



POCKET

NEPHROLOGY

SECOND EDITION

Wooin Ahn

Jai Radhakrishnan

 Wolters Kluwer



Pocket **NEPHROLOGY**

Second Edition

Edited by

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

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Prepress Vendor: Aptara, Inc.

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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.

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Properties of Vasopressors and Inotropes				
Dose	Mechanisms	Afterload (SVR)	Inotropy (CO)	Chronotropy (HR)
Dopamine (mcg/kg/min)				
1–3	DA >> β_1	↑ or ↓	+	+
3–10	$\beta_1 > \beta_2$, DA > α_1	↑	++	++
>10	$\alpha_1 > \beta_1 >> \beta_2$	↑↑	+++	++
Low dose does not prevent ATN; can lead to HoTN and tachycardia (KI 2006;69:1669)				
Epinephrine (mcg/min)				
1–20	$\alpha_1, \alpha_2, \beta_1 > \beta_2$	↑↑↑	++++	++++
Low dose may ↓ SVR; A first-line agent in anaphylactic shock				
Norepinephrine (mcg/min)				
1–40	$\alpha_1, \alpha_2 >> \beta_1$	↑↑↑↑	++	+
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	$\alpha_1 >> \alpha_2$	↑↑↑↑	-	-
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 hydroxycobalamin: 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,

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)

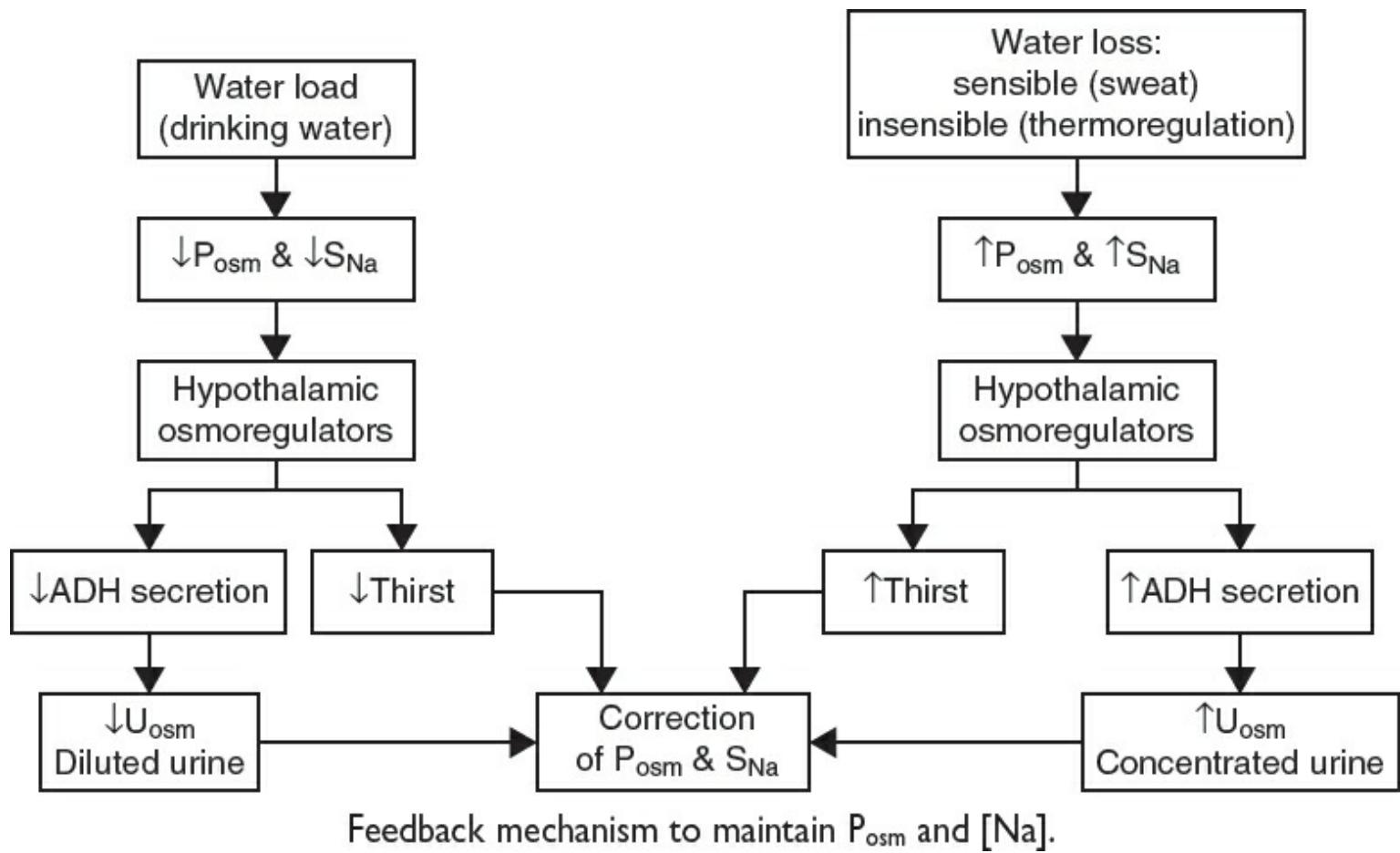
ORTHOSTATIC HYPOTENSION (OH)

- 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 $\downarrow U_{Na}$)
- Dehydration (water loss) $\uparrow [Na] \neq$ volume depletion (sodium and water loss)
- P_{osm} (and P_{Na}) is regulated by ADH secretion and thirst



- 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 \downarrow water excretion and rarely solely from \uparrow 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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