POCKET NOTEBOOK

POCKET NEPHROLOGY

SECOND EDITION

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Wolters Kluwer

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Second Edition

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Philadelphia • Baltimore • New York • London
Buenos Aires • Hong Kong • Sydney • Tokyo

Acquisitions Editor: James Sherman
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Manufacturing Coordinator: Lisa Bowling
Prepress Vendor: Aptara, Inc.

9 8 7 6 5 4 3 2 1

Printed in Mexico

978-1-9752-1493-7

Library of Congress Cataloging-in-Publication Data available upon request.

CONTENTS

Josh Earl, Jacob Stevens
Acute Tubular Necrosis
Pigment Nephropathy
Crystal Nephropathy
Urinary Stone Disease
Interstitial Disease
Immunoglobulin G4-Related Disease

GLOMERULAR AND VASCULAR DISEASES
Syeda Behjat Ahmad, Pietro Canetta, Yonatan Peleg
Minimal Change Disease (Mcd)
Focal Segmental Glomerulosclerosis
Apolipoprotein L1 Nephropathy
Membranous Nephropathy
Pauci-Immune Glomerulonephritis
Anti-Gbm Disease
Immugnoglobulin A Nephropathy (IgAN)
Immune Complex-mediated MPGN
Cryoglobulinemia
C3 Glomerulopathy
Complement-Mediated Tma (Cm-Tma)
Hus and Ttp
Antiphospholipid Syndrome (Aps)
Amyloidosis
Nonamyloid Deposition Diseases
Renal Vascular Diseases

UROLOGY
Hilda Elena Fernandez
Urinary Tract Obstruction (UTO)
Reflux Nephropathy
Urinary Tract Infection (UTI)
Renal Cell Carcinoma (RCC)

HYPERTENSION
Andrew S. Bomback, Yelena Drexler
General Hypertension
Hypertension in Dialysis
Resistant Hypertension
Hypertensive Emergencies
Antihypertensives
Obstructive Sleep Apnea (Osa)
Renal Artery Stenosis (Ras)
Primary Aldosteronism (PA)
Pheochromocytoma
Hypertension after Kidney Transplantation

CARDIOLOGY–PULMONOLOGY
Wooin Ahn, Geoffrey K. Dube
Cardiology
Pulmonology
Mechanical Circulatory Support

GASTROENTEROLOGY–HEPATOLOGY
Anushya Jeyabalan, Heedeok Han, Shayan Shirazian, Meghan E. Sise
Gastroenterology
Hepatology
Hepatitis B Virus (Hbv)
Hepatitis C Virus (Hcv)

HEMATOLOGY–ONCOLOGY
Abdallah S. Geara
Hematology
Anticoagulation
Monoclonal Gammopathy (Mg)
Hematopoietic Cell Transplantation (Hct)
Oncology

Properties of Vasopressors and Inotropes				
Dose	**Mechanisms**	**Afterload (SVR)**	**Inotropy (CO)**	**Chronotropy (HR)**
Dopamine (mcg/kg/min)				
1–3	DA >> β1	↑ or ↓	+	+
3–10	β1 > β2, DA > α1	↑	++	++
>10	α1 > β1 >>> β2	↑↑	+++	++
Low dose does not prevent ATN; can lead to HoTN and tachycardia (*KI* 2006;69:1669)				
Epinephrine (mcg/min)				
1–20	α1, α2, β1 > β2	↑↑↑	++++	++++
Low dose may ↓ SVR; A first-line agent in anaphylactic shock				
Norepinephrine (mcg/min)				
1–40	α1, α2 >> β1	↑↑↑↑	++	+
Preferred as the first-line agent over other vasopressors in septic shock;Vasopressin then epinephrine can be added to norepinephrine to meet MAP target				
Phenylephrine (mcg/min)				
10–400	α1 >> α2	↑↑↑↑	-	-
May be considered when tachyarrhythmias preclude use of norepinephrine				
Vasopressin (U/min)				
0.04–0.06	V1/V2	↑↑	-	-
Vasopressin vs Norepinephrine: in septic shock no difference in kidney failure-free days or death, but vasopressin group had less use of KRT (VANISH *JAMA* 2016;316:509)				
Dobutamine (mcg/kg/min)				

- Vasopressors generally improve GFR (*J Physiol* 1981;321:21; *CJASN* 2008;3:546)
- Angiotensin II (Giapreza®): in vasodilatory shock, ↑ MAP (*NEJM* 2017;377:419); in AKI requiring KRT, ↓ 28-d mortality and ↑ KRT liberation (*Crit Care Med* 2018;46:949)
- Methylene blue: early initiation reduces time to vasopressor discontinuation and increases vasopressor-free days (*Crit Care* 2023;27:110)
- High-dose hydroxycobalamine: used in refractory shock; can cause false blood leak alarm in certain HD machine (*CKJ* 2017;10:357)
- Inotropes are added in pts with septic shock and cardiac dysfunction with persistent hypoperfusion despite adequate volume status and arterial blood pressure,

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Kidney Replacement Therapy in Shock

CKRT

- More likely to ↓ fluid accumulation than HD (*KI* 2009;76:422)
- Timing of KRT (early initiation vs standard of care): No differences in outcome: (*NEJM* 2016;375:122; STARRT-AKI *NEJM* 2020;383:240; IDEAL-ICU *NEJM* 2018;379:1431)
- Early KRT initiation did not ↓ mortality, no differences in KRT dependence or serum Cr on discharge or in ventilator or vasopressor use (*Lancet* 2020;395:1506)

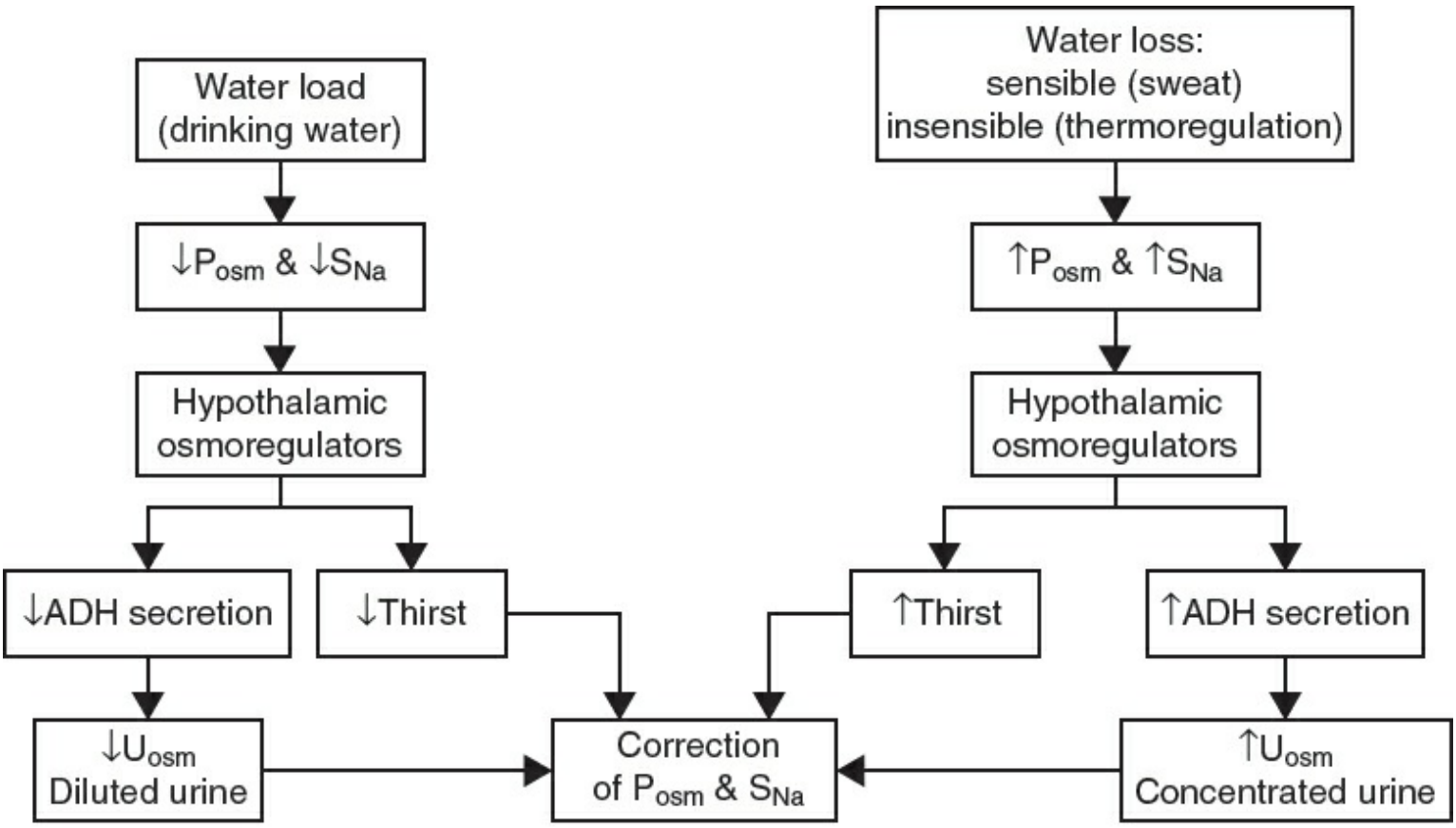
Orthostatic Hypotension (OH)

- Physiologic response to standing: the pooling of 500–1,000 mL of blood in the lower extremities and splanchnic circulation → ↓ venous return to the heart and ↓ cardiac output and BP → ↑ sympathetic outflow (baroreceptor reflex) → ↑ peripheral vascular resistance, venous return, cardiac output, and BP
- OH: postural reduction in SBP ≥20 or DBP ≥10 w/i 3 min of standing
- BP fall w/i 1 min was a/w dizziness, fracture, syncope, and death (*JAMA IM* 2017;177:1316)
- Delayed OH: OH after 3 min of standing; a/w Parkinson ds (*Neurology* 2015;85:1362)
- Postural tachycardia synd (POTS): ↑ HR ≥30 beats/min w/i 10 min of standing or head-up tilt in the absence of OH

Causes

- Volume depletion: fluid loss, overdiuresis, overdialysis; adrenal insufficiency, anemia
- Autonomic dysfunction: amyloidosis, DM, Parkinson ds, multiple system atrophy

- Urine sodium (U_{Na}): often correlates w/ volume status (hypovolemia ↓ U_{Na})
- Dehydration (water loss) ↑ [Na] ≠ volume depletion (sodium and water loss)
- P_{osm} (and P_{Na}) is regulated by ADH secretion and thirst



Feedback mechanism to maintain P_{osm} and [Na].

- Free water clearance is determined by ADH activity and solute excretion (*JASN* 2008;19:1076).

$$\text{Free water clearance} = \frac{\text{Solute Excretion}}{U_{osm}}\left(1 - \frac{U_{osm}}{P_{osm}}\right)$$

$$\text{Electrolyte free water clearance} = \frac{\text{Solute Excretion}}{U_{osm}}\left(1 - \frac{U_{Na} + U_K}{P_{Na}}\right)$$

Hyponatremia

Background

- Definition: serum sodium concentration <135
- Usually due to ↓ water excretion and rarely solely from ↑ free water intake

Clinical Manifestations

- Water movement into the brain acutely leads to brain edema
- Cerebral edema is related to the degree and the rapidity of hyponatremia
- Symptoms in acute hyponatremia can be nonspecific—malaise, nausea, progressing to headache, lethargy, gait imbalance, and in extreme cases seizures and coma
- Chronic hyponatremia <130 is associated with subtle neurologic symptoms such as general malaise, decreased attention span, and gait instability/falls
- Severe acute hyponatremia can lead to brain herniation in premenopausal women and young children

Acute Hyponatremia

- Caused by high free water intake with additional pathogenesis: postop, exercise, ecstasy, haloperidol, thiazide, desmopressin, oxytocin, nonconductive irrigation solutions, IV CYC

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