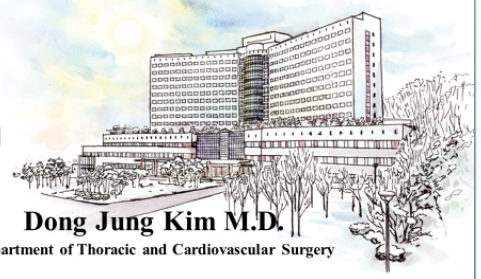
Fluid

Body water distribution

- 60% of body weight (BW) is water
  - ⅔ is in the intracellular space (ICF, 40% of BW)
  - ⅓ is in the extracellular space (ECF, 20% of BW)
    - 3/4 is in the interstitial space (15% of BW)
      - so-called “third space”
    - 1/4 constitutes the intravascular volume (5% of BW)
      - 1/12 of total body water (TBW)

Fluid, renal and metabolic problem management



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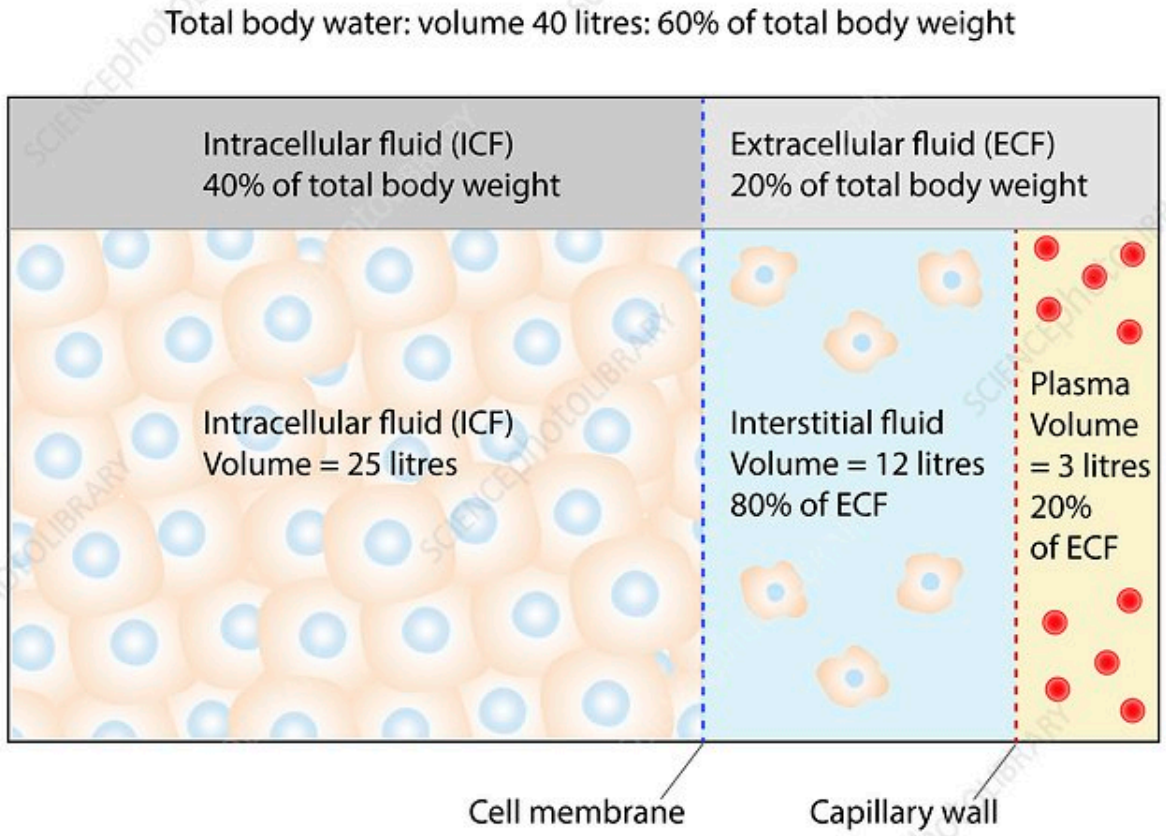
Fluid

Body water distribution



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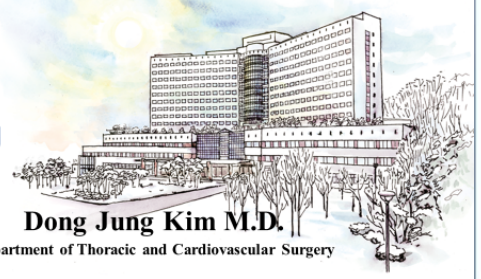

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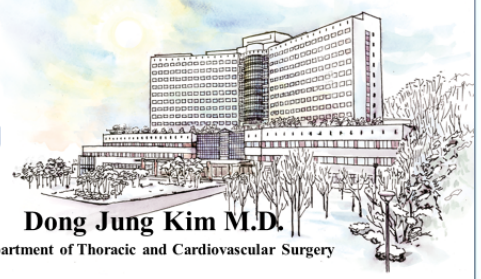
Fluid

Crystalloid

- Infusion of 1L of 5% DW
  - dextrose is utilized, water is distributed into TBW
  - intravascular volume (1/12 of TBW) expands 83mL
- Infusion of 1L of half saline
  - NS ½ + water ½
  - intravascular volume (500×1/4 + 500×1/12) expands 167mL
- Infusion of 1L of 0.9% normal saline
  - distributed into ECF
  - intravascular volume (1/4 of ECF) expands 250 mL
  - 25% is retained in intravascular compartment after 1 hour

Fluid, renal and metabolic problem management



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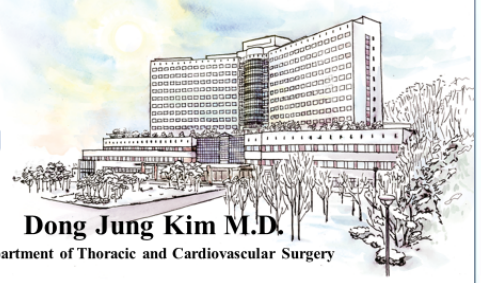
Fluid

Colloid

- Infusion of 1L of 6% hetastarch
  - intravascular volume expands by 1123mL
  - more long-lasting effects than crystalloid
- Infusion of 5% albumin
  - expand plasma volume five times (vs NS)
- Infusion of of 20% albumin
  - 100ml, in patient with 4g/dl of serum albumin level
  - 20% is diluted to 4% (5×)
  - intravascular volume (100×5) expands by 500cc
  - similar effect with infusion of 2L of NS

Fluid, renal and metabolic problem management



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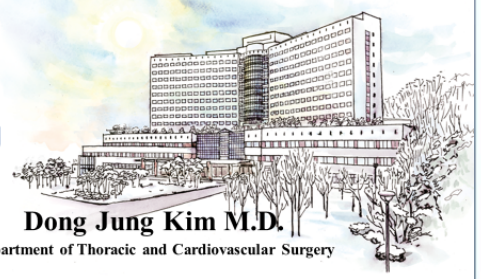
Fluid

Types of fluid

- Blood and colloids
  - superior to hypotonic or even isotonic crystalloid solutions in expanding intravascular volume
- Rapid infusion of crystalloid
  - transiently beneficial in increasing intravascular volume acutely
    - d/t rapid redistribution into interstitial space

Fluid, renal and metabolic problem management



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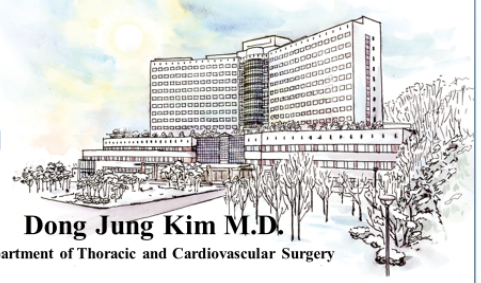
Fluid

Albumin (5%)

- Excellent volume expander
  - approximately 200ml retained per 250ml bottle administered
  - leak into interstitial space d/t capillary leak
- Dilutional effects on clotting parameters
  - preserve coagulation better than 1st generation HES
- Protective effects on kidney
  - O_2 free-radical scavenging & anti-inflammatory properties
- Half-life : 16 hours
  - leaves bloodstream at rate of about 5~8 g/h

Fluid, renal and metabolic problem management



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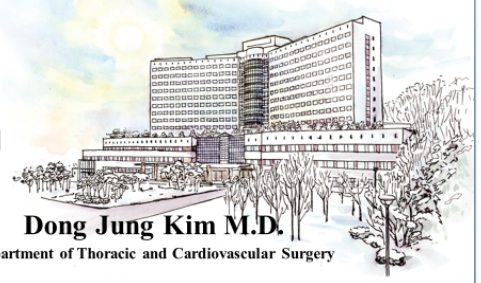
Fluid

Hydroxyethyl starch (HES)

- Nonprotein colloid volume expanders
  - characterized by molecular weight (MW) in kilodaltons (kDa) and molar substitution (MS)
- 1st generation HES (high MW solutions)
  - Hespan (6% hetastarch in saline)
  - Hextend (6% hetastarch in balanced electrolyte solution)
  - >600 kDa and 0.75 MS
  - excellent volume expansion
    - decreases gradually over ensuing 24~36 hours
  - retained in the intravascular space better than 5% albumin
    - in conditions of capillary endothelial leakage

Fluid, renal and metabolic problem management



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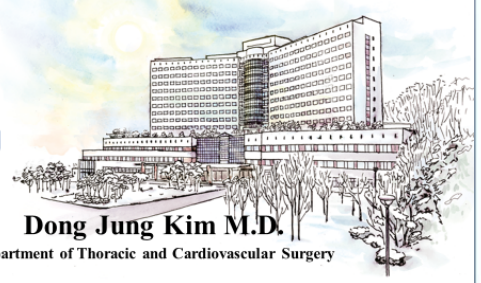
Fluid

Hydroxyethyl starch (HES)

- Concerns about use of HES
  - high MW compounds
    - cause renal dysfunction and coagulopathy
  - low MW HES solutions
    - cause less of coagulopathy than 1st generation HES
      - associated with postoperative bleeding

Fluid, renal and metabolic problem management



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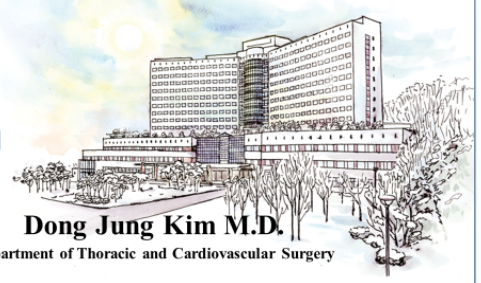
Fluid

Hydroxyethyl starch (HES)

- Low MW compounds
  - pentastarch (6% HES 200/0.5 [Pentaspan])
  - tetrastarch (6% HES 130/0.4 [Voluven])
  - shorter duration, more rapid elimination
    - at least 6 hours for tetrastarch
  - recommended daily doses : limited to 28~50 mL/kg
  - less risk of renal dysfunction and coagulopathy
  - increase in tissue oxygenation
    - demonstrated with HES 130/0.4 compared with lactated Ringer's
    - perhaps because of better microcirculatory flow

Fluid, renal and metabolic problem management



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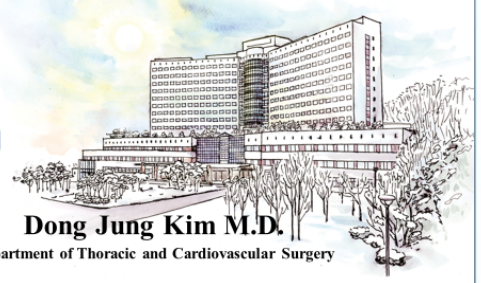
Fluid

Routine fluid management

- Early extubation
  - helpful in reducing fluid requirements
  - ↓ adverse effects of positive-pressure ventilation on venous return & ventricular function

Fluid, renal and metabolic problem management



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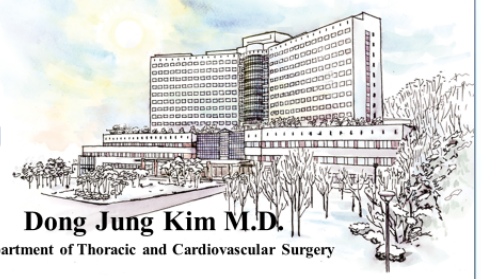
Acute kidney injury

Postoperative AKI

- Oliguria (<0.5 mL/kg/hr)
  - commonly in the 1st 12 hours after surgery
    - responds to volume infusion or low-dose inotropes
  - acute renal insult
    - by prolonged hypotension or low cardiac output state

Fluid, renal and metabolic problem management



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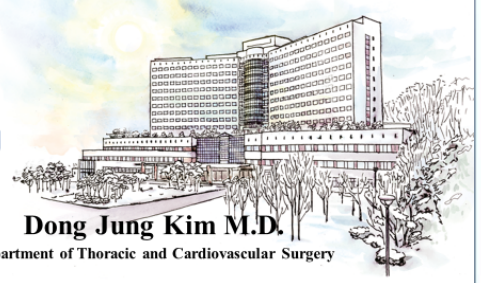
Acute kidney injury

Evaluation of oliguria

	Prerenal	Renal
BUN/Cr	>20:1	<10:1
U/P creatinine	>40	<20
U_{osm}	>500	<400
U/P osmolality	>1.3	<1.1
Urine specific gravity	>1.016	<1.010
U_{Na} (mEq/L)	<20	>40
FE_{Na}	<1%	>2%
Urinary sediment	Hyaline casts	Tubular epithelial cells Granular casts

(Ann Intern Med 2002;137:744–52)

Fluid, renal and metabolic problem management



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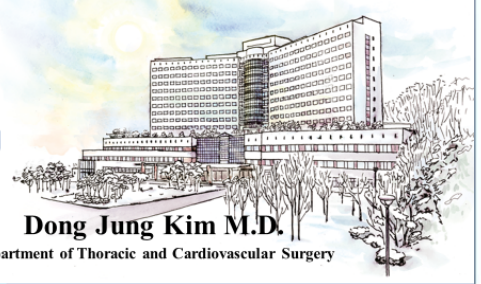
Acute kidney injury

Fractional excretion of sodium (FE_{Na})

- FE_{Na} <1%
  - retained tubular function with absorption of Na^+ & water
  - prerenal problem
    - except in cases of contrast nephrotoxicity & hepatorenal syndrome

- FE_{Na} >2%
  - usually caused by AKI
  - when prerenal process is superimposed on CKD
    - kidneys at baseline cannot conserve water & sodium appropriately

Fluid, renal and metabolic problem management



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Acute kidney injury

AKI : definition

	Scr/GFR Criteria	Urine Output	Overall Incidence
Risk	Increase in Scr × 1.5 *or* Decrease in GFR >25%	<0.5 mL/kg/h × 6 h	20–25%
Injury	Increase in Scr × 2 *or* Decrease in GFR >50%	<0.5 ml/kg/h × 12 h	5–7%
Failure	Increase in Scr × 3 *or* Decrease in GFR >75% *or* Scr >350 µmol/L (>4 mg/dL) *or* Acute Scr rise >44 µmol/L (>0.5 mg/dL)	<0.3 ml/kg/h × 24 h	1–3%
Loss	Persistent acute renal failure with complete loss of kidney function >4 weeks		
ESKD	End-stage kidney disease >3 months		

(N Engl J Med 1986;314:97–105)

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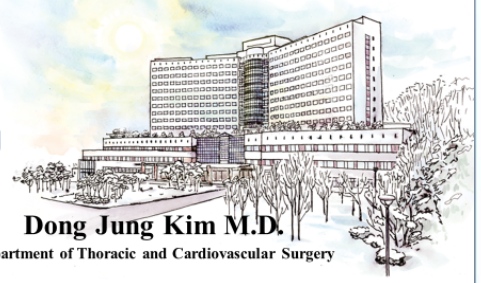

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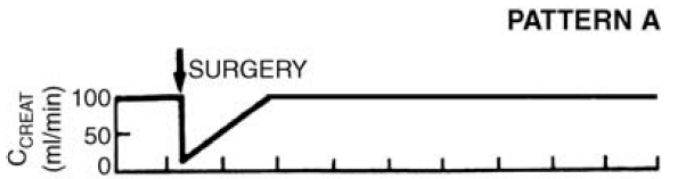
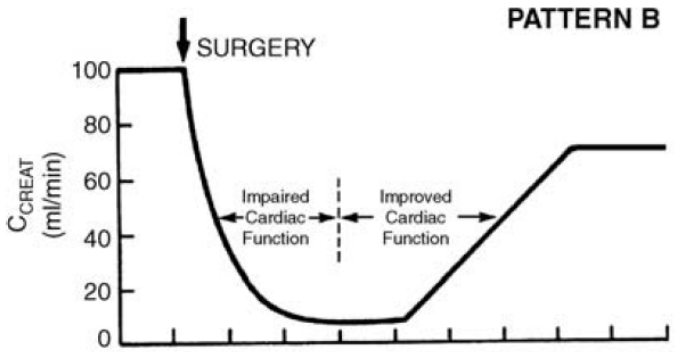
Acute kidney injury

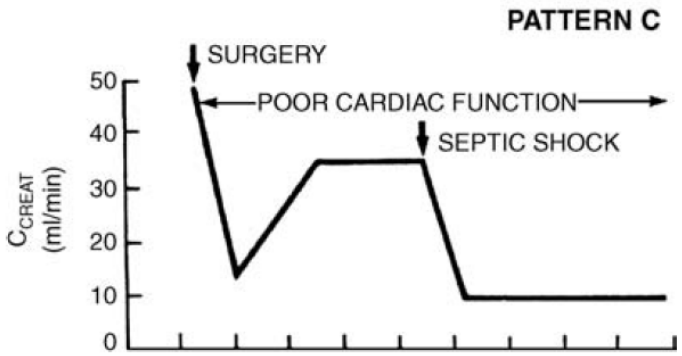
Postop. AKI : patterns



Abbreviated ARF



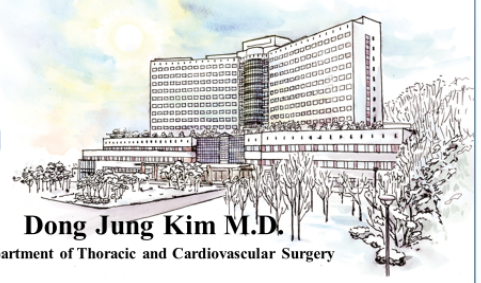
Overt ARF



Protracted ARF

(N Engl J Med 1986;314:97–105)

Fluid, renal and metabolic problem management



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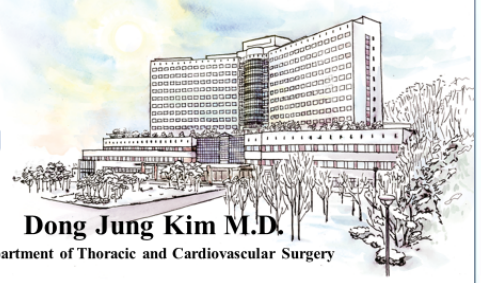
Acute kidney injury

Postop. AKI : patterns

- Abbreviated ARF
  - transient intraop. insult occurs that causes renal ischemia
  - Scr peaks on 4th postoperative day and returns to normal
- Overt ARF
  - acute insult followed by prolonged period of cardiac dysfunction
  - Scr rises to higher level and gradually returns to baseline
    - over 1~2 weeks once hemodynamics improve
- Protracted ARF
  - initial insult followed by a period of cardiac dysfunction
  - another insult from sepsis or a period of hypoperfusion/hypotension
  - progressive, often irreversible rise in Scr

Fluid, renal and metabolic problem management



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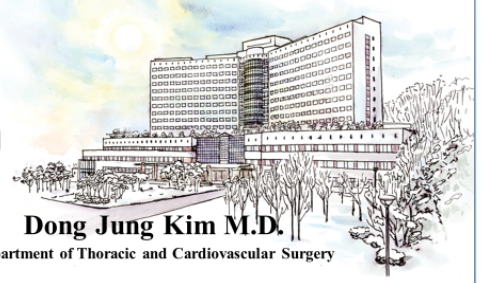
Acute kidney injury

AKI : management

- Once AKI is established
  - very little can be done to promote recovery of renal function
    - except to prevent additional insults
- Early aggressive intervention
  - patients with oliguria and early evidence of AKI
  - to prevent progressive tubular injury and worsening of renal function

Fluid, renal and metabolic problem management



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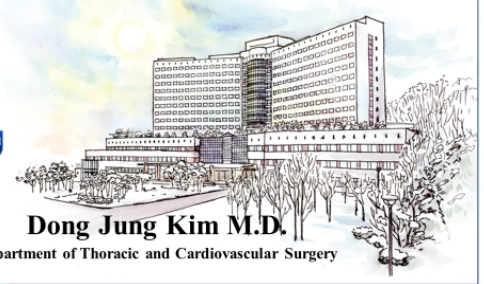
Acute kidney injury

AKI : management

- Increase renal blood flow
  - to reduce tubular obstruction
  - to impact on enhancing tubular epithelial cell proliferation and recovery of function
- Maintain urine output
  - to reduce tissue edema
  - to treat electrolyte or metabolic problems

Fluid, renal and metabolic problem management



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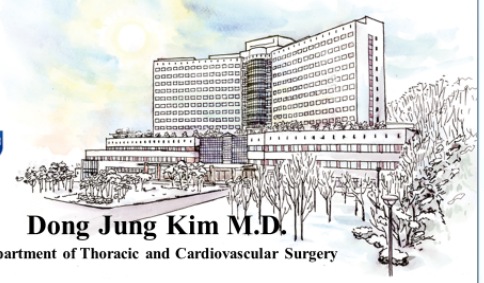
Acute kidney injury

AKI : management

- Patency of Foley catheter
  - hematuria, clamping
  - irrigation with saline, if necessary
  - consideration of changing catheter empirically
- Bladder scan
  - indicate whether oliguria is real or spurious
    - if Foley catheter has been removed
  - significant post-void residual
    - provide evidence of a post-obstructive uropathy

Fluid, renal and metabolic problem management



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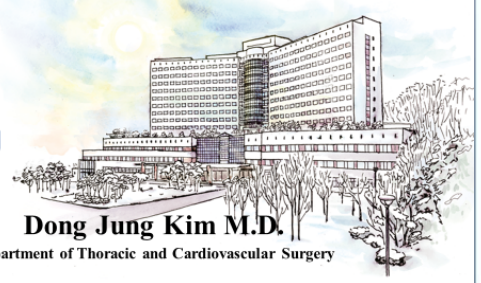
Acute kidney injury

AKI : management

- Optimize cardiac function
  - treat hypovolemia (optimize preload)
  - control arrhythmias
  - improve contractility
  - reduce elevated afterload
    - allow BP to drift up to 150mmHg

Fluid, renal and metabolic problem management



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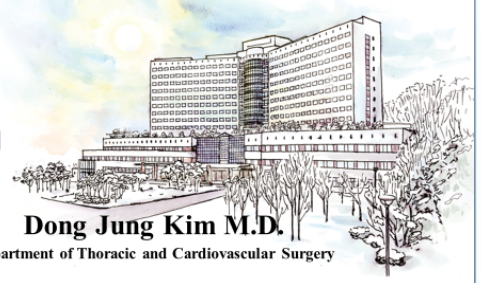
Acute kidney injury

AKI : management

- Optimize preload
  - hemodynamic monitoring with Swan-Ganz catheter
  - echocardiography
    - ventricular failure VS tamponade or hypervolemia
  - assessment of fluid balance & intravascular volume
    - fluid responsiveness, CVP monitoring, strict I & O's
  - avoid excessive fluid administration
    - noncardiogenic pulmonary edema
    - capillary leak
      - sepsis, long duration of CPB, persistent low cardiac output state
    - ↓ oncotic pressure
      - hemodilution, poor nutritional condition

Fluid, renal and metabolic problem management



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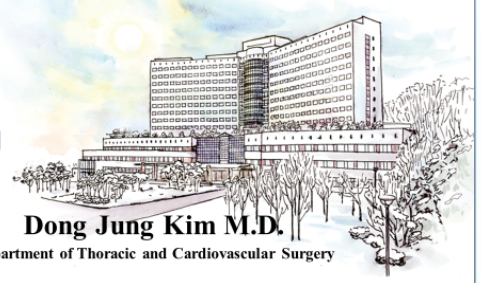
Acute kidney injury

AKI : management

- Optimize HR & treat arrhythmias
  - increase rate with pacing to augment cardiac output
    - even though patient have satisfactory HR
  - beneficial in improving renal perfusion & GFR
- Improve contractility with inotropes
  - if low cardiac output state is present
- IABP
  - low cardiac output despite use of multiple inotropes
  - increase in urine output

Fluid, renal and metabolic problem management



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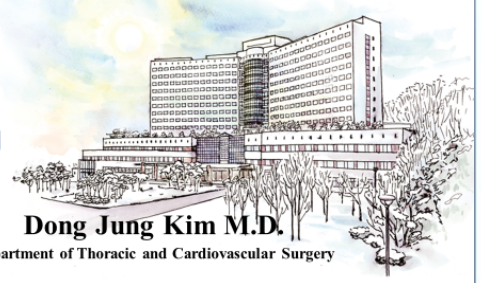
Acute kidney injury

AKI : management

- Reduce afterload with vasodilators
  - milrinone or dobutamine
    - inotropic drugs with vasodilator properties
  - avoid drugs that can cause renal vasoconstriction
    - ACE inhibitors & ARBs
  - avoid aggressive in the reduction of systemic BP
    - in patients with preexisting hypertension & CKD
      - usually require higher sBP (130~150mmHg) to maintain renal perfusion
    - α-agent (norepinephrine)
      - may be necessary to maintain systemic BP
      - it will also provide some inotropic support

Fluid, renal and metabolic problem management



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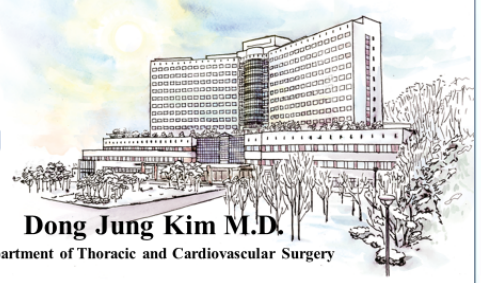
Acute kidney injury

AKI : management

- Optimize hemodynamics
  - augmentation of cardiac output
    - to prevent additional insult that cause hypoperfusion
  - prevention of protracted ARF
    - hypovolemia (often GI bleeding)
    - low cardiac output states (tamponade)
    - arrhythmia (rapid atrial fibrillation, VT)
    - antihypertensive medications
    - sepsis

Fluid, renal and metabolic problem management



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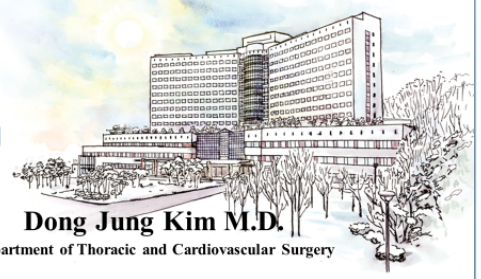
Acute kidney injury

AKI : management

- Diuretics
  - oliguria despite optimization of hemodynamics
- Controversy over loop diuretics
  - con
    - No direct effect on renal functional recovery or natural history of AKI
    - may increase operative mortality and delay recovery of renal function
  - pro
    - may hasten decline in Scr and possibly shorten duration of RRT
    - improve urine output and can convert oliguric to non-oliguric RF
    - minimize adverse impact of fluid retention on pulmonary function

Fluid, renal and metabolic problem management



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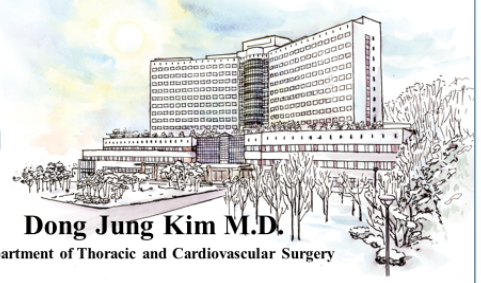
Acute kidney injury

AKI : management

- Diuretic-responsive RF
  - >400 mL/day, the most common form of AKI
  - preexisting renal dysfunction or risk factors
    - occasionally without any precipitating factors
  - usually reflects less renal damage
  - associated with a mortality rate of about 5~10%
    - Non-oliguric RF : mortality rate approaches up to 50%
  - earlier decrease in Scr
    - contributing to improvement in short- and long-term survival

Fluid, renal and metabolic problem management



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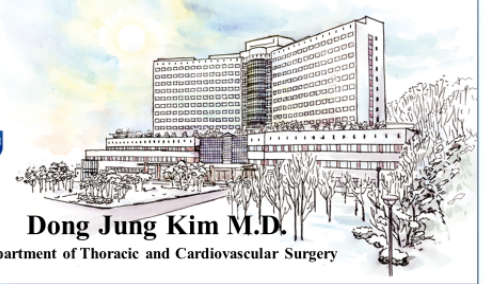
Acute kidney injury

AKI : management

- Furosemide
  - incremental doses starting at 10 mg IV
  - 100 mg IV over 20~30 min to minimize ototoxicity
  - increase up to 200 mg IV
    - if urine output fails to increase within few hours
    - daily cumulative dose to 1 g
  - continuous infusion
    - the best means of maintaining adequate urine output
    - loading dose of 40~100 mg and initiate infusion of 10~20 mg/hr
    - re-bolus before increase in the infusion rate

Fluid, renal and metabolic problem management



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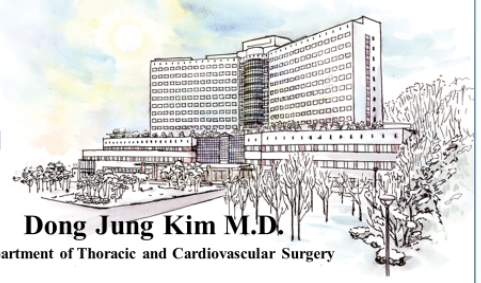
Acute kidney injury

AKI : management

- Mannitol
  - osmotic diuretic
    - frequently used during surgery
      - increase serum osmolality during hemodilution to minimize tissue edema
  - improves urine output
    - improves renal tubular flow, reduces tubular cell swelling
  - avoided in the postoperative period
    - significant increase in serum osmolality
      - can cause renal vasoconstriction and induce renal failure

Fluid, renal and metabolic problem management



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Acute kidney injury

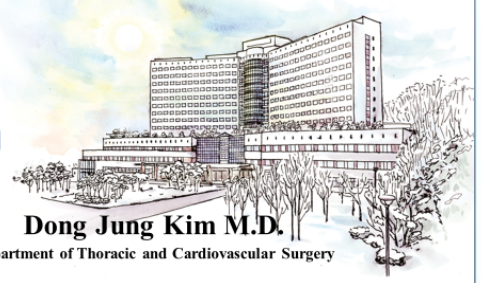
AKI : management

- Renal-dose dopamine
  - 2~3 μg/kg/min
  - no effect on duration of AKI, need for dialysis, survival
  - comparisons with dobutamine
    - dopamine increased urine output without improving Ccr, whereas dobutamine did just the opposite

      (Crit Care Med 1994;22:1919–25)
    - dopamine produced diuresis and improved Ccr unrelated to any hemodynamic effects, whereas dobutamine had no effect

      (Crit Care Med 2000;28:921–8)

Fluid, renal and metabolic problem management



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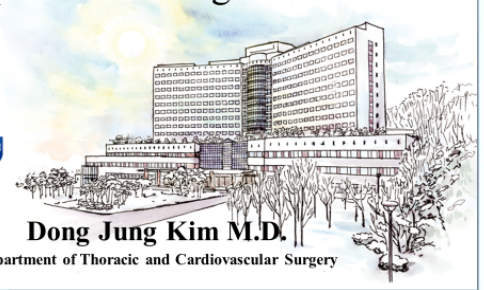
Acute kidney injury

AKI : management

- Renal dose dopamine + furosemide
  - renal vasodilation and improved RBF
    - produced by dopamine improve delivery of furosemide

- Mannitol + furosemide + renal dose dopamine
  - start within 1st 6 hours of oliguria
  - significant diuresis
  - early restoration of renal function

Fluid, renal and metabolic problem management



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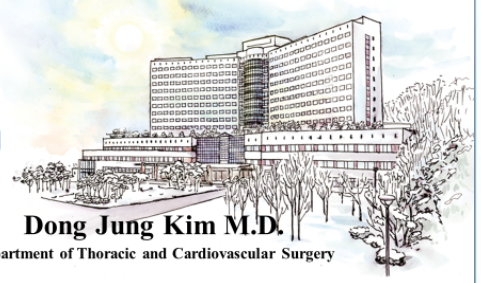
Acute kidney injury

AKI : management

- Limit fluid to insensible losses
- Readjust drug doses
  - discontinue all potentially nephrotoxic drugs
    - ACE inhibitors, ARBs, NSAIDs, nephrotoxic antibiotics
  - avoid any diagnostic studies requiring IV contrast
- Avoid potassium supplements
- Consider early renal replacement therapy

Fluid, renal and metabolic problem management



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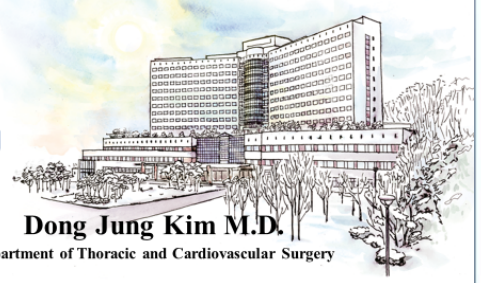
Renal Replacement Therapy

- Indications
  - volume overload, hyperkalemia, and metabolic acidosis
  - signs of uremia
    - such as a change in mental status
- Early and aggressive dialysis
  - before patient develops signs and symptoms of RF
  - before marked elevation in Scr occurs
  - marked oliguria in a patient with significant fluid overload
    - delay in initiating RRT may lead to respiratory compromise and prolonged ventilation
  - might improve outcomes

Fluid, renal and metabolic problem management



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Renal Replacement Therapy

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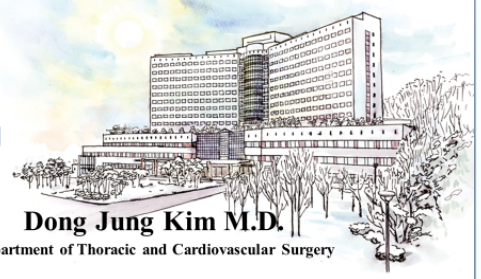
If the Patient Has:	HD	SCUF	CVVH	CVVHD
Unstable hemodynamics	–	+ + +	+ + +	+ + +
Contraindication to heparin	+ +	+	+	+
Vascular access problems	+ + +	+ + +	+ + +	+ + +
Volume overload	+ +	+ + +	+ + +	+ + +
Hyperkalemia	+ + +	0	+ +	+ + +
Severe uremia	+ + +	0	+	+ +
Respiratory compromise	+ +	+ + +	+ + +	+ + +

HD, hemodialysis; SCUF, slow continuous ultrafiltration; CVVH, continuous venovenous hemofiltration; CVVHD, continuous venovenous hemofiltration with dialysis.

– avoid; 0 minimal effect; + useful; + + better; + + + even better.

CVVHDF

Fluid, renal and metabolic problem management



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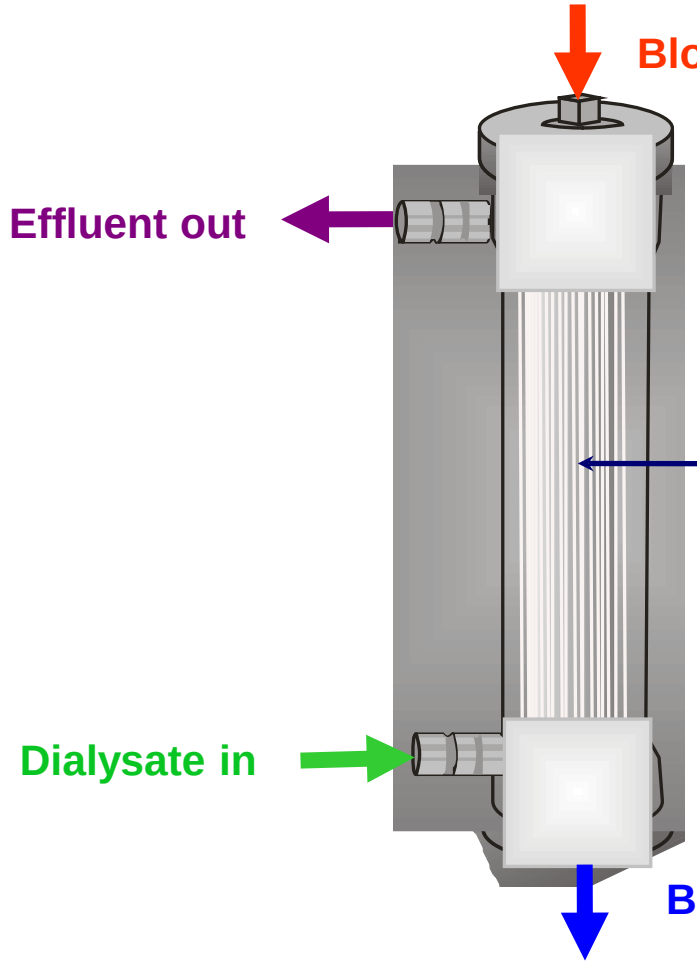
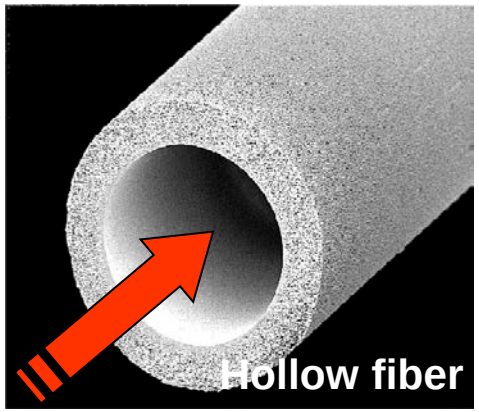
Continuous RRT





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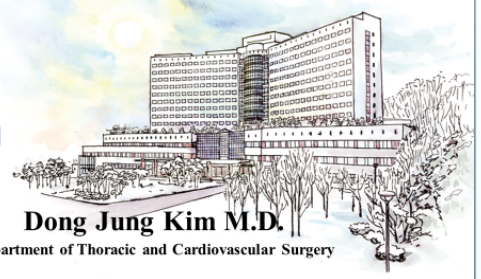

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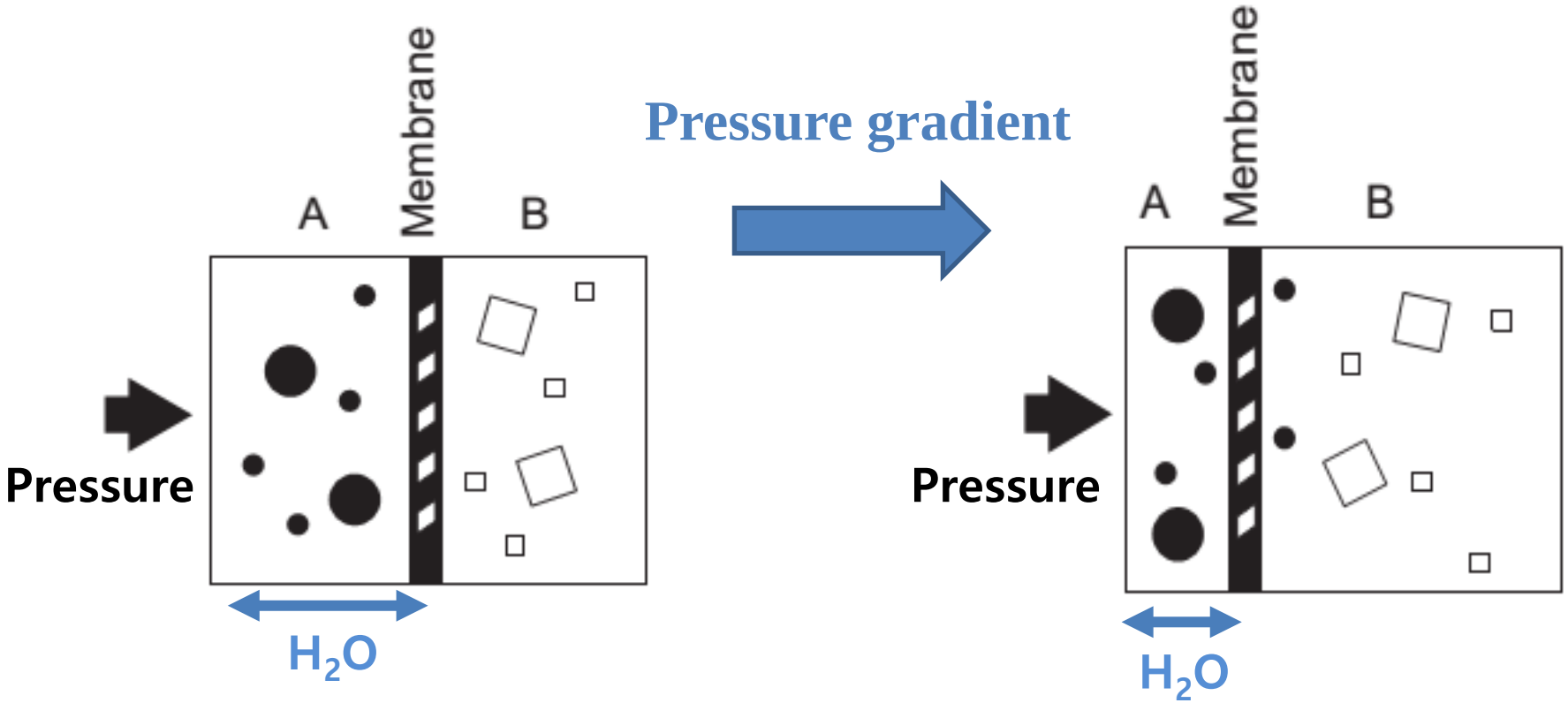
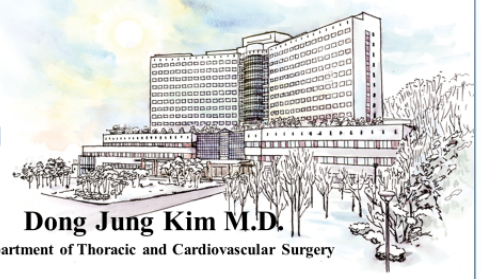
Ultrafiltration and Convection



Fluid, renal and metabolic problem management



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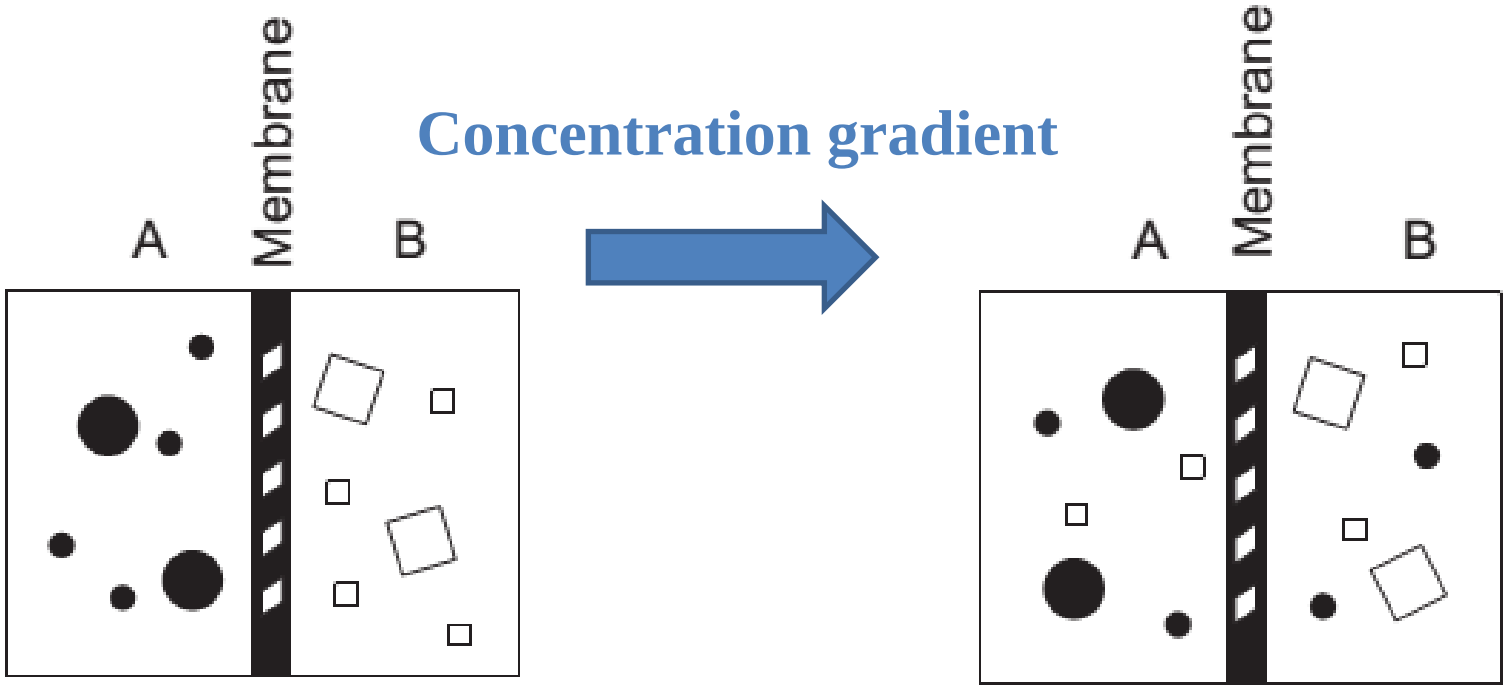
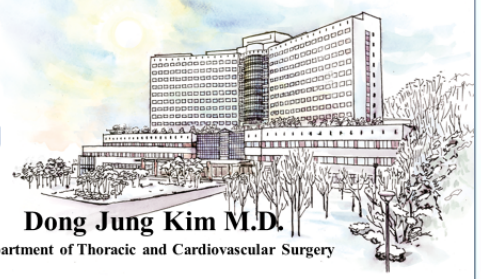
Diffusion



Fluid, renal and metabolic problem management



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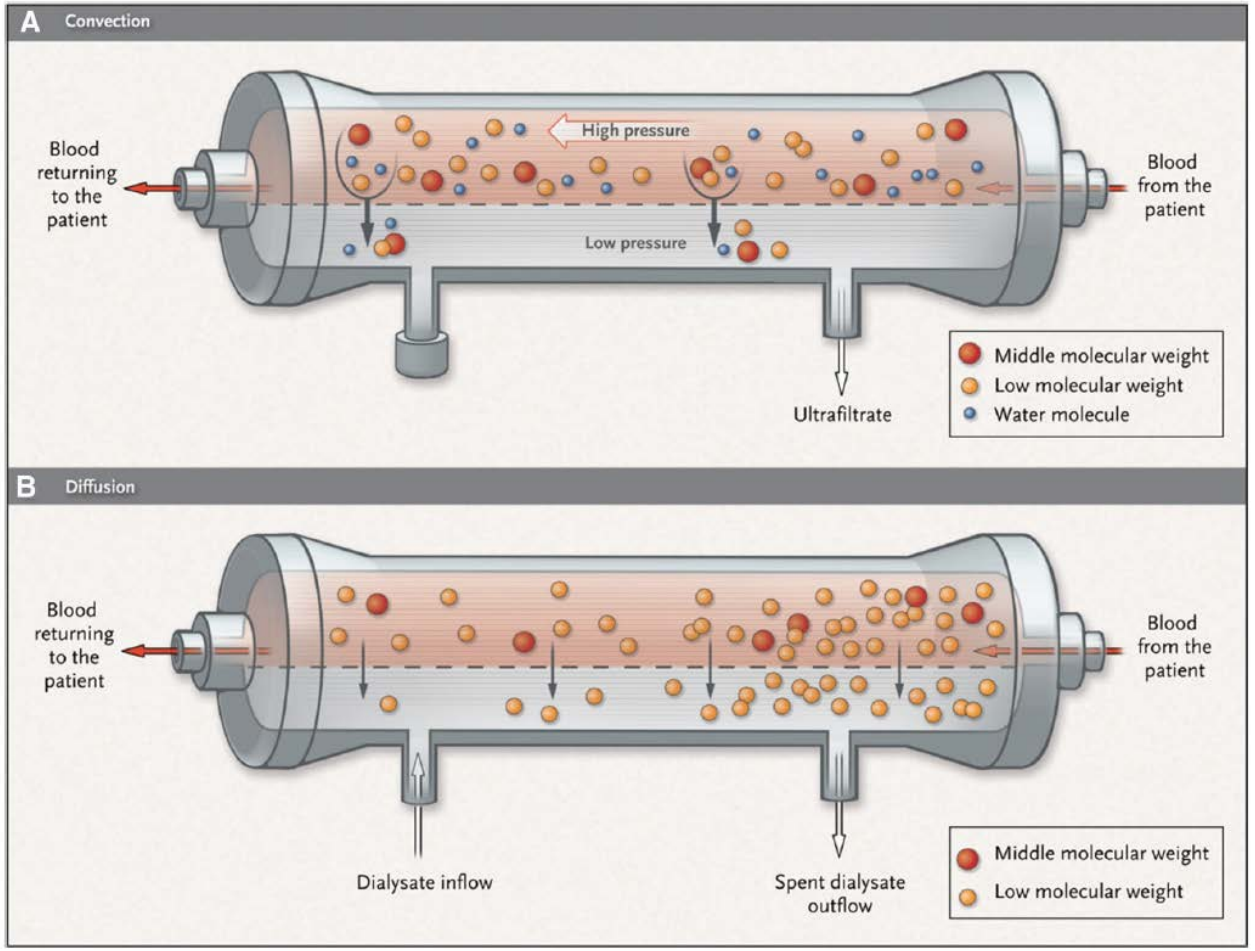
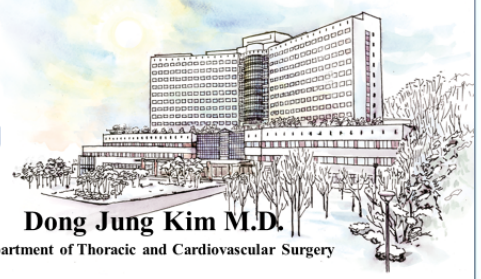
Convection vs Diffusion



(Can J Anesth/J Can Anesth (2019) 66:593–604)

Fluid, renal and metabolic problem management



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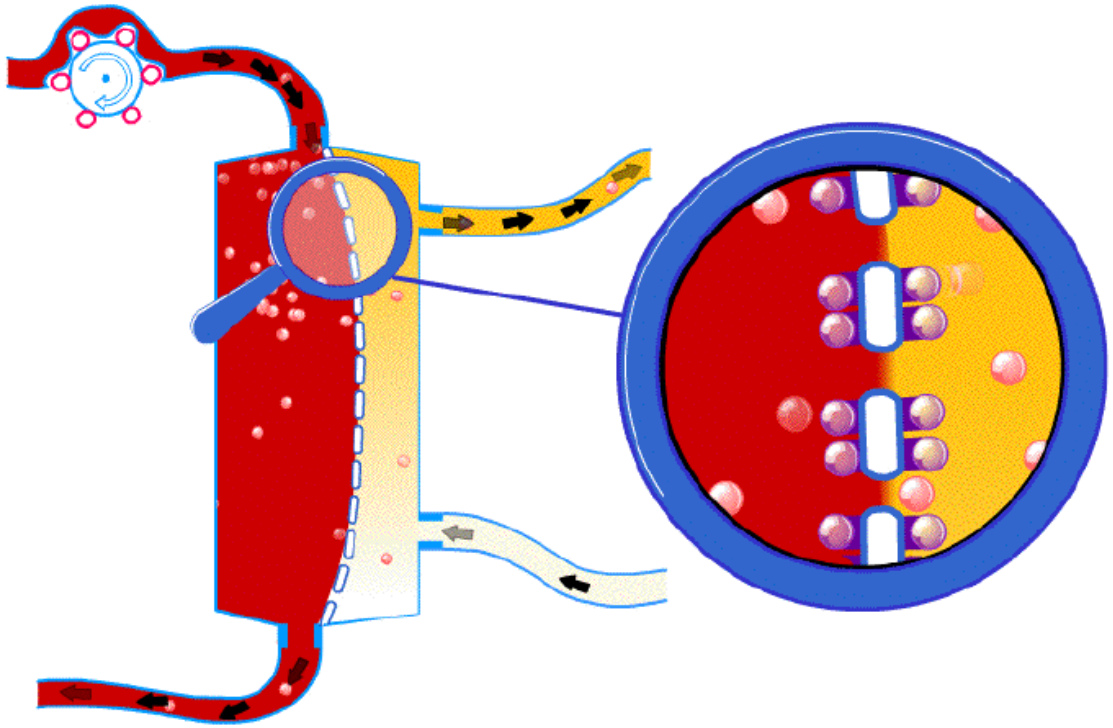
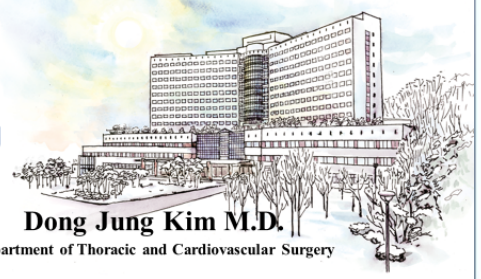
Absorption



Adherence to surface semi-permeable membrane

Fluid, renal and metabolic problem management



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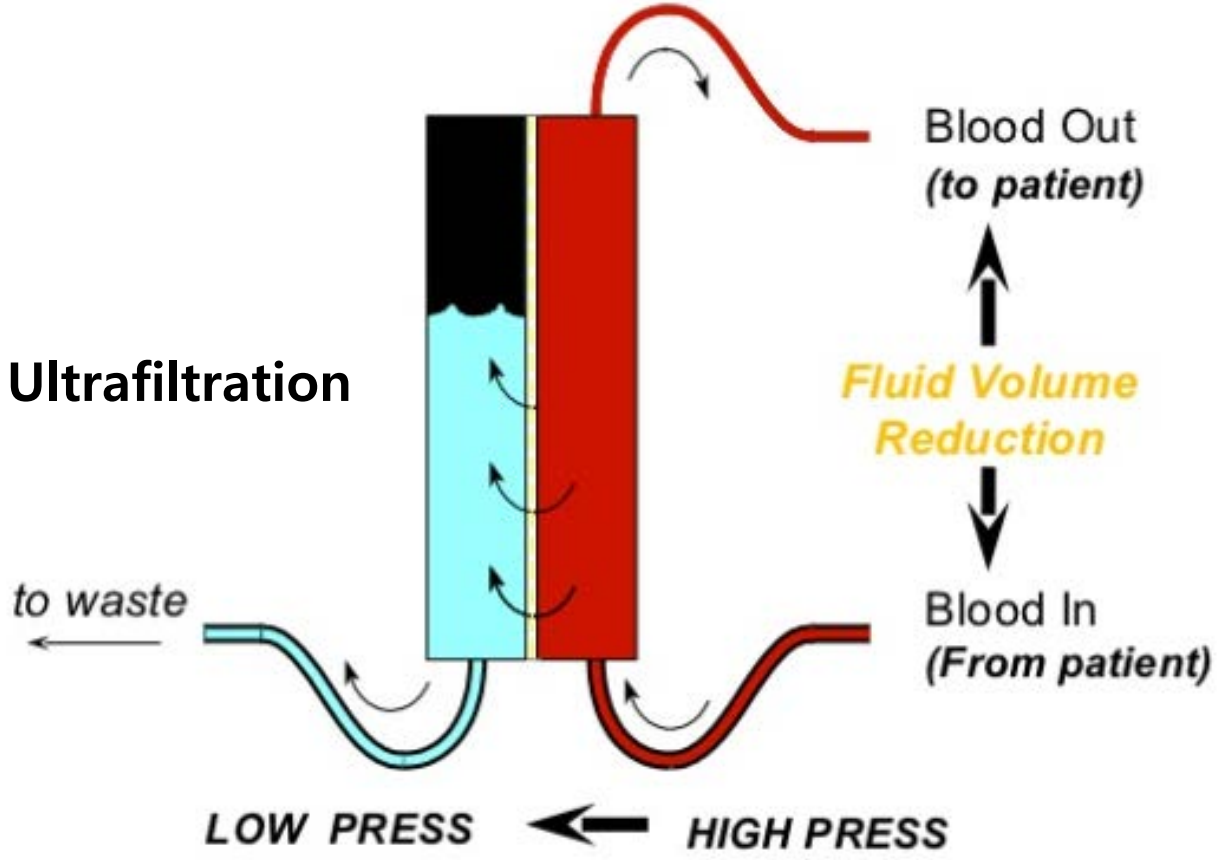
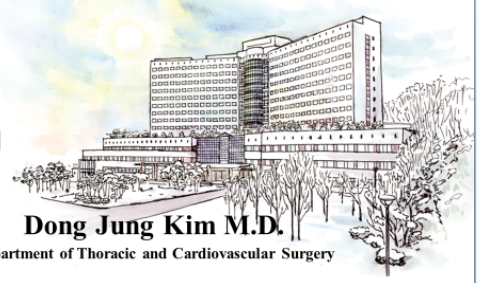
SCUltraFiltration



Fluid, renal and metabolic problem management



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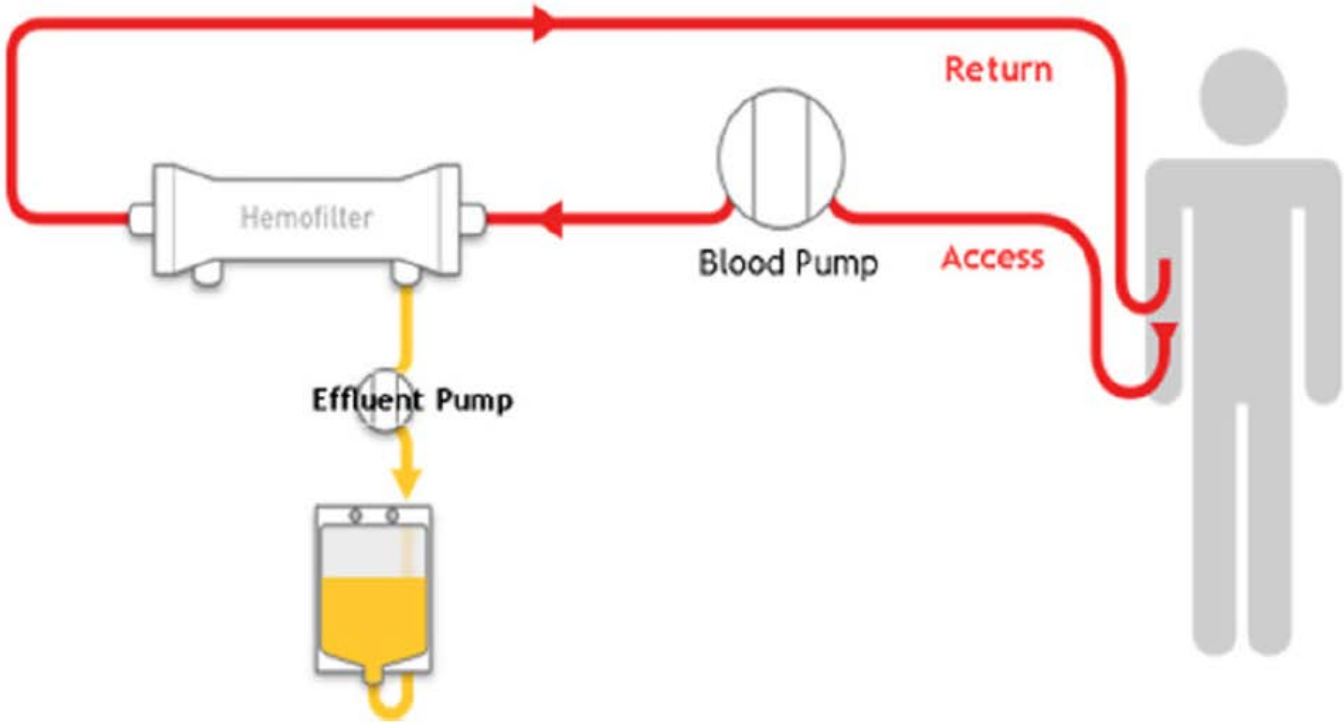
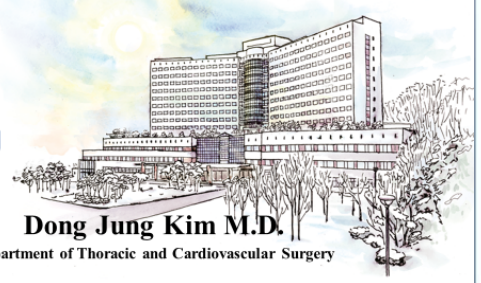


Fluid, renal and metabolic problem management



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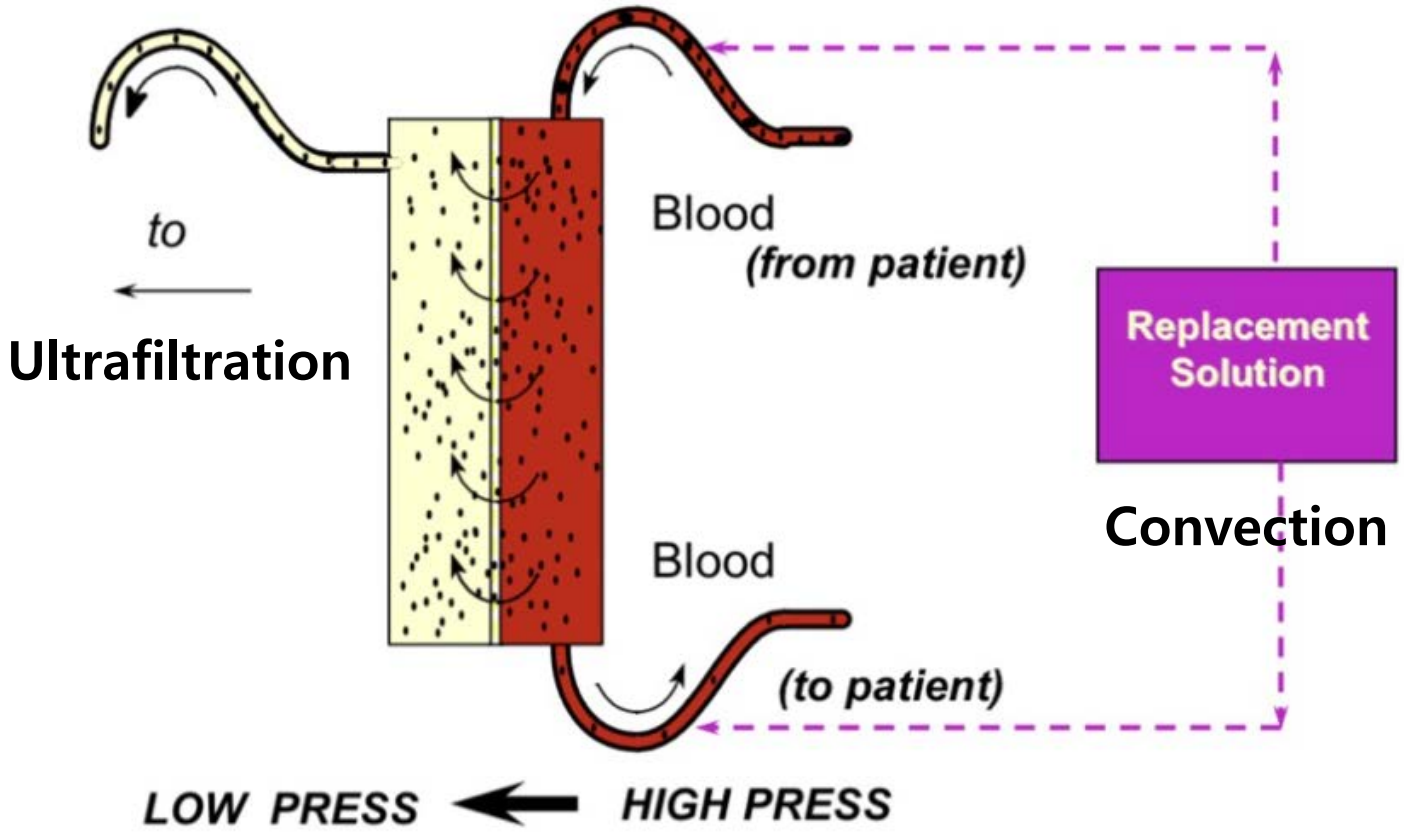
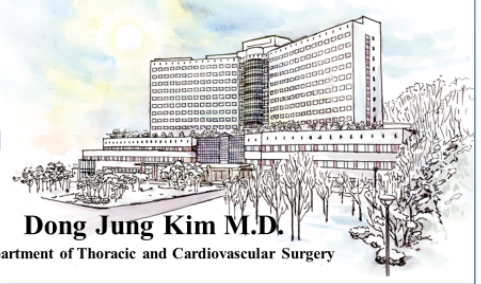
CVVHemofiltration



Fluid, renal and metabolic problem management



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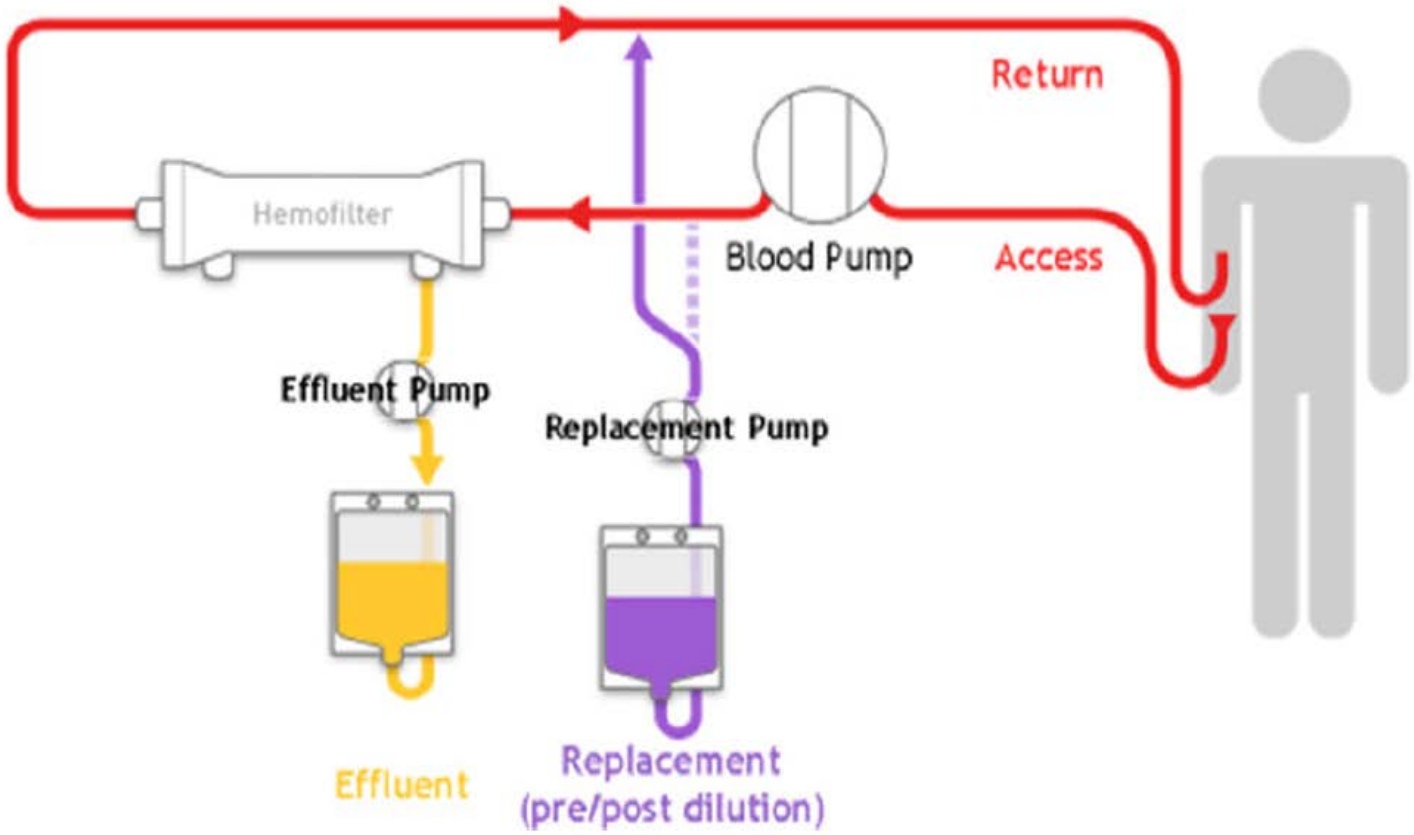

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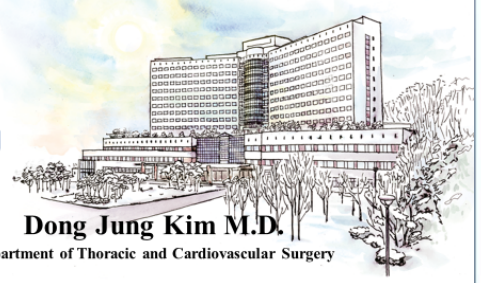

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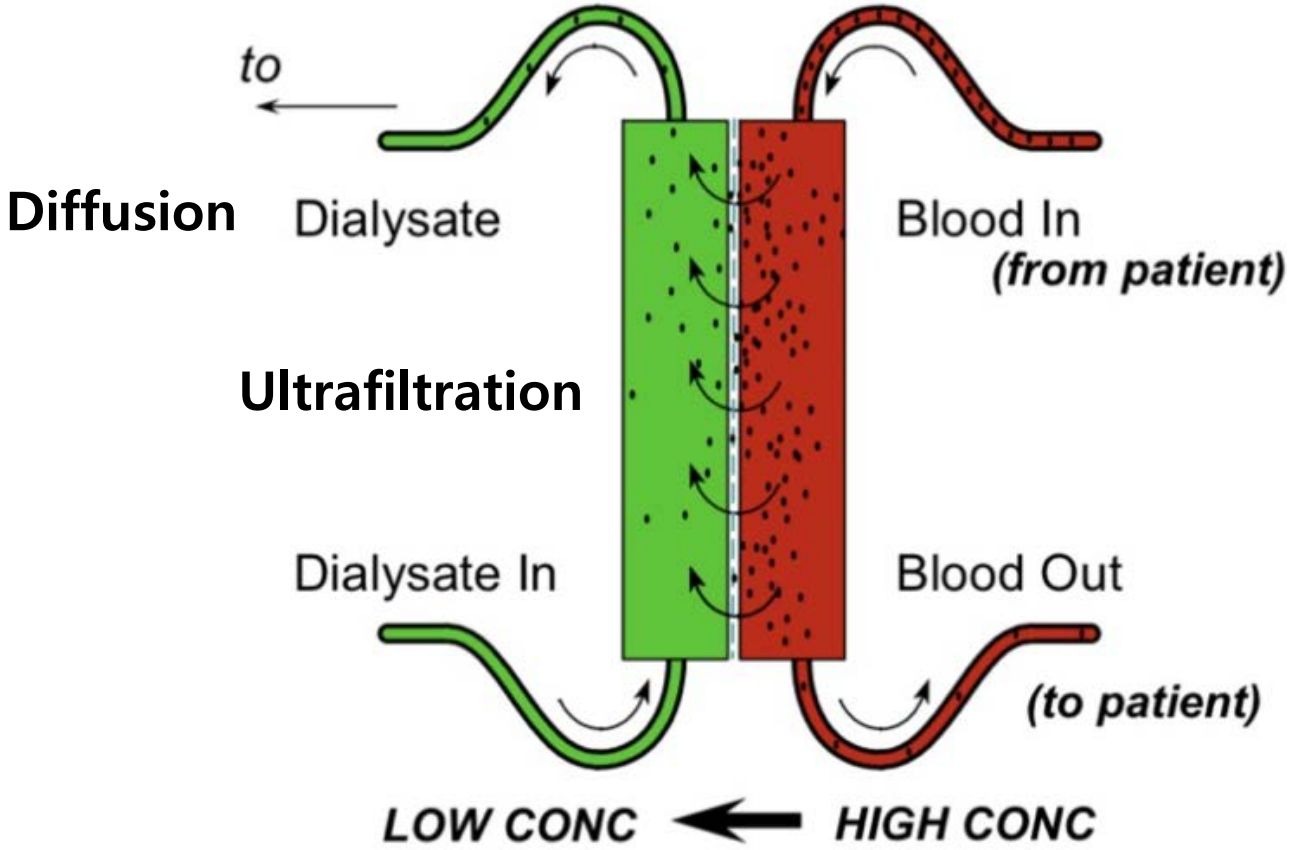
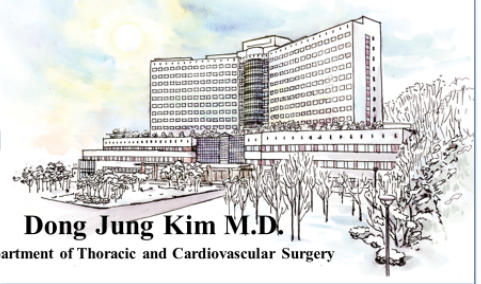
CVVHemoDialysis



Fluid, renal and metabolic problem management



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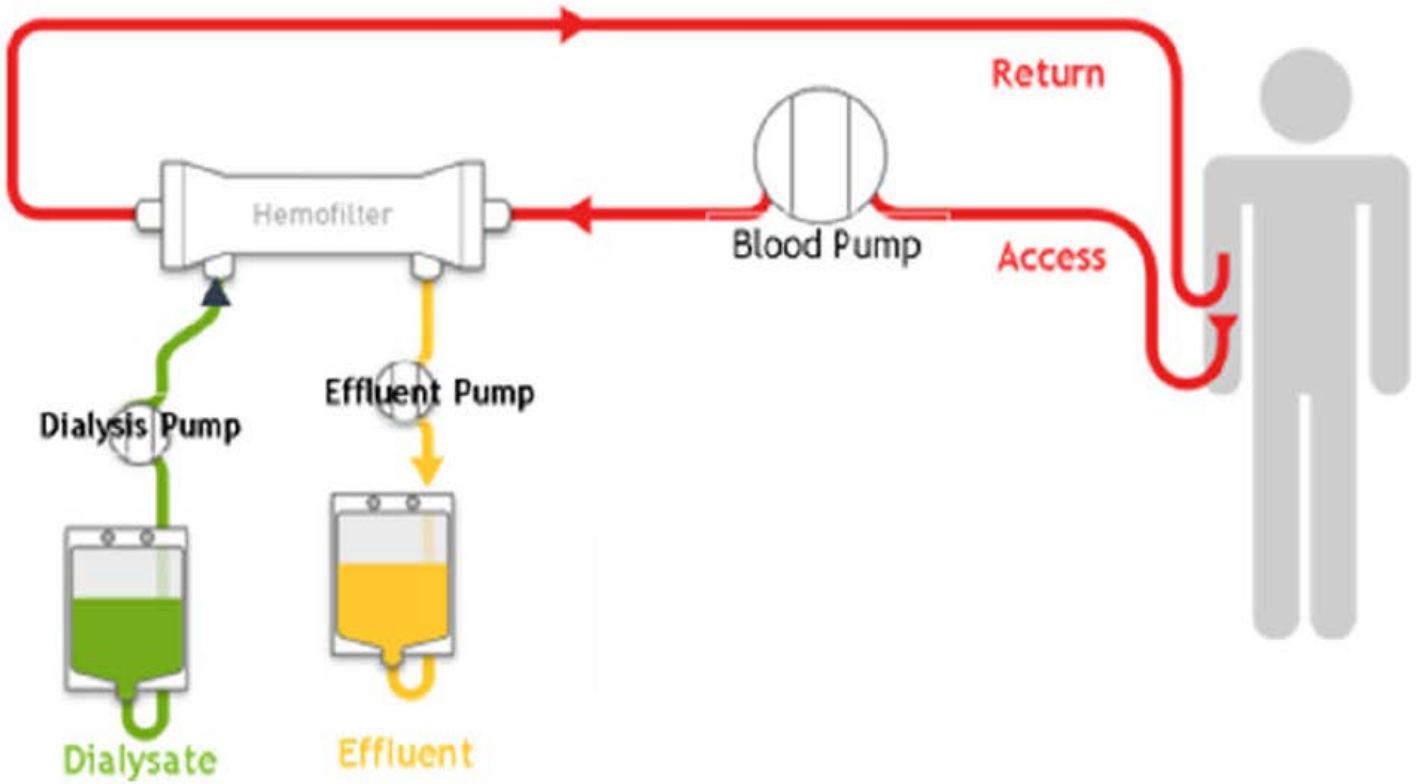
CVVHemoDialysis



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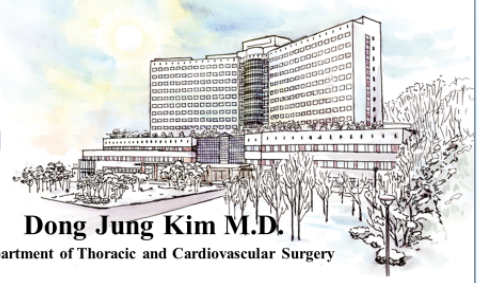

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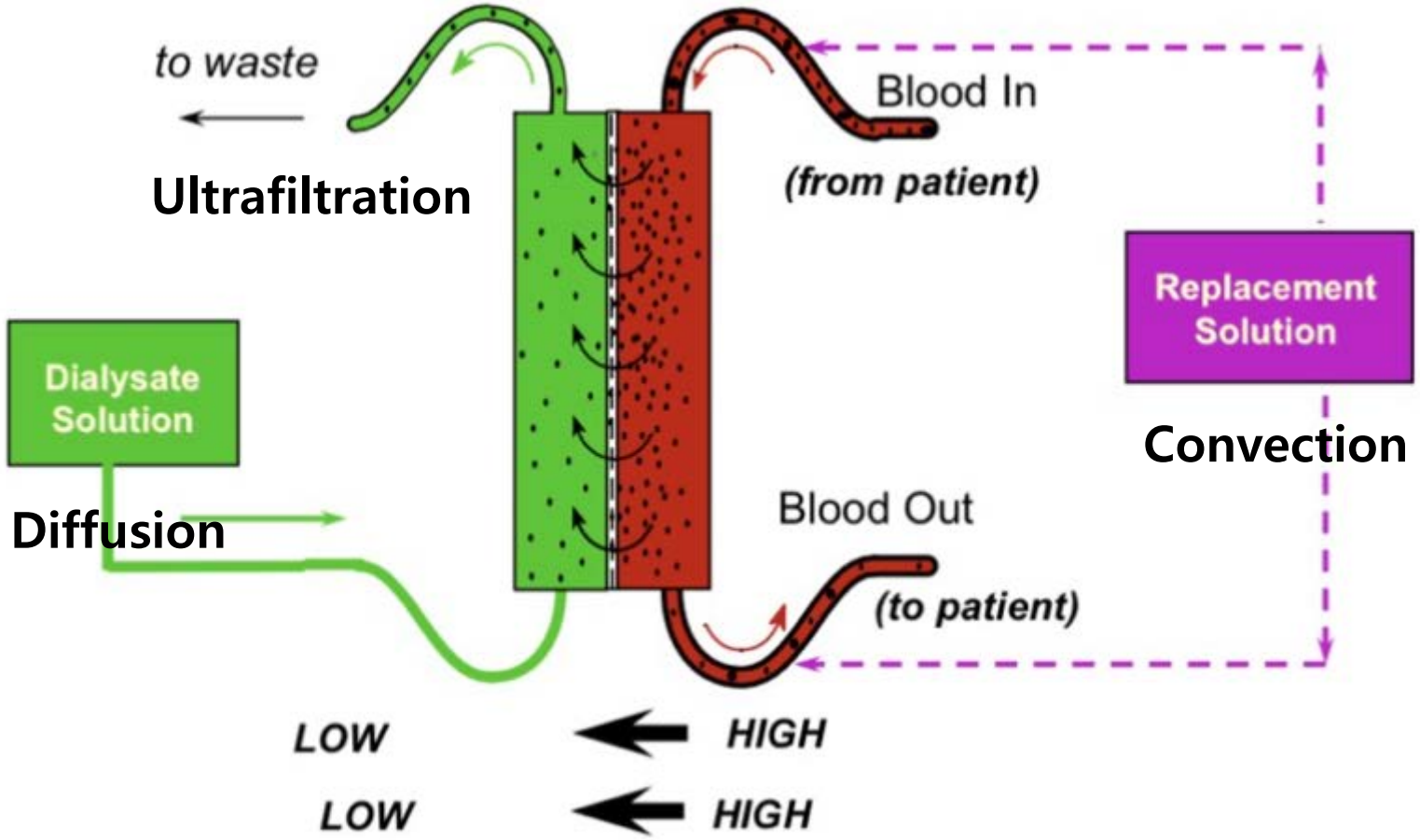
CVVHemoDiaFiltration



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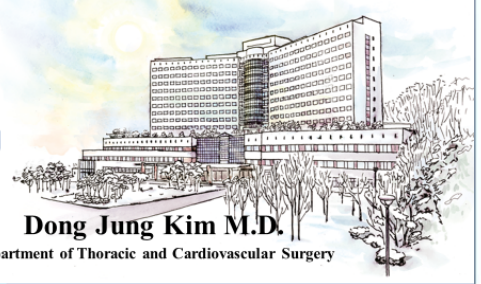

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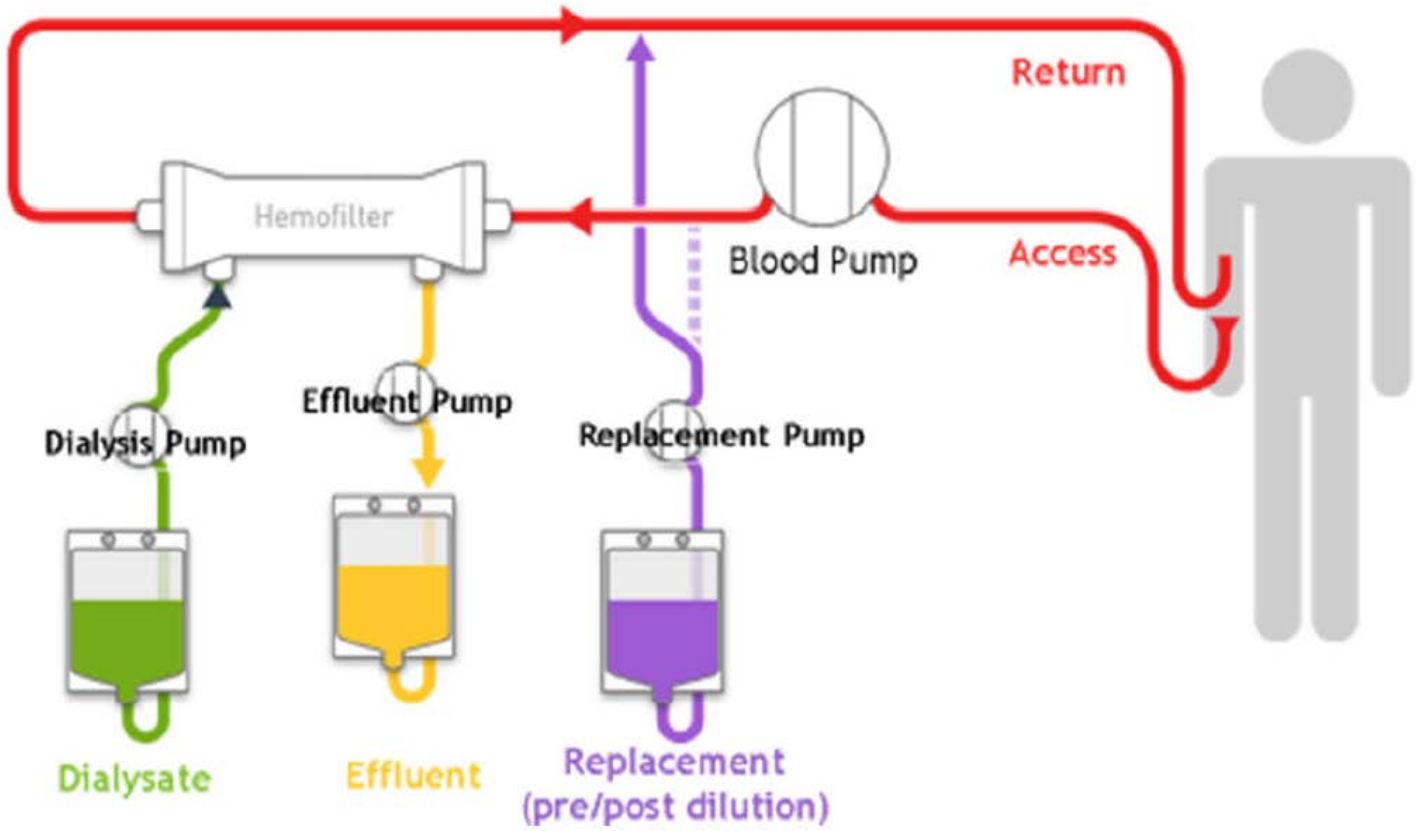
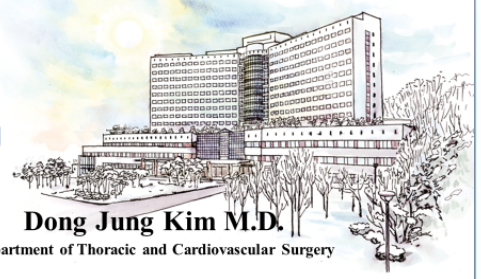


Fluid, renal and metabolic problem management



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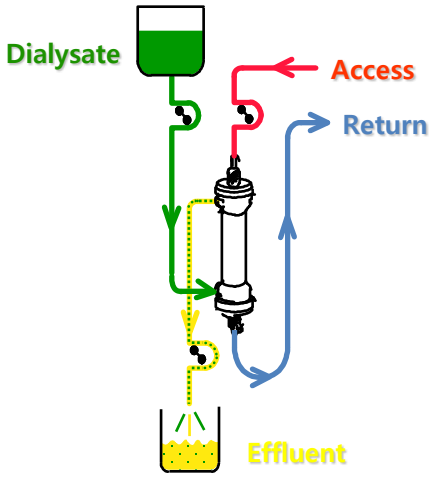
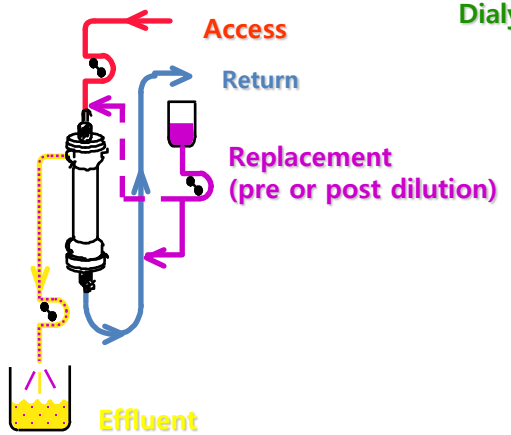
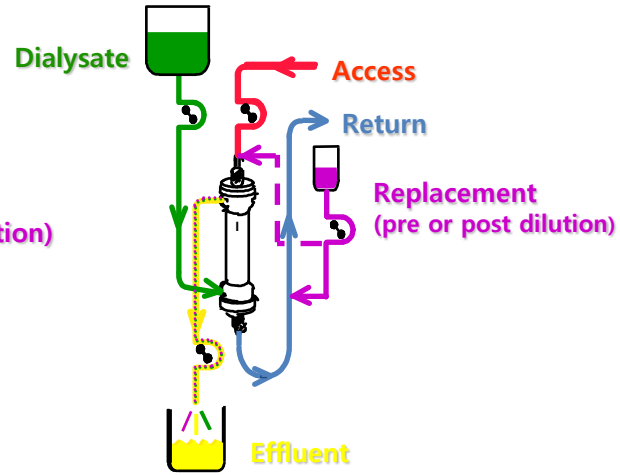
CRRT : mode







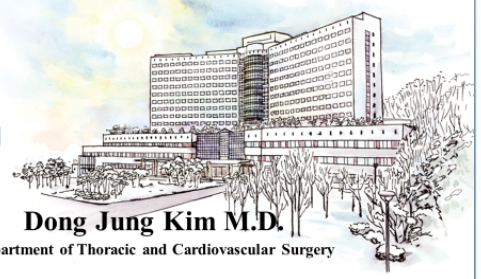
CVVHD + **CVVH** = **CVVHDF**

- **Ultrafiltration**
- **Diffusion**

- **Ultrafiltration**
- **Convection**

- **Ultrafiltration**
- **Diffusion**
- **Convection**

Fluid, renal and metabolic problem management



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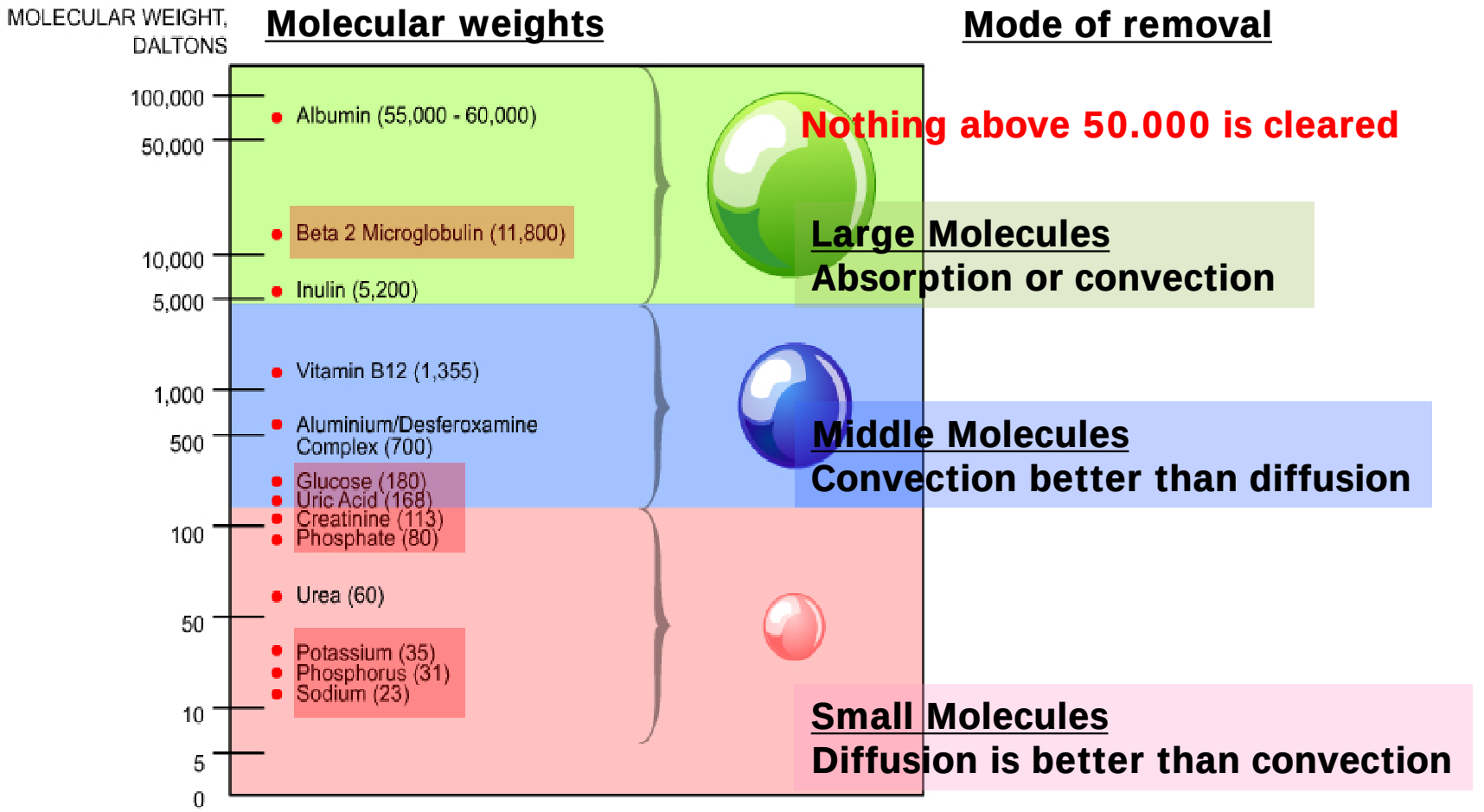
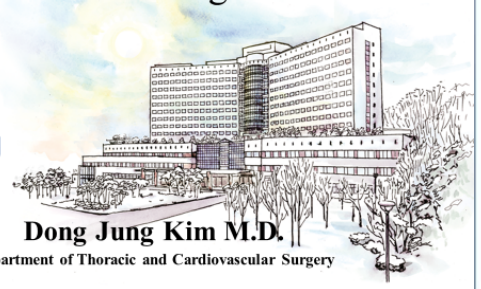
CRRT

CRRT : molecules



Fluid, renal and metabolic problem management



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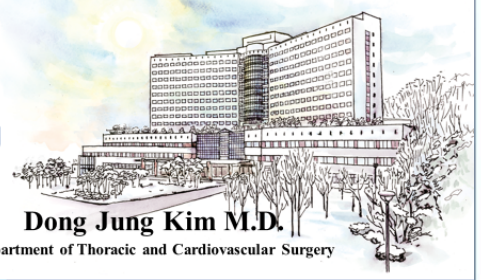
CRRT

CRRT : order

≪정규≫	▶ CRRT ◀	
기본	(1) Blood flow : (100) ml/min	처음 1시간 동안은 100ml/hr, 이후 SBP > 100mmHg 이면 150 ml/hr 까지 상향조정.
	(2) Dialysate : (1000) ml/hr	
	(3) Replacement : (1000) ml/hr	pre-dilution (1000) ml/hr
	(4) Removal (100)ml/hr	처음 1시간 동안은 0, 이후 SBP > 90mmHg 이면 상향조정.
	(5) Anticoagulation	
수액	◆6 Futhan 50mg(SK) 《Nafamostat Mesilate》	4 VL IVF 2 회(수액에 혼합하여 IV infusion) 1000mL/hr(333gtt)(병동Mix) [♣출혈위험♣]
	◆6 5% DW 100mL/BTL(대한) 《Dextrose》	40 ML IVF 2 회(수액에 혼합하여 IV infusion) 1000mL/hr(333gtt)(병동Mix)
기본	-4 ml/hr의 속도로 시작	
	-arterial line에서 ACT 6시간 간격으로 시행	(180 ~ 200sec로 유지)
	-V-ACT > 200 : futhan 1 ml/hr씩 감소시킴	
	-V-ACT < 180 : futhan 1 ml/hr씩 증가시킴	
	(6) Lab F/U	

Fluid, renal and metabolic problem management



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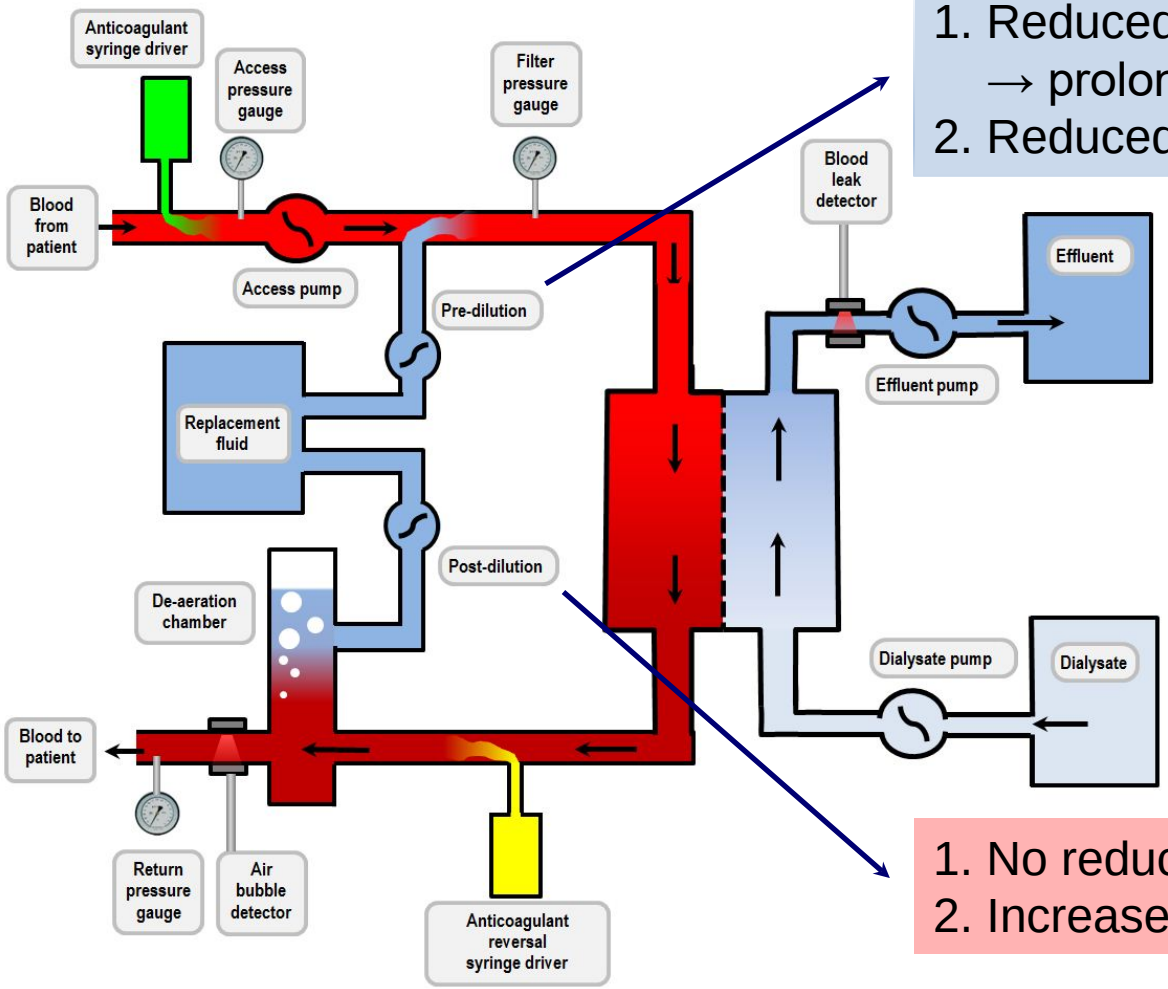
CRRT

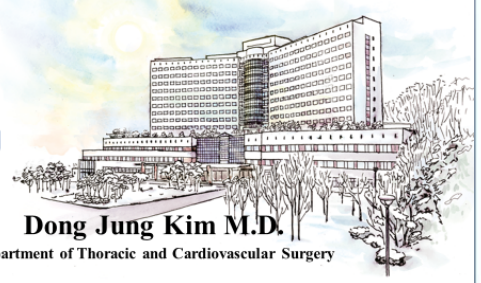
CRRT : pre- or post-dilution



1. Reduced filter clotting
   → prolonged filter life span
2. Reduced effective clearance

1. No reduction of clearance
2. Increased need of anticoagulant

Fluid, renal and metabolic problem management



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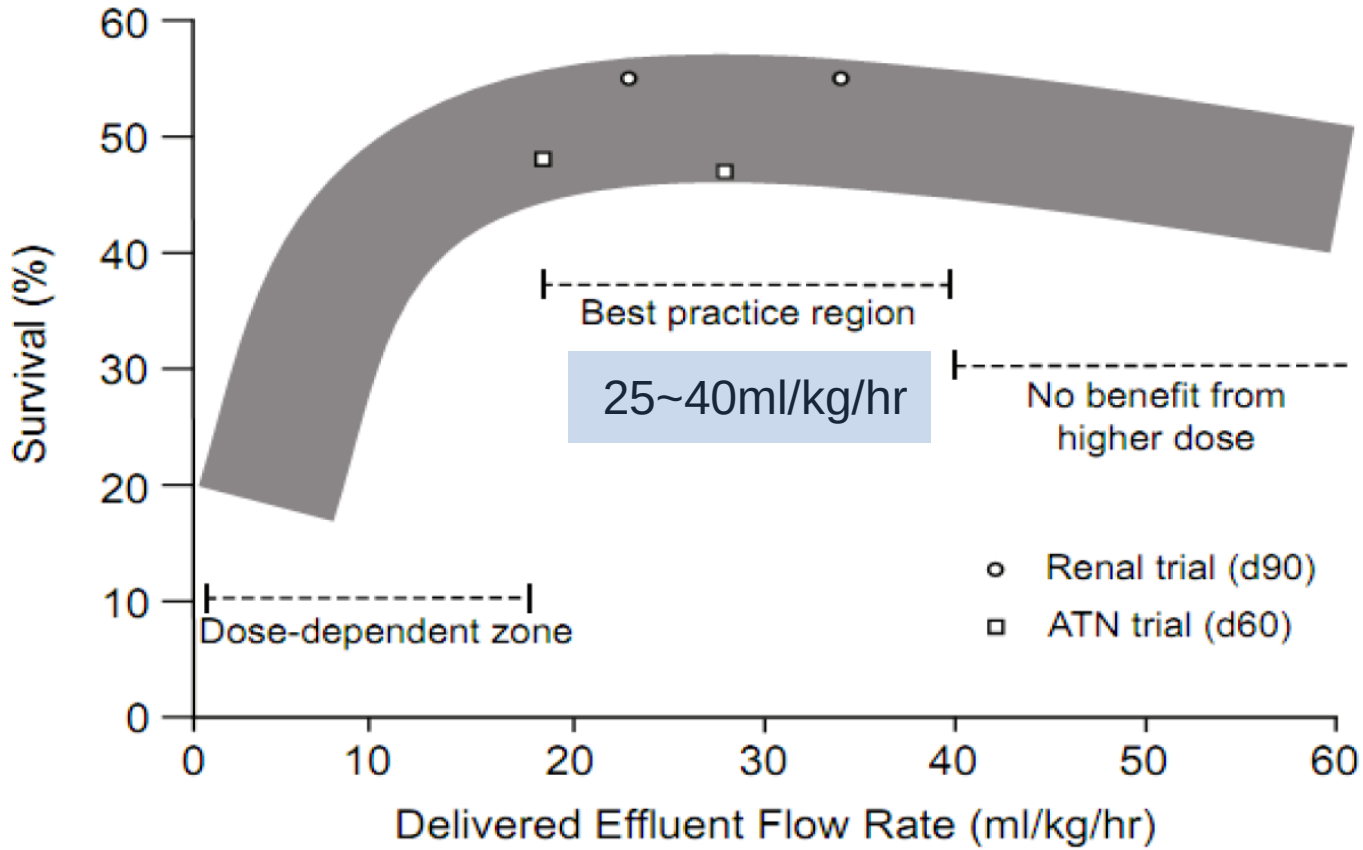
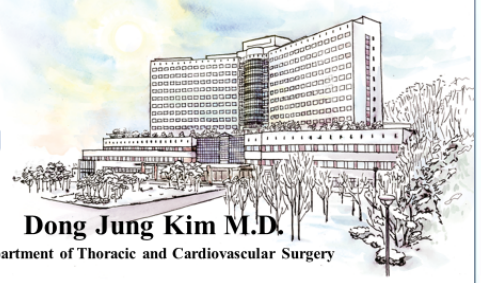
CRRT

CRRT : dose



(Can J Anesth/J Can Anesth (2019) 66:593–604)

Fluid, renal and metabolic problem management



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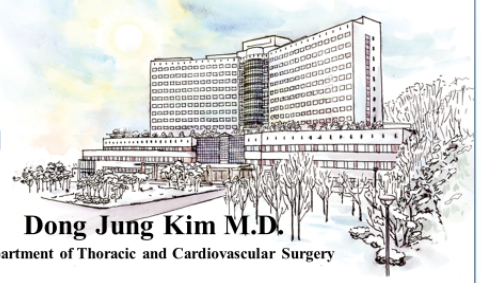
CRRT

CRRT : case – initial setup

- Bwt : 60kg
- Mode : CVVHDF
- Blood flow : 100ml/min
- Effluent flow rate (dose) : 40ml/kg/hr
- Flow rate (dialysate + replacement = 1 : 1)
  - 60kg × 40ml/kg/hr = 2400ml/hr
  - dialysate = 1200ml/hr
  - replacement = 1200ml/hr (pre : post = 1 : 1)
    - pre = 600ml/hr, post = 600ml/hr

Fluid, renal and metabolic problem management



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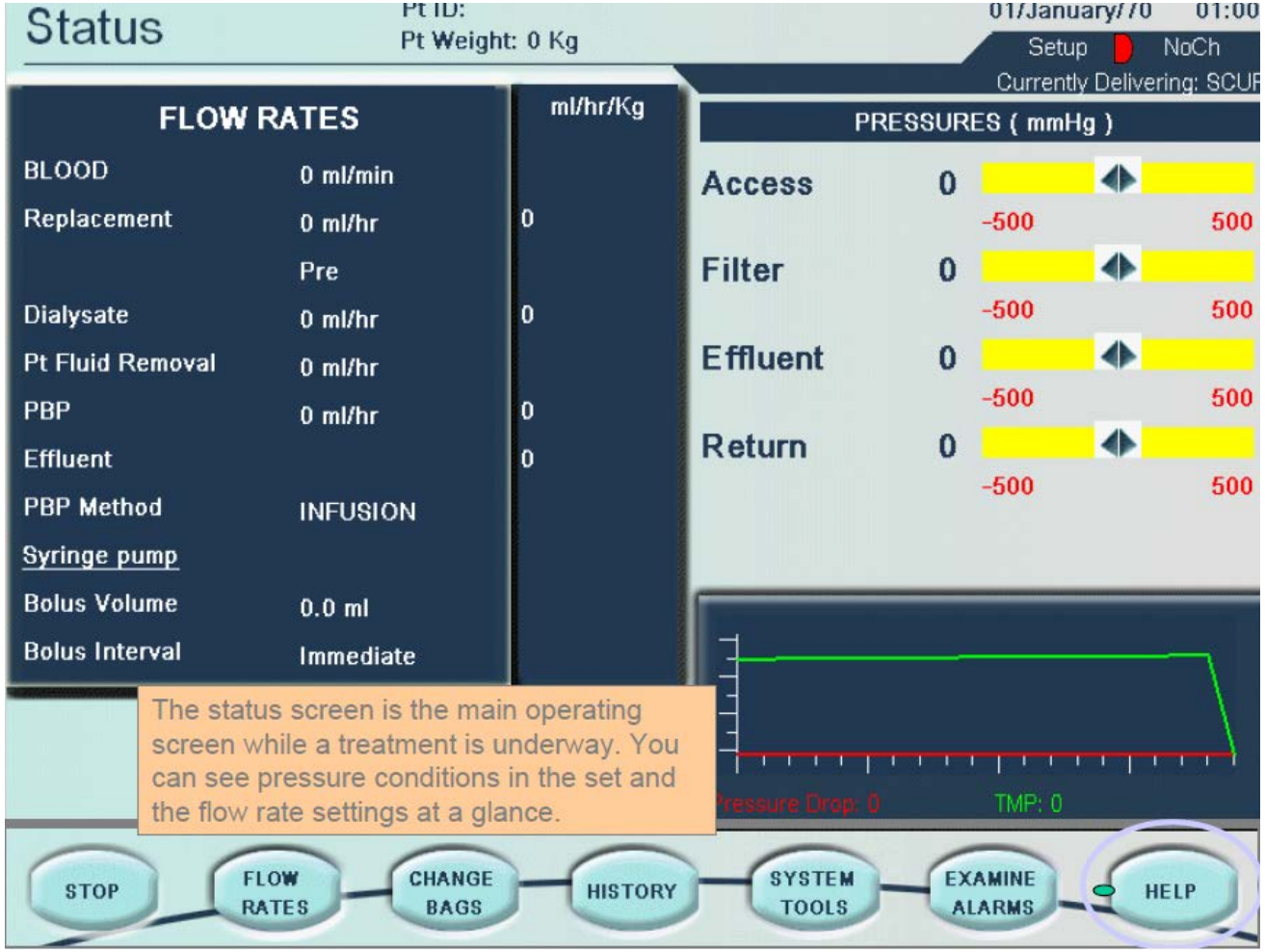
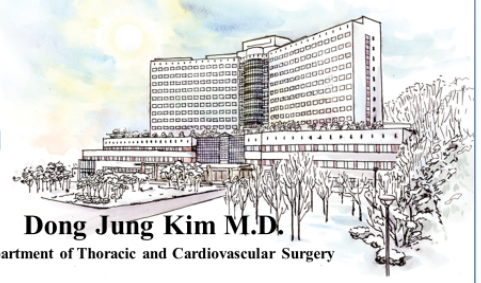
CRRT

CRRT : setup



Fluid, renal and metabolic problem management



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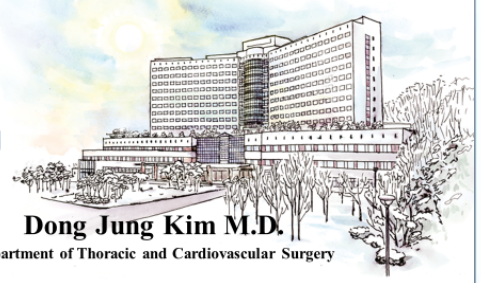
Hyperkalemia

Hyperkalemia : Etiology

- High-volume, high-potassium cardioplegia solutions
  - K^+ load is usually eliminated promptly by normally functioning kidneys
  - problematic in patients with intrinsic renal dysfunction or oliguria from other causes
- Low cardiac output
  - K^+ levels may rise with alarming and life-threatening rapidity
- Severe tissue ischemia
  - peripheral (from severe peripheral vascular disease or complication of IABP)
  - intra-abdominal (mesenteric ischemia)
  - Hyperkalemia is often the 1st clue to existence of these problems
- Acute and chronic renal insufficiency
- Medications
  - impair K^+ excretion or increase K^+ levels
  - ACE inhibitors, potassium-sparing diuretics, NSAIDs, ARBs, β-blockers

Fluid, renal and metabolic problem management



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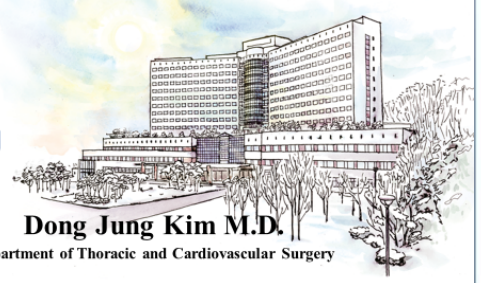
Hyperkalemia

Hyperkalemia : acidosis

- Exacerbated by acidosis
  - low cardiac output or ischemic syndromes
- 0.2unit change in pH
  - about 1mEq/L change in serum K^+ concentration
- Organic acidosis
  - lactic acidosis
    - from tissue breakdown & release of K^+ from cells
  - ketoacidosis
    - from insulin deficiency & hyperglycemia

Fluid, renal and metabolic problem management



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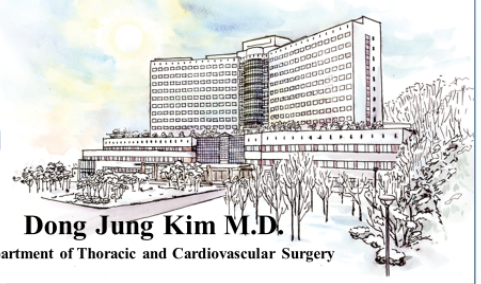
Hyperkalemia

Hyperkalemia : manifestations

- ECG changes
  - d/t depolarization of cardiac cell resting membrane potentials that decreases membrane excitability
  - do not always develop in classic progressive fashion
  - more related to rate of rise of serum K^+ than to absolute level
  - asystolic arrest
    - when K^+ rises rapidly to level exceeding 6.5 mEq/L
  - peaked T waves, ST depression, smaller R waves, prolonged PR interval, loss of P waves, QRS widening, bradycardia, Vf.
- Failure to respond to pacemaker stimulus

Fluid, renal and metabolic problem management



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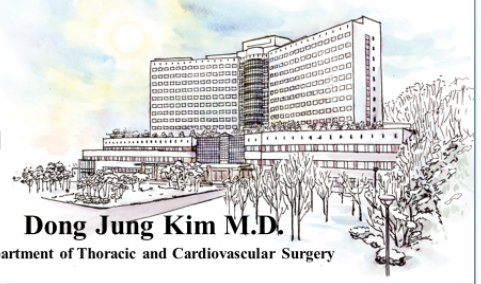
Hyperkalemia

Hyperkalemia : treatment

- Principle
  - stabilize cell membrane
  - shift K^+ into cells
  - increase K^+ excretion
  - identify and remove any potential source of K^+
    - diet or medications that may increase K^+ level

Fluid, renal and metabolic problem management



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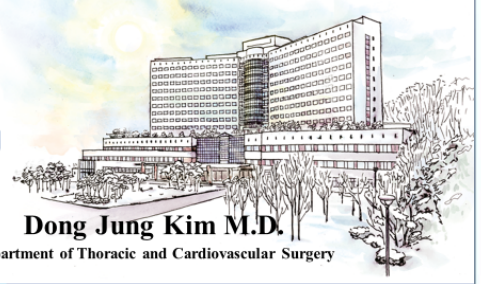
Hyperkalemia

Hyperkalemia : treatment

Medication	Dosage	Onset of Action	Duration of Action
Calcium gluconate	10 mL of 10% solution over 2–3 min	Immediate	30 min
Insulin	10 units regular insulin IV in 50 mL of 50% dextrose	15–30 min	2–6 hours
Sodium bicarbonate	1 amp 7.5% (44.6 mEq)	30 min	1–2 hours
Albuterol	10–20 mg by nebulizer	90 min (peak effect)	2–3 hours
Furosemide	20–40 mg IV	15–60 min	4 hours
Sodium polystyrene sulfonate (Kayexalate)	Oral: 30 g in 60–120 mL sorbitol PR: 50 g in retention enema	1–2 hours	4–6 hours

(Lancet 2008;372:1863–5)

Fluid, renal and metabolic problem management



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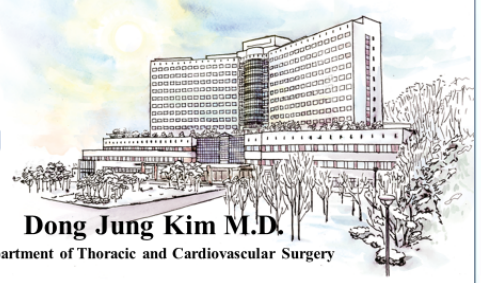
Hyperkalemia

Hyperkalemia : treatment

- Optimization of cardiac function
  - stabilization of cell membranes
  - calcium gluconate
    - advanced cardiac toxicity or ECG changes
    - usually when K^+ >6.5 mEq/L
    - 10mL of a 10% solution (1 g) IV over 2~3 minutes
    - should be avoided in patients on digoxin

Fluid, renal and metabolic problem management



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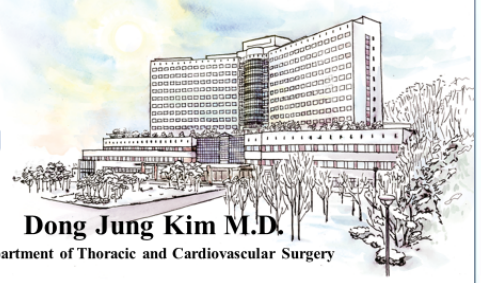
Hyperkalemia

Hyperkalemia : treatment

- Shift K^+ into cells
  - Regular insulin
    - 10 units in 50mL of 50% dextrose solution
    - lower K^+ 0.5~1.5 mEq/L within 15 min and last for several hours
    - 1st choice in case of marked hyperkalemia but no ECG changes
  - Sodium bicarbonate ($NaHCO_3$)
    - to raise pH to 7.40~7.50 in patients with metabolic acidosis
    - lower serum K^+ within 30 min and last for several hours
    - direct effect on hyperkalemia independent of change in pH
    - Na^+ load may reverse ECG changes of hyperkalemia
      - in hyponatremic patients

Fluid, renal and metabolic problem management



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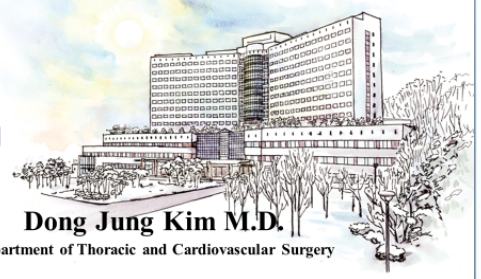
Hyperkalemia

Hyperkalemia : treatment

- Enhance K^+ excretion
  - Furosemide
    - 20–40 mg IV is effective in patients with well-functioning kidneys
    - higher doses may be required in patients with AKI or CKD
  - Sodium polystyrene sulfonate (Kayexalate) enema
    - 50 g in 150 mL of water can be given every 2~4 hours
    - each gram may bind up to 1mEq of K^+
    - reasonable 1st step in stable postop. patients
      - slowly rising K^+ >5.5 mEq/L despite withdrawal of contributing factors
  - Hemodialysis
    - indicated if above measures fail to lower K^+ to adequate levels
    - remove up to 50mEq of K^+ per hour

Fluid, renal and metabolic problem management



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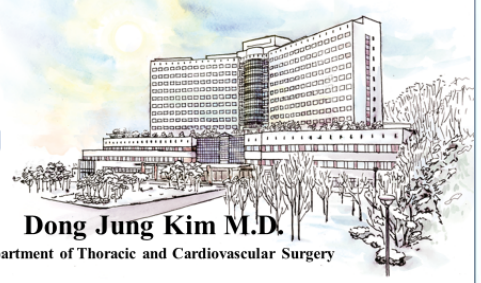
Metabolic acidosis

Metabolic acidosis : etiology

- Low cardiac output state
  - vasoconstriction from hypothermia or use of vasoconstrictive drugs
  - primary cause in cardiac surgery patient
- Mesenteric ischemia from a low-flow state
  - should be considered when progressive metabolic acidosis occurs
- Sepsis
- Renal failure
- Acute hepatic dysfunction
- Low-dose epinephrine
  - type B lactic acidosis (not associated with tissue hypoxia)

Fluid, renal and metabolic problem management



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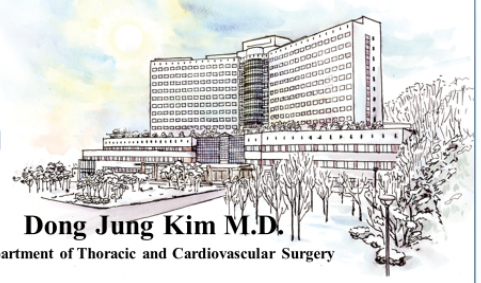
Metabolic acidosis

Metabolic acidosis : effect

- Compensatory hyperventilation
  - neutralization of acidosis in patients who can breathe spontaneously
    - when 1 mEq/L fall in bicarbonate, PCO_2 is reduced 1.2 torr
  - incomplete compensation
    - with mixed respiratory/metabolic acidosis

- Adverse effects of metabolic acidosis
  - usually do not occur until pH is less than 7.20
  - related to metabolic products associated with acidosis
    - rather than absolute level of pH
    - may be reversed by administration of sodium bicarbonate

Fluid, renal and metabolic problem management



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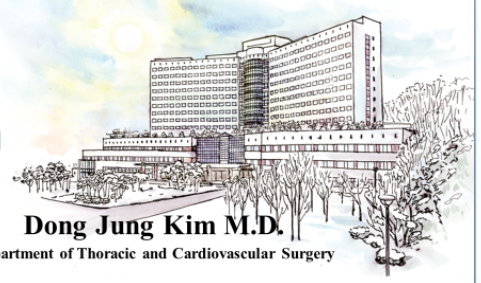
Metabolic acidosis

Metabolic acidosis : effect

- Cardiovascular effects
  - decreased contractility and cardiac output
    - reduction in hepatic and renal blood flow
  - ***attenuation of positive inotropic effects of catecholamines***
  - venoconstriction and arteriolar dilatation
    - increase filling pressures and decrease systemic pressures
  - increased pulmonary vascular resistance
  - arrhythmias
    - reduction in threshold for ventricular fibrillation
- Respiratory effects
  - dyspnea and tachypnea
  - decreased respiratory muscle strength

Fluid, renal and metabolic problem management



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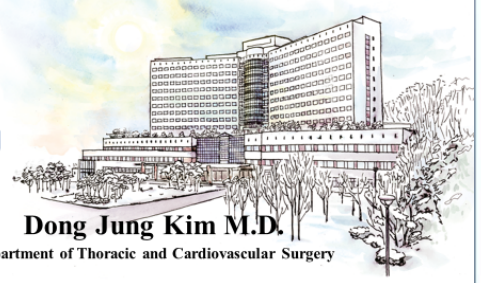
Metabolic acidosis

Metabolic acidosis : effect

- Metabolic changes
  - increased metabolic demands
  - hyperglycemia
    - caused by tissue insulin resistance and inhibition of anaerobic glycolysis
  - decreased hepatic update and increased hepatic production of lactate
  - hyperkalemia
  - increased protein catabolism

- Cerebral function
  - inhibition of brain metabolism and cell volume regulation
  - obtundation and coma

Fluid, renal and metabolic problem management



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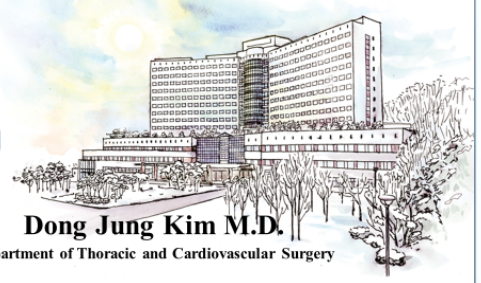
Metabolic acidosis

Type A lactic acidosis

- Impaired tissue oxygenation and anaerobic metabolism
  - resulting from circulatory failure
- Self-perpetuating
  - excessive production & suppression of hepatic utilization
- Lactate ion
  - probably more than acidosis
  - contributes to potential cardiovascular dysfunction

Fluid, renal and metabolic problem management



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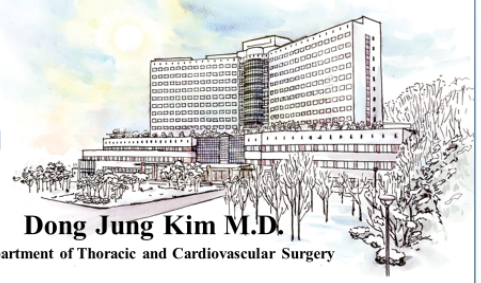
Metabolic acidosis

Type A lactic acidosis

- Elevated lactate levels (>3 mmol/L) upon arrival in ICU
  - associated with worse outcome
  - requires prompt attention
  - etiology
    - preexisting renal dysfunction
    - after long pump runs
    - use of intraoperative vasopressors
  - inadequate oxygen delivery during CPB
    - contribute to splanchnic and renal ischemia perpetuated by low cardiac output
- Development several days after surgery
  - raises specter of mesenteric ischemia
    - especially in patients requiring additional days of ICU care

Fluid, renal and metabolic problem management



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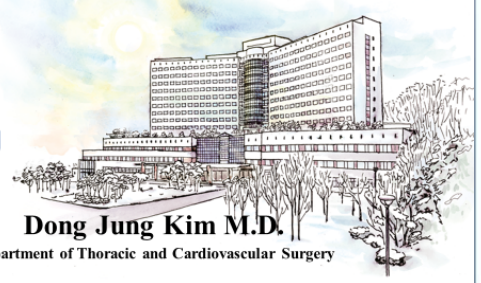
Metabolic acidosis

Type B lactic acidosis

- Occurs in the absence of tissue hypoxia
- Catecholamine-induced metabolic effect
  - especially with low dose epinephrine (<0.04μg/kg/min)
  - hyperglycemia and alterations in fatty acid metabolism
    - pyruvate accumulation and elevated levels of lactic acid
- Acute hepatic failure
  - present with severe lactic acidosis d/t failure to clear lactic acid
- Metformin
  - in patients with renal insufficiency, low cardiac output states, and liver disease, and with use of contrast agent

Fluid, renal and metabolic problem management



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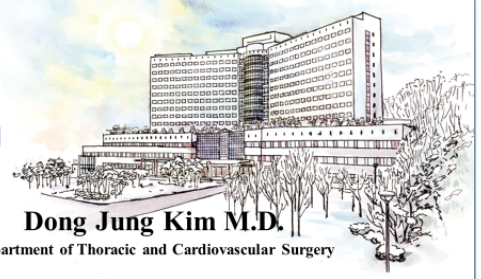
Metabolic acidosis

Metabolic acidosis : assessment

- Measurement of anion gap (AG)
  - $Na^+ – (Cl^– + HCO_3^–)$
  - normal range : 3~13 mEq/L
  - high AG metabolic acidosis
    - reflects additional acid production
    - most common after cardiac surgery
    - also be elevated in diabetic ketoacidosis
      - d/t production of hydroxybutyrate or in renal failure from retention of H^+
  - normal or low anion gap
    - represents loss of bicarbonate (diarrhea, renal tubular acidosis)

Fluid, renal and metabolic problem management



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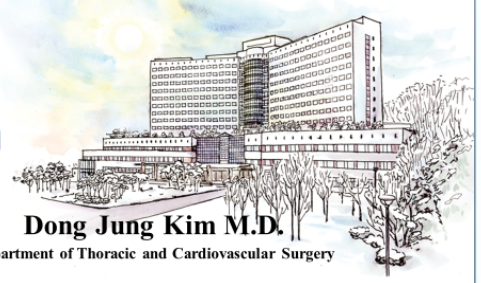
Metabolic acidosis

Metabolic acidosis : treatment

- Principle
  - reversal of underlying cause
  - oxidation of lactate and regeneration of HCO_3^-
- Proponents of $NaHCO_3$ administration
  - significant deleterious effects on cardiovascular function
    - can be corrected with more normal pH
  - ***more responsiveness to catecholamines***
    - occur with more normal pH
  - correction of acidosis may be important when etiology of acidosis is unclear or not imminently remediable

Fluid, renal and metabolic problem management



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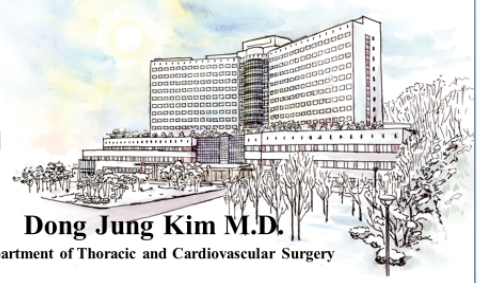
Metabolic acidosis

Metabolic acidosis : $NaHCO_3$

- Controversy
  - little evidence of hemodynamic improvement
  - metabolic derangements
    - fluid overload, hypernatremia, and hyperosmolarity
    - increased affinity of hemoglobin for oxygen
      - less tissue release
    - reduced ionized calcium
      - may reduce cardiac contractility
  - correct only the blood pH, not the intracellular pH
  - increased production of CO_2
    - may not be eliminated in low output states
    - may impair lactate utilization, perpetuating elevated lactate levels

Fluid, renal and metabolic problem management



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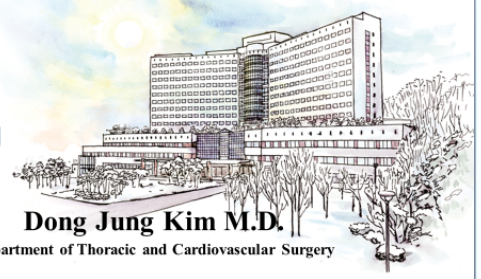
Metabolic acidosis

Metabolic acidosis : $NaHCO_3$

- Dose calculation
  - body weight in kg × 0.2 × base deficit = mEq $NaHCO_3$
- Severe metabolic acidosis
  - administered over several hours
  - with careful monitoring of serum Na^+ concentration
  - bicarbonate is metabolized to CO_2
    - worsen respiratory acidosis
      - in patients with compromised pulmonary function
    - hyperventilate patient to lower PCO_2
      - in mechanically ventilated patients

Fluid, renal and metabolic problem management



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Summary

Summary

- Fluid
  - type, intravascular volume, redistribution
- AKI
  - optimization of hemodynamics, CRRT
- Hyperkalemia
  - manifestations, treatment
- Metabolic acidosis
  - etiology, effect

Thank you for your attention !

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