

POLYURIA AND POLYDIPSIA



Our mission is to redefine and elevate your pet's health through collaborative, minimally-invasive, and compassionate care that supports the well-being of your pet and peace of mind for your family.

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Pacific Coast Veterinary Specialists

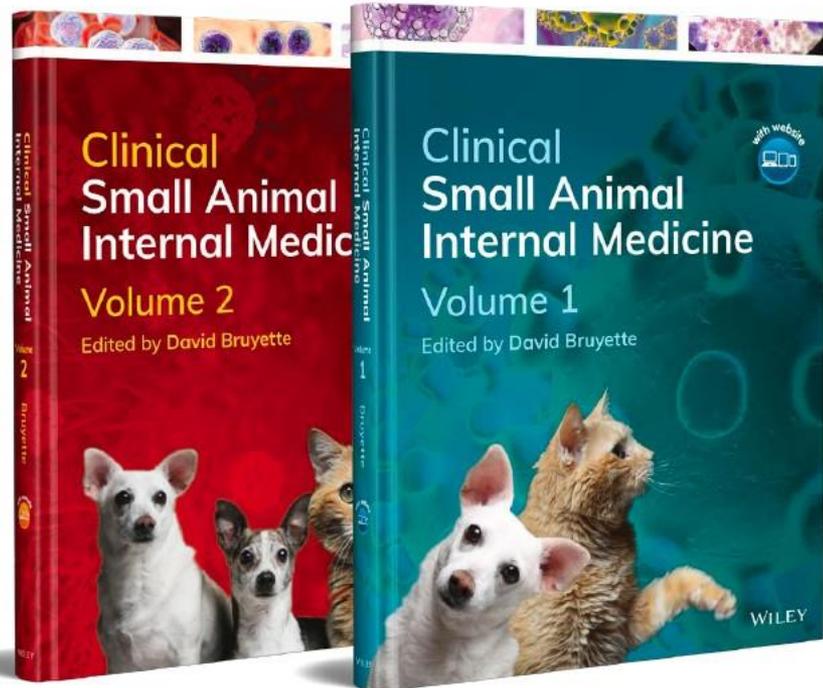
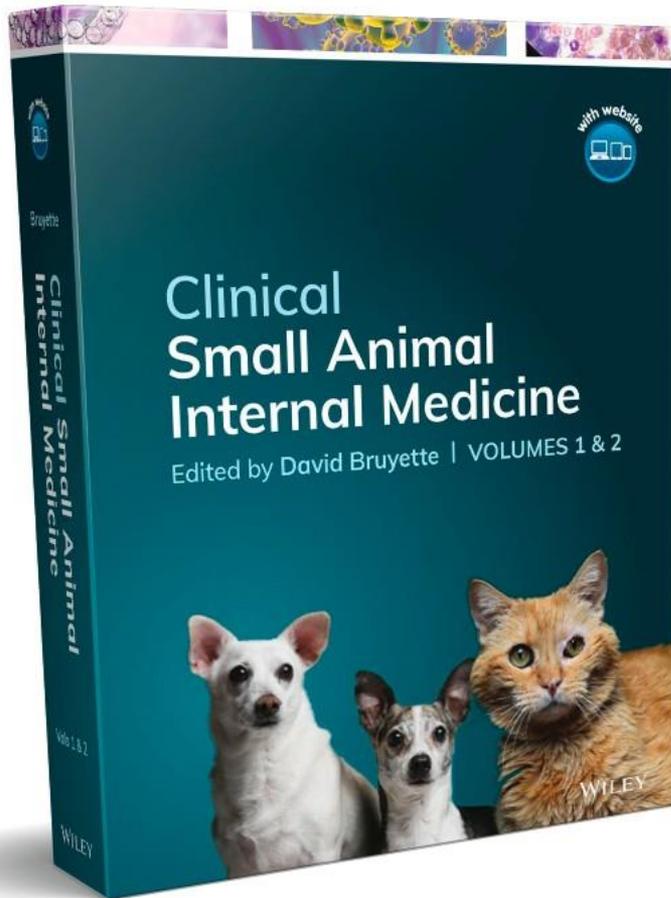
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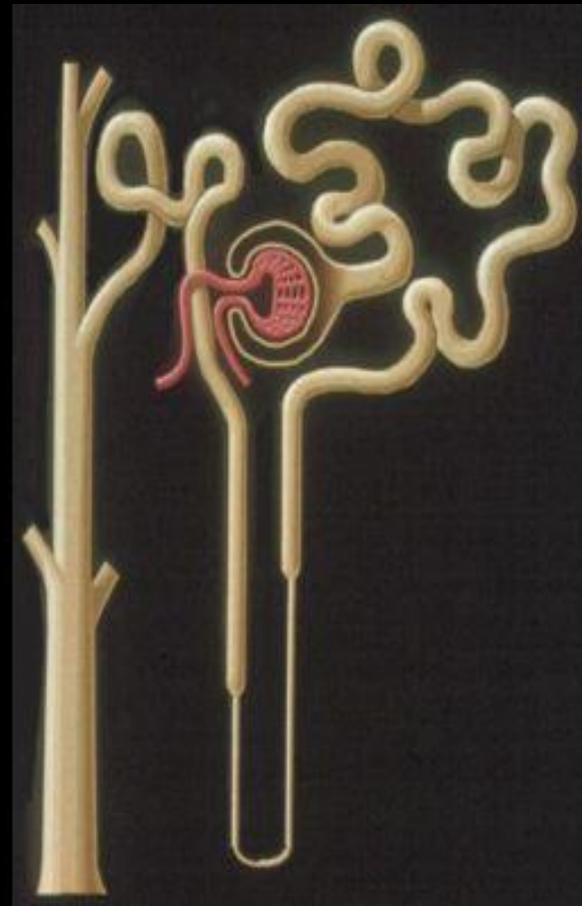
NORMAL WATER HOMEOSTASIS

Fluid Intake and Urine Output

Intact Pituitary – Renal Axis

Antidiuretic Hormone (ADH)

Intact Renal Medullary
Interstitialium



NORMAL WATER HOMEOSTASIS

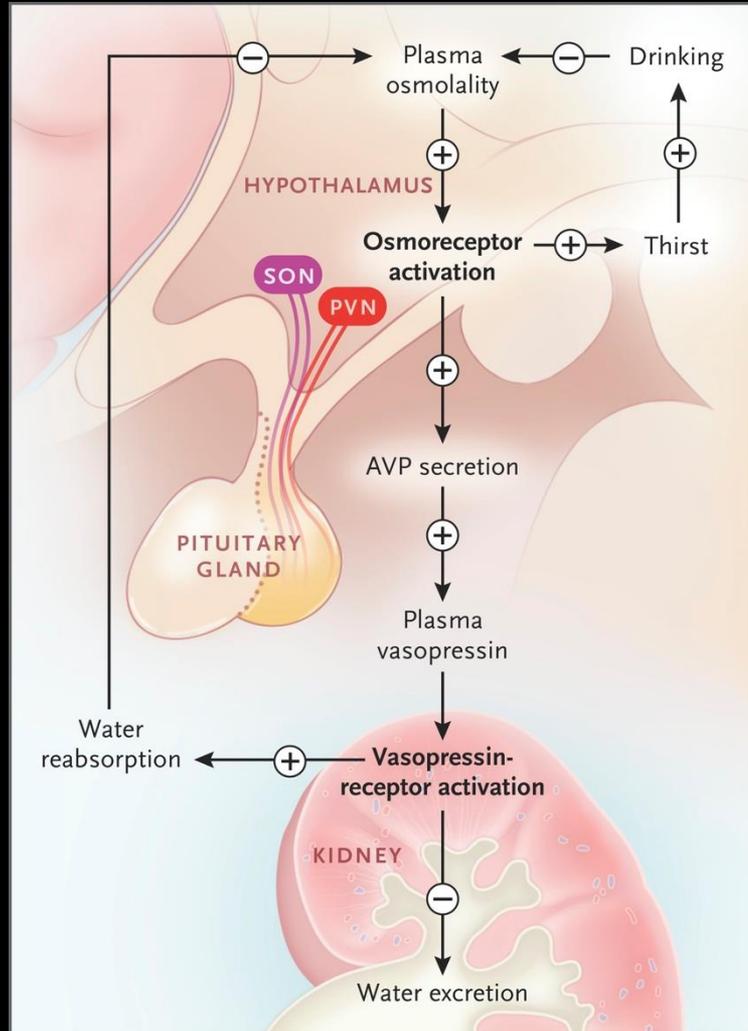
Fluid Intake and Urine Output

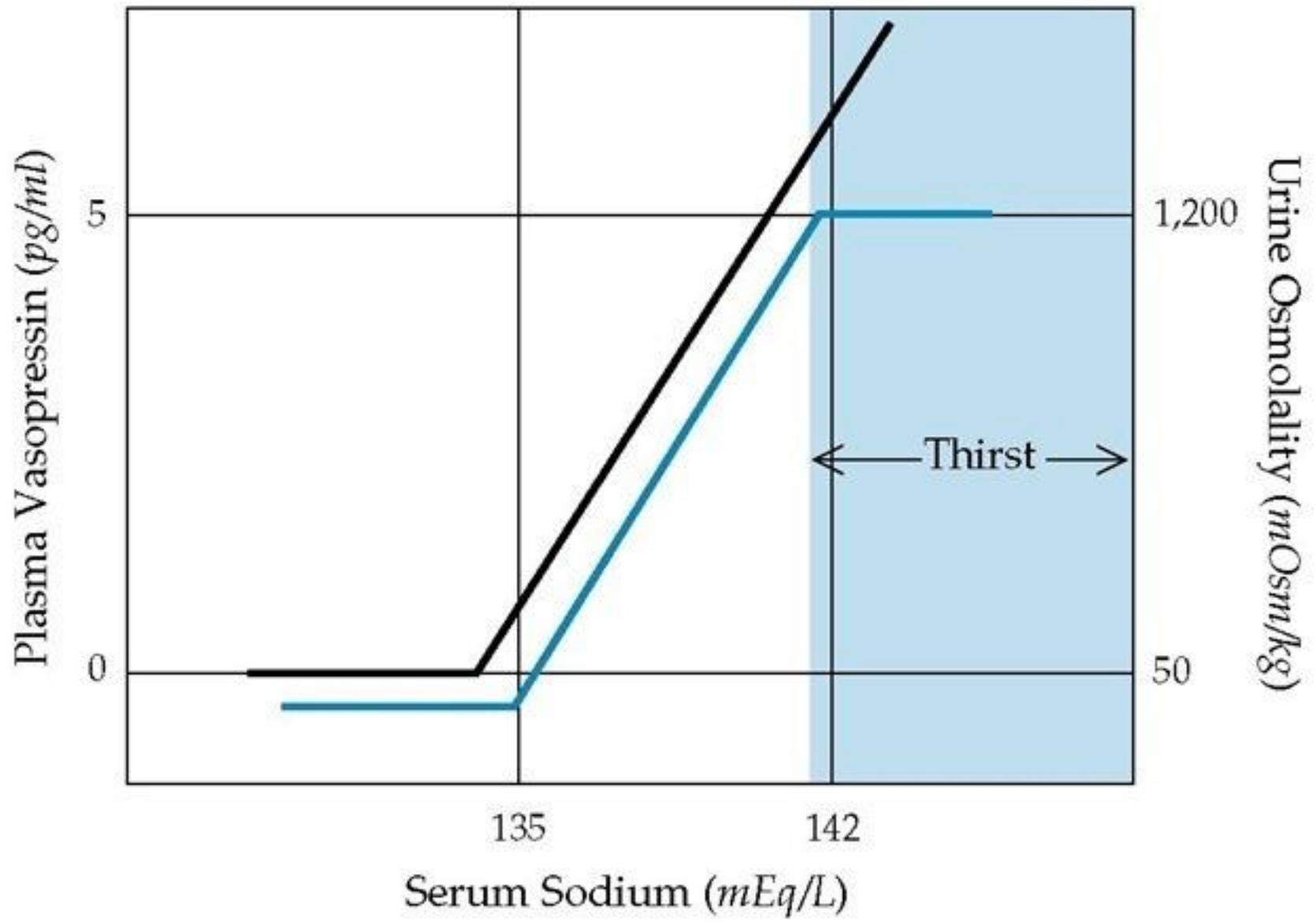
Thirst

Plasma Osmolality

Serum Na concentration

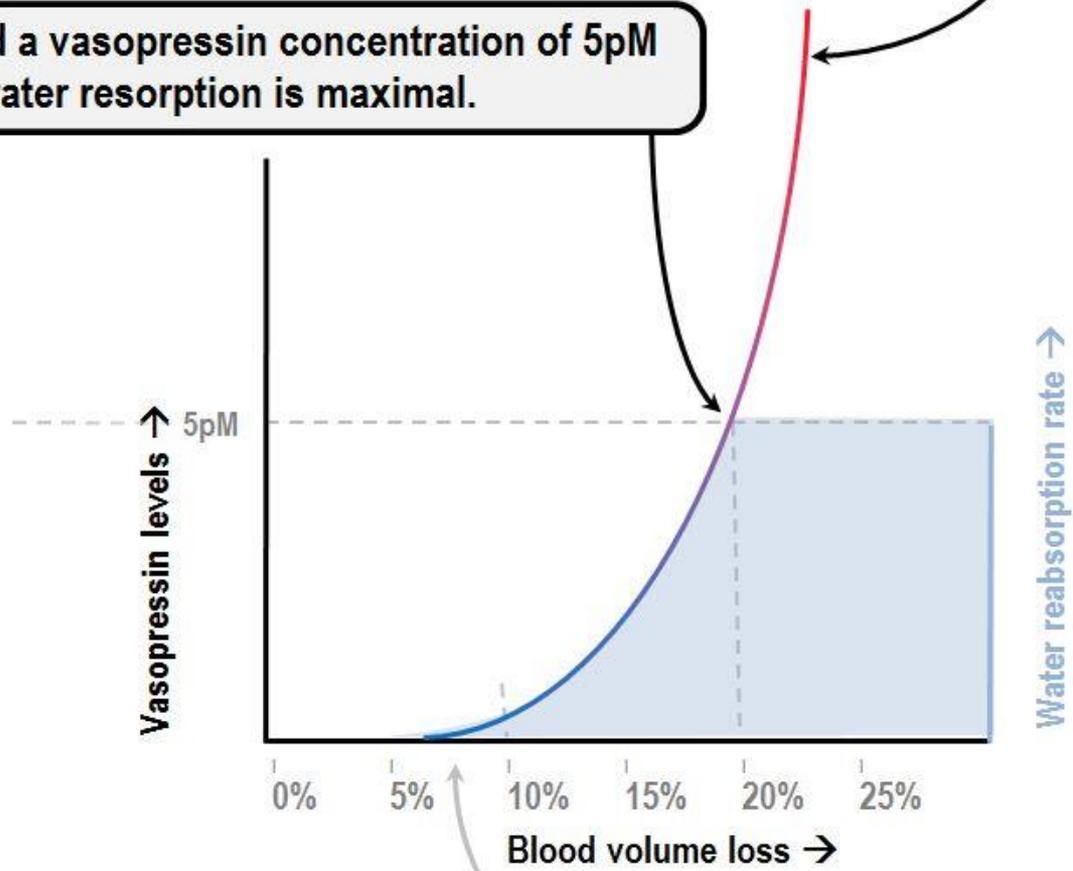
Hypovolemia



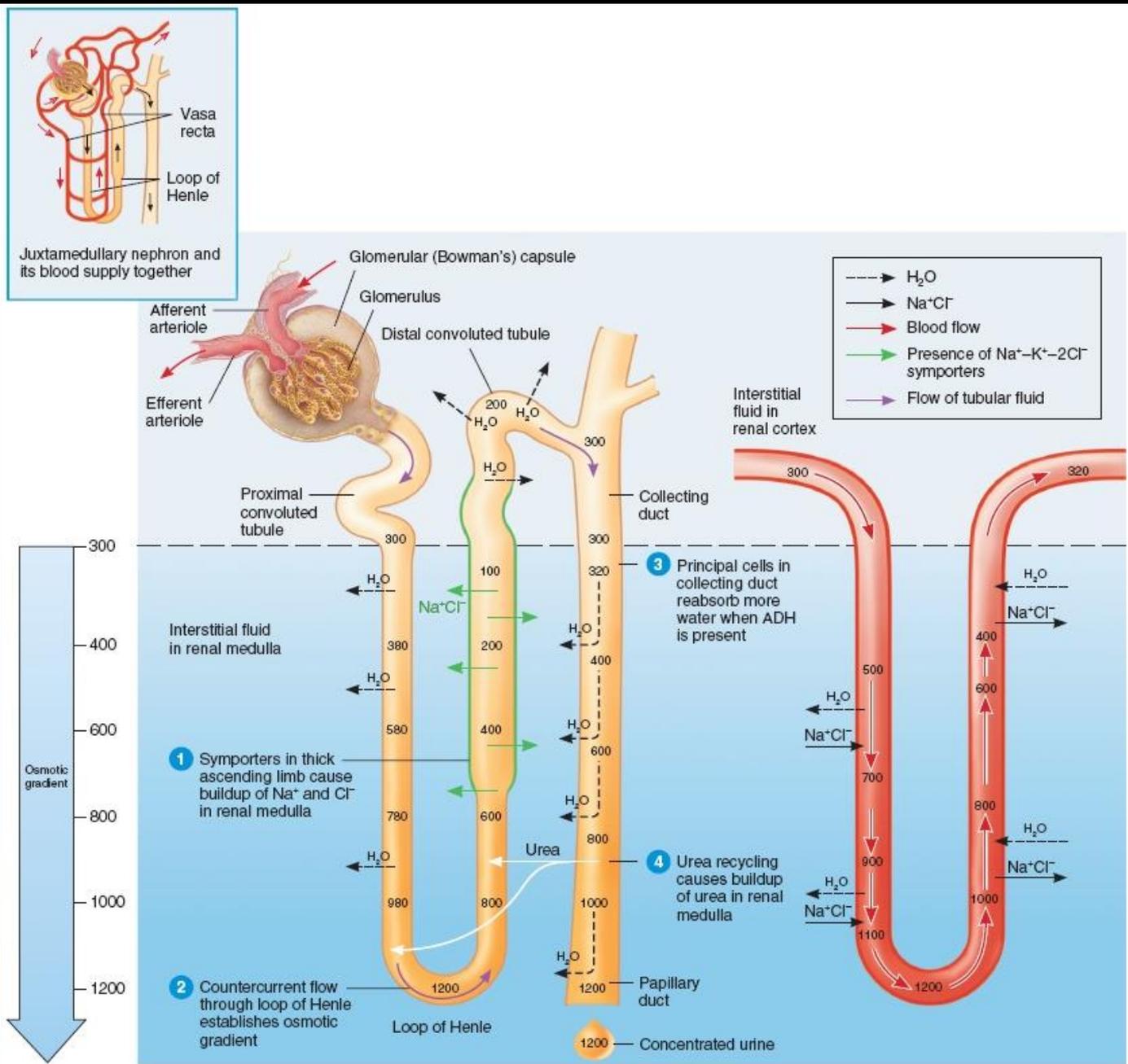


Beyond a vasopressin concentration of 5pM renal water resorption is maximal.

However, vasopressin levels continue to rise. At these high concentrations, it begins to act as a vasoconstrictor

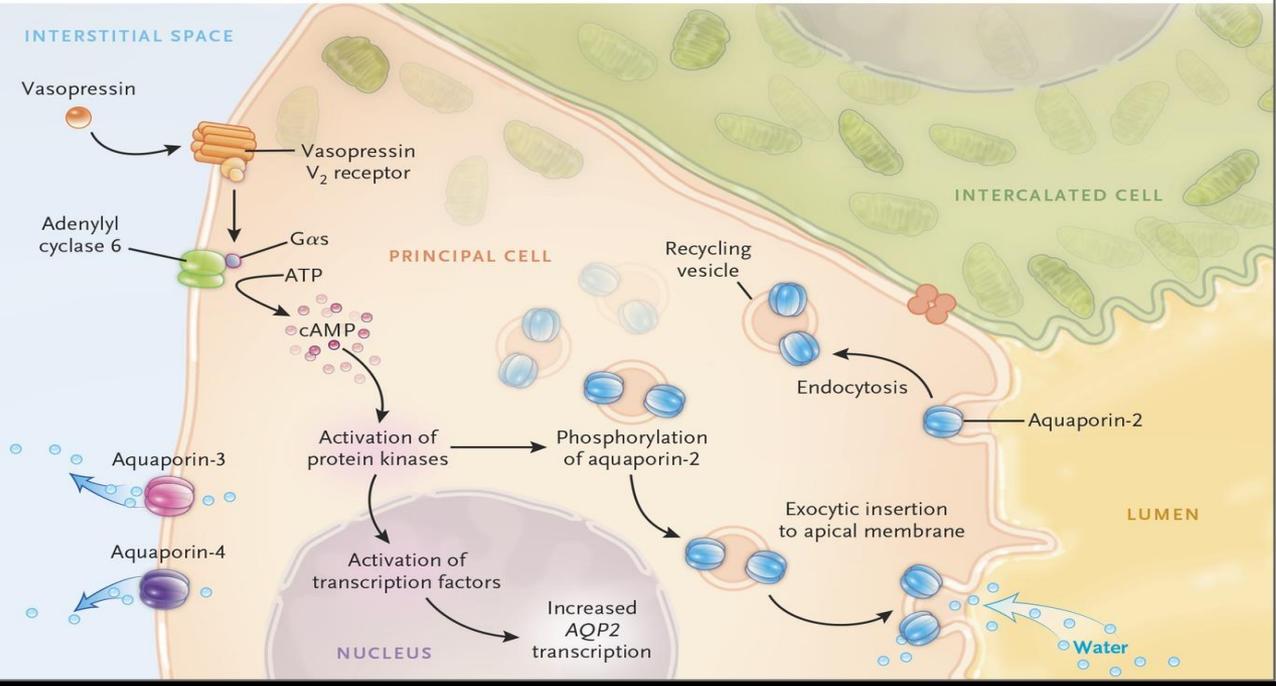
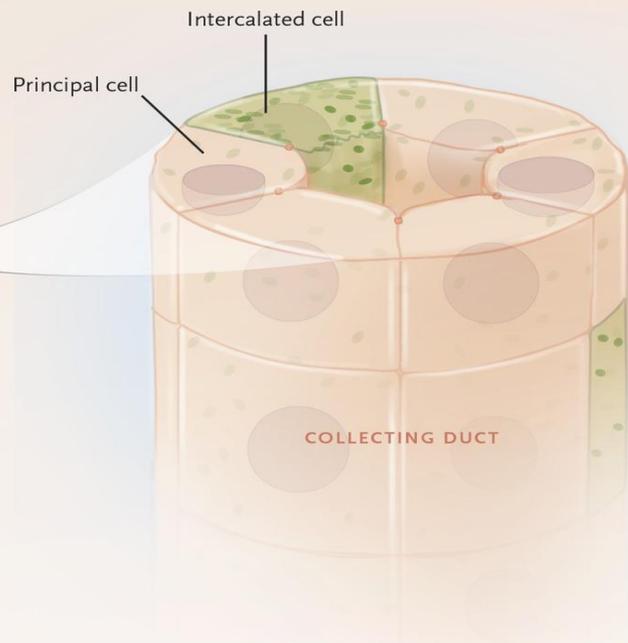
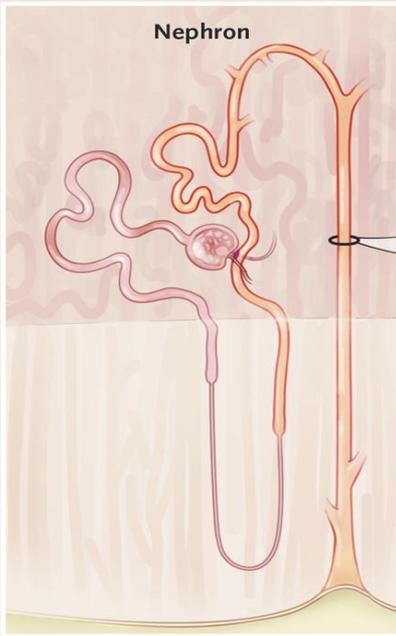


Small isotonic changes in blood volume have no effect on vasopressin release

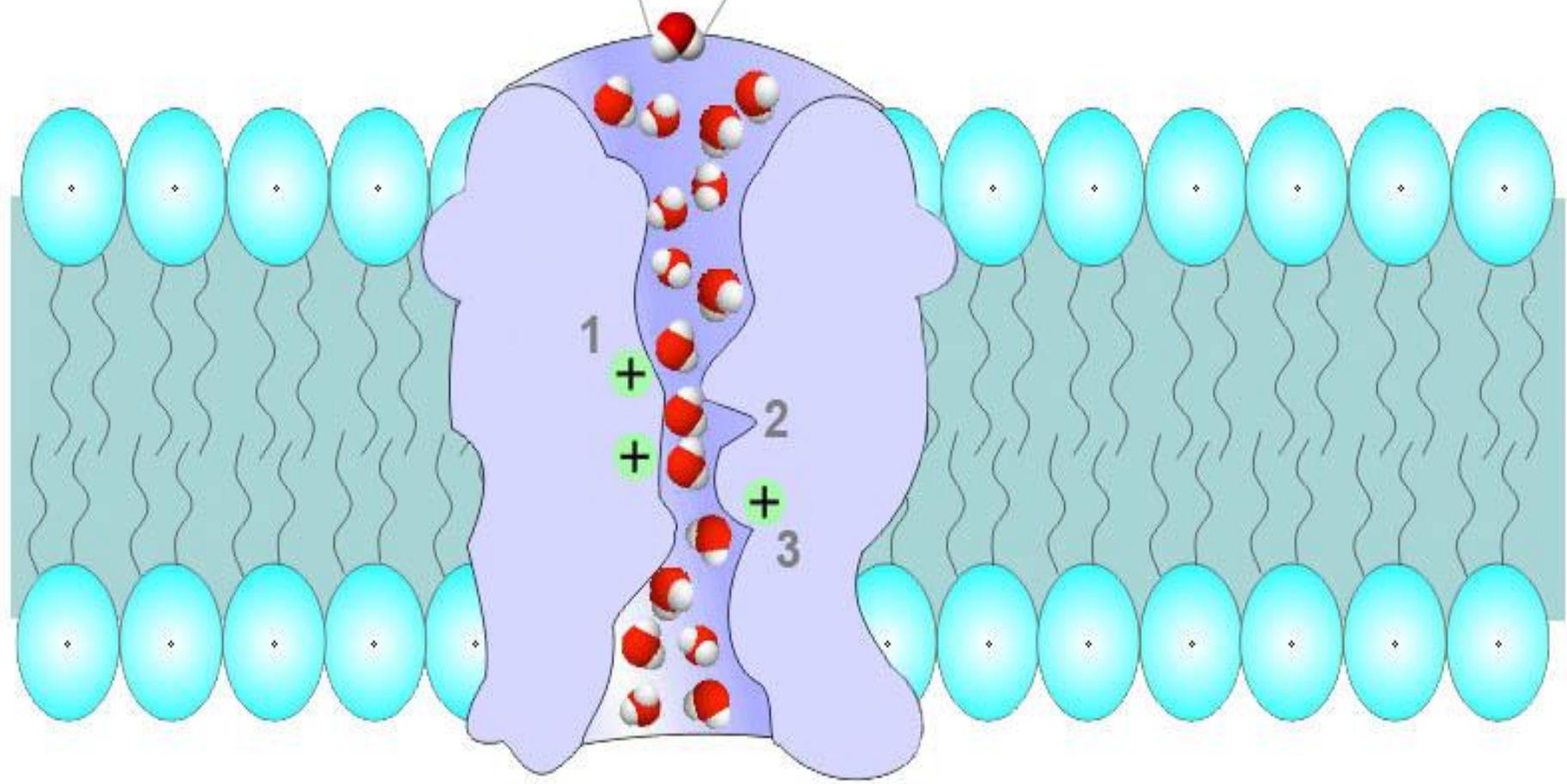


(a) Reabsorption of Na^+ , Cl^- , and water in long-loop juxtamedullary nephron

(b) Recycling of salts and urea in vasa recta



δ^- (Partial negative charge)



Key Proteins Involved in Regulation of Water Balance.

Table 1. Key Proteins Involved in Regulation of Water Balance.

Protein	Gene	Structure or Cell Type Relevant to Water Balance	Manifestation of Loss of Function*	Drugs That Target Protein
Arginine vasopressin	<i>AVP</i>	Neurons of supraoptic nucleus and paraventricular nucleus	Central diabetes insipidus	None
Vasopressin receptor				
V ₂	<i>AVPR2</i>	Renal thick ascending limb of the loop of Henle, distal convoluted tubule, connecting tubule, collecting duct	X-linked nephrogenic diabetes insipidus	Desmopressin acetate (agonist), tolvaptan (antagonist)
V _{1a}	<i>AVPR1A</i>	Renal medullary vasculature (vasa recta)	None	Conivaptan (nonselective V _{1a} and V ₂ antagonist)
Bumetanide-sensitive sodium-potassium-chloride cotransporter	<i>SLC12A1</i>	Renal thick ascending limb of the loop of Henle	Type I Bartter's syndrome	Loop diuretics
Thiazide-sensitive sodium-chloride cotransporter	<i>SLC12A3</i>	Renal distal convoluted tubule	Gitelman's syndrome	Thiazide diuretics
Aquaporin				
Aquaporin-1	<i>AQP1</i>	Renal proximal tubule, thin descending limb of the loop of Henle, erythrocyte	Colton blood group-null	None
Aquaporin-2	<i>AQP2</i>	Renal connecting tubule, collecting duct	Autosomal nephrogenic diabetes insipidus	None
Aquaporin-3	<i>AQP3</i>	Renal connecting tubule, collecting duct, erythrocyte	GIL blood group-null	None
Aquaporin-4	<i>AQP4</i>	Renal connecting tubule, collecting duct	None	None
Vasopressin-regulated urea channel	<i>SLC14A2</i>	Renal inner medullary collecting duct, thin descending limb of the loop of Henle	None	None
Epithelial sodium channel				
Beta subunit	<i>SCNN1B</i>	Renal connecting tubule, collecting duct	Type I pseudohypoaldosteronism	Amiloride
Gamma subunit	<i>SCNN1G</i>	Renal connecting tubule, collecting duct	Type I pseudohypoaldosteronism	Amiloride

* Data are from the Online Mendelian Inheritance in Man database.

POLYURIA AND POLYDIPSIA

Physiologic vs pathologic

Ambient temperature/humidity

Activity level

Dietary factors

Medications

POLYURIA AND POLYDIPSIA

Polyuria vs Pollakiuria

Lower urinary tract disease

Diagnostic approach

UA's, urine culture and sensitivity

Imaging : radiographs, US, contrast
studies

POLYURIA AND POLYDIPSIA

Owner Assessment

Subjective

Water consumption at home

Hospitalization

Measurement of water consumption and
urine output

POLYURIA AND POLYDIPSIA

Normal Water Consumption

40 – 60 mls/kg day

Laboratory animals

Abnormal Water Consumption

> 100 mls/kg/day

POLYURIA AND POLYDIPSIA

Diagnostic Approach

Documentation of pu/pd

Physical examination

Urinalysis

Sp gravity

Sediment and dip stick exam

Culture and sensitivity

CBC, serum chemistries

DIFFERENTIAL DIAGNOSIS

Renal Disease

Diabetes Mellitus

Liver Disease

Hyperthyroidism

Hyperadrenocorticism

Diabetes Insipidus

CDI

NDI

Hypercalcemia

Hypoadrenocorticism

Pyometra

Hypokalemia

Polycythemia

Medications

Psychogenic

DIFFERENTIAL DIAGNOSIS

Renal Disease

Pyelonephritis	>	Endotoxins
CRF	>	Loss of nephrons
Fanconi's	>	Glycosuria
Post-obstructive diuresis	>	Osmotic diuresis

DIFFERENTIAL DIAGNOSIS

Diabetes Mellitus

Hyperglycemia

Glycosuria and/or ketonuria

Osmotic diuresis

DIFFERENTIAL DIAGNOSIS

Liver Disease

Medullary washout

Decreased urea production

Low BUN

Altered release of ADH

Altered GABA and dopaminergic tone

Increased secretion of cortisol

DIFFERENTIAL DIAGNOSIS

Hyperthyroidism

Increased GFR

Increased renal blood flow

Psychogenic

DIFFERENTIAL DIAGNOSIS

Hypercalcemia

Renal tubular mineralization

Alteration in renal blood flow

Renal artery vasoconstriction

Altered release of ADH

Tubular resistance to ADH

DIFFERENTIAL DIAGNOSIS

Hyperadrenocorticism

Altered release of ADH

Tubular resistance to ADH

Concurrent UTI's

Pyelonephritis

85 % of patients will be pu/pd

DIFFERENTIAL DIAGNOSIS

Hypoadrenocorticism

Mechanism unknown

Renal Na loss

Osmotic diuresis

20-25 % of patients will be pu/pd

DIFFERENTIAL DIAGNOSIS

Pyometra

Concurrent pyelonephritis

E. coli endotoxins

Tubular resistance to ADH

DIFFERENTIAL DIAGNOSIS

Hypokalemia

Tubular vacuolization

Tubular resistance to ADH

Usually in conjunction with other causes of
pu/pd

DIFFERENTIAL DIAGNOSIS

Polycythemia (PCV > 65 %)

Altered CNS blood flow

Altered release of ADH

Altered renal blood flow

Tubular resistance to ADH

DIFFERENTIAL DIAGNOSIS

Medications

Steroids

Anticonvulsants

Diuretics

Salts

Na or K bromide

DIFFERENTIAL DIAGNOSIS

Central Diabetes Insipidus

Failure to manufacture,
store or secrete ADH

Congenital

Acquired



DIFFERENTIAL DIAGNOSIS

Nephrogenic Diabetes Insipidus

Congenital lack of tubular receptors
for ADH

Extremely rare in veterinary medicine

DIAGNOSTIC APPROACH

Completion of Initial Data Base

Normal physical examination

Normal laboratory work

Differential Diagnosis

Hyperadrenocorticism, diabetes insipidus, portosystemic shunt (young animals), psychogenic

ADH RESPONSE TESTING

Other differentials have been eliminated

No signs of azotemia/dehydration

Close owner monitoring

Gradual water restriction

ADH administration

ADH RESPONSE TESTING

Gradual water restriction at home for

2 - 3 days

Re-establishment of medullary gradient

Failure to perform gradual water restriction will result in poor response to ADH

ADH RESPONSE TESTING

DDAVP

1-2 drops into the conjunctival sac
once to twice a day

2 to 5 mcg SQ q12h–q24h

Oral 0.1 to 0.2 mg PO once to twice a day

ADH RESPONSE TESTING

Interpretation

Central Diabetes Insipidus

No response to water restriction

Marked increase in urine sp gravity
with DDAVP

ADH RESPONSE TESTING

Interpretation

Nephrogenic Diabetes Insipidus

No response to water restriction

No increase in urine sp gravity

with DDAVP

TREATMENT OF CENTRAL DIABETES INSIPIDUS

Constant access to free water

DDAVP

1-2 drops into the conjunctival sac

once to twice a day

2 to 5 mcg SQ q12h–q24h

Oral 0.1 to 0.2 mg once to twice a day

Expense

TREATMENT OF NEPHROGENIC DIABETES INSIPIDUS

Constant access to free water

Salt restriction

Thiazide diuretics

Hydrochlorothiazide 2 - 4 mg/kg BID

Loop diuretics such as furosemide will not be
effective

PROGNOSIS FOR CENTRAL DIABETES INSIPIDUS

Congenital form

Other pituitary abnormalities

TSH, ACTH, GH, FSH, LH

Adult-onset form

High incidence of pituitary/hypothalamic
neoplasia

PROGNOSIS FOR NEPHROGENIC DIABETES INSIPIDUS

Monitor renal function

May progress to chronic renal failure

Only mild to moderate reductions in pu/pd
with medical management