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Woojin Ahn

Jai Radhakrishnan

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Edited by

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Properties of Vasopressors and Inotropes				
Dose	Mechanisms	Afterload (SVR)	Inotropy (CO)	Chronotropy (HR)
Dopamine (mcg/kg/min)				
1–3	DA >> $\beta 1$	\uparrow or \downarrow	+	+
3–10	$\beta 1 > \beta 2$, DA > $\alpha 1$	\uparrow	++	++
>10	$\alpha 1 > \beta 1 >>> \beta 2$	$\uparrow\uparrow$	+++	++
Low dose does not prevent ATN; can lead to HoTN and tachycardia (KI 2006;69:1669)				
Epinephrine (mcg/min)				
1–20	$\alpha 1, \alpha 2, \beta 1 > \beta 2$	$\uparrow\uparrow\uparrow$	++++	++++
Low dose may \downarrow SVR; A first-line agent in anaphylactic shock				
Norepinephrine (mcg/min)				
1–40	$\alpha 1, \alpha 2 >> \beta 1$	$\uparrow\uparrow\uparrow\uparrow$	++	+
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	$\alpha 1 >> \alpha 2$	$\uparrow\uparrow\uparrow\uparrow$	-	-
May be considered when tachyarrhythmias preclude use of norepinephrine				
Vasopressin (U/min)				
0.04–0.06	V1/V2	$\uparrow\uparrow$	-	-
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, \uparrow MAP (*NEJM* 2017;377:419); in AKI requiring KRT, \downarrow 28-d mortality and \uparrow 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 \downarrow 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 \downarrow mortality, no differences in KRT dependence or serum Cr on discharge or in ventilator or vasopressor use (*Lancet* 2020;395:1506)

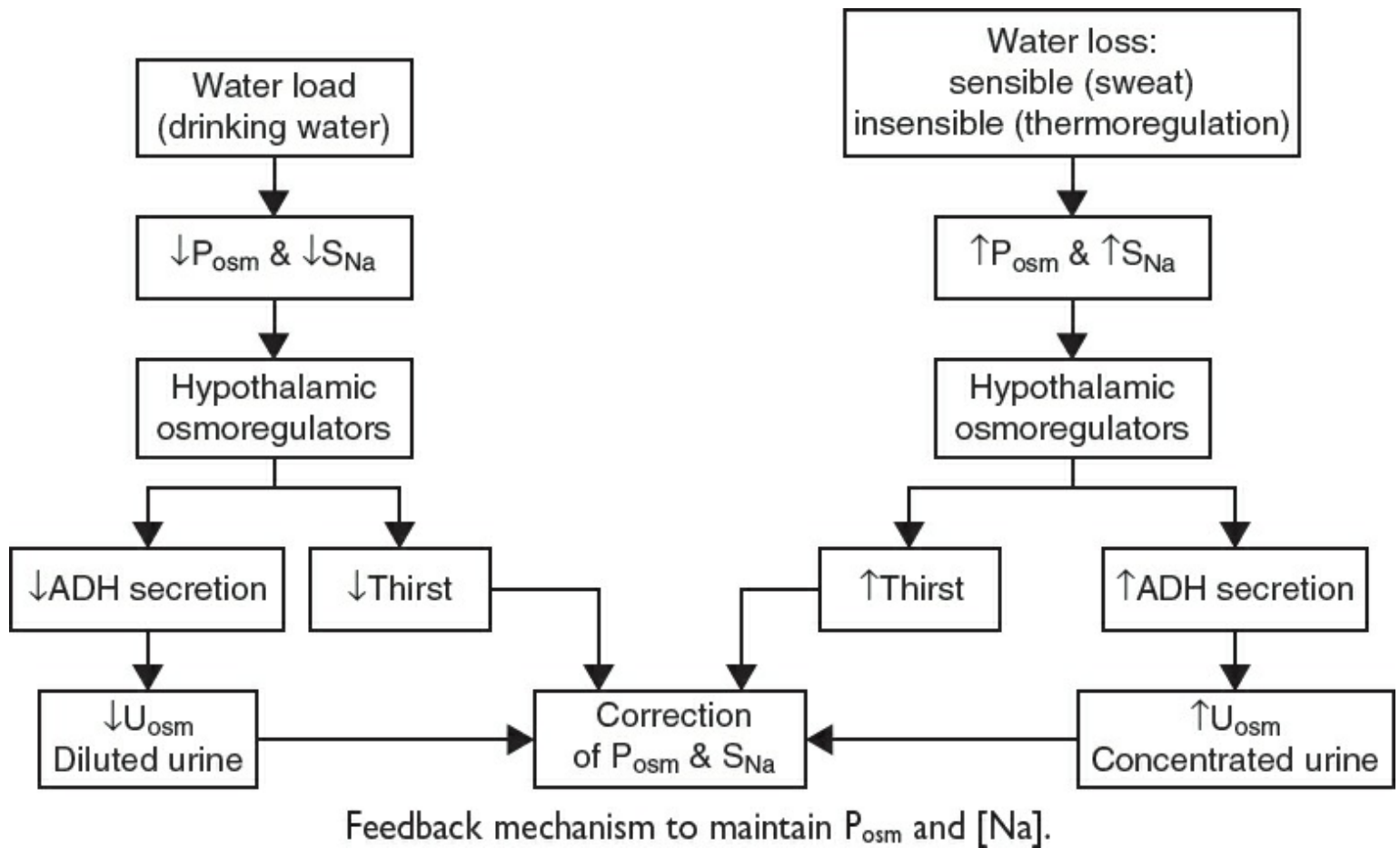
ORTHOSTATIC HYPOTENSION (OH)

- Physiologic response to standing: the pooling of 500–1,000 mL of blood in the lower extremities and splanchnic circulation \rightarrow \downarrow venous return to the heart and \downarrow cardiac output and BP \rightarrow \uparrow sympathetic outflow (baroreceptor reflex) \rightarrow \uparrow 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): \uparrow 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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