

Water Intoxication:

Psychiatric Management of Polydipsia

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Learning Objectives

- **LO1:** Review the epidemiology and pathophysiology of primary polydipsia
- **LO2:** Differentiate clinical manifestations of psychogenic polydipsia from other causes with similar symptoms
- **LO3:** Identify various treatment modalities for psychogenic polydipsia



Outline

- Epidemiology
- Symptoms
- Pathogenesis
- Dx / DDx
- Tx
- Conclusions



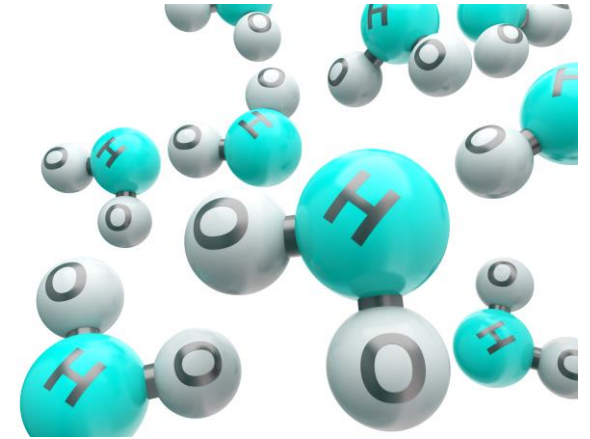
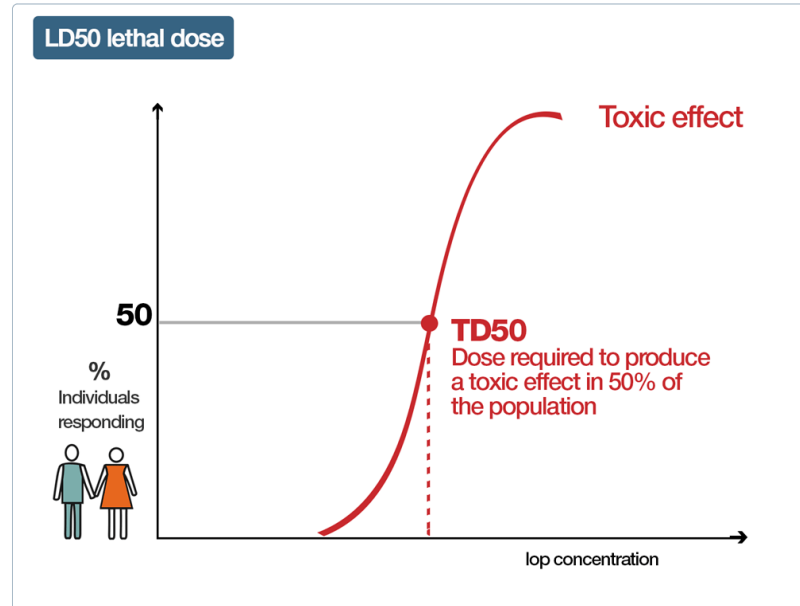
Water, water, everywhere, but not a drop to drink



Dose determines toxicity



Paracelsus (b 1493 Switzerland; d 1541 Austria)





In the news...







Primary polydipsia

- Psychogenic polydipsia
- Increase in thirst / water-seeking behaviors
- Polyuria: $>3\text{L} / \text{day}$
- National Academies of Sciences, Engineering, and Medicine
 - Men: 3.5L (15.5 cups) $\text{H}_2\text{O} / \text{day}$
 - Women: 2.5L (11.5 cups) $\text{H}_2\text{O} / \text{day}$
 - Modifying factors





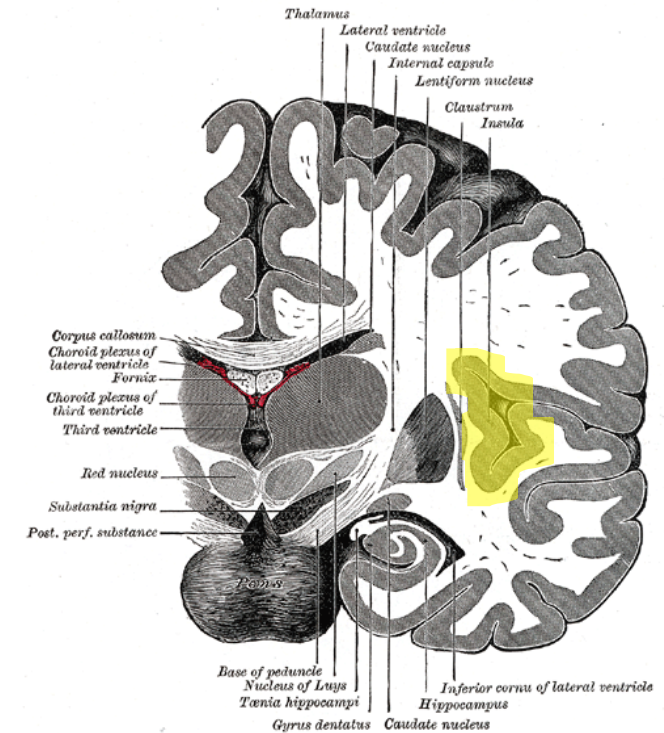
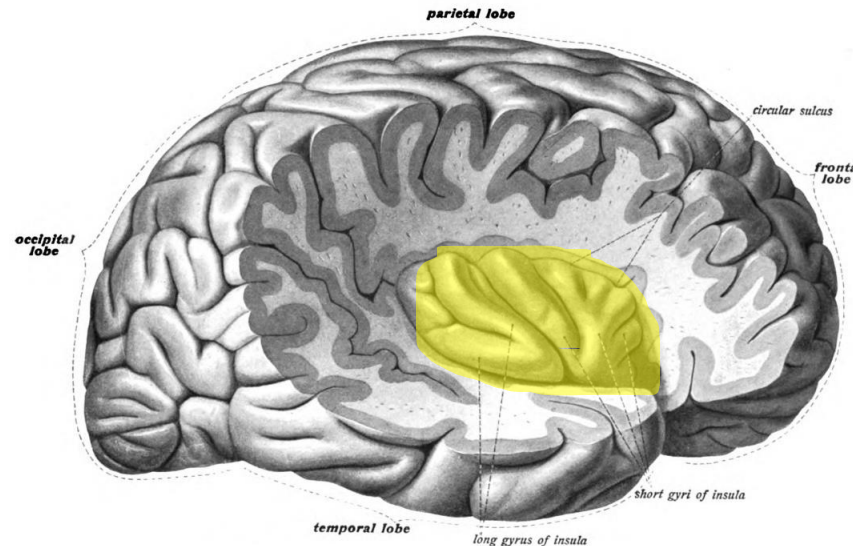
PPD: Epidemiology

- Most often seen in psychiatric illnesses
 - 6%-18% of psychiatric inpatients
- 80% of pts with PPD have schizophrenia
 - ASD, IDD, middle-aged women
- Up to 20% schizophrenic pts with PPD
 - 20% develop hypoNa
 - 5% develop serious complications
 - Px: increased morbidity, mortality



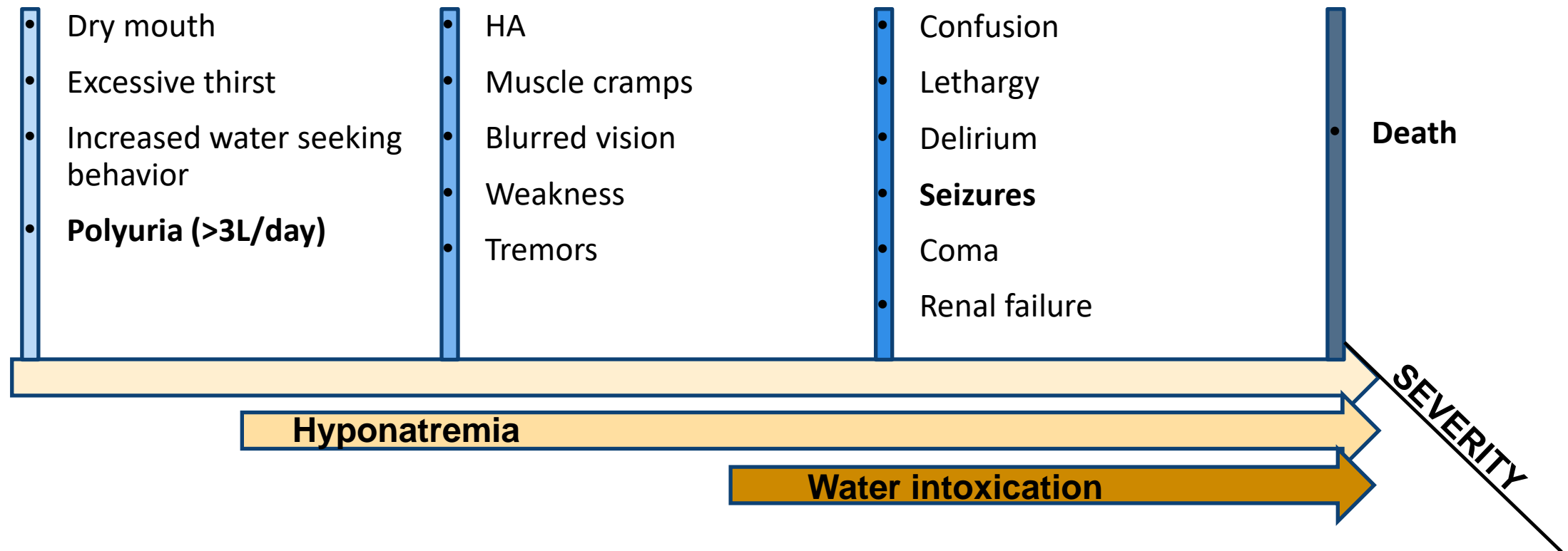
PPD: Neuroimaging

- Volume reduction
 - Rt posterior lobe
 - Rt inferior temporal gyrus
 - **Hippocampus**
 - **Insular cortex**
- Cause and effect?
 - Dysfunction of thirst mechanism
 - Involvement of hippocampus
 - Stress-reducing behaviors





PPD: Symptoms





Normal homeostasis

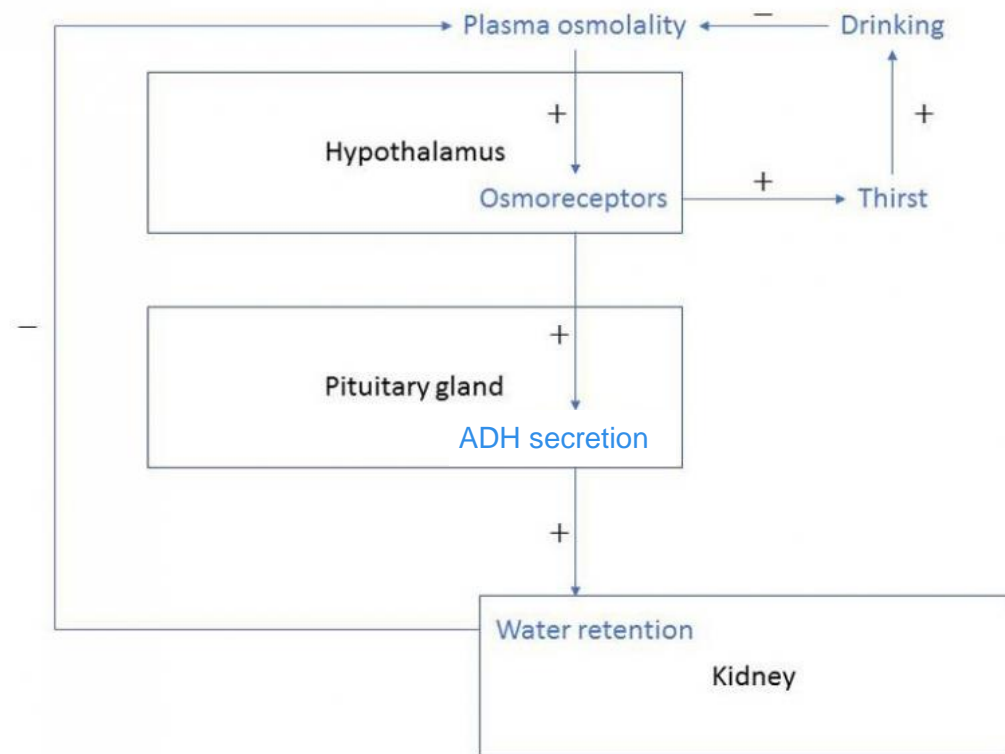


Fig. 1:
ADH feedback loop responding to increased plasma osmolality

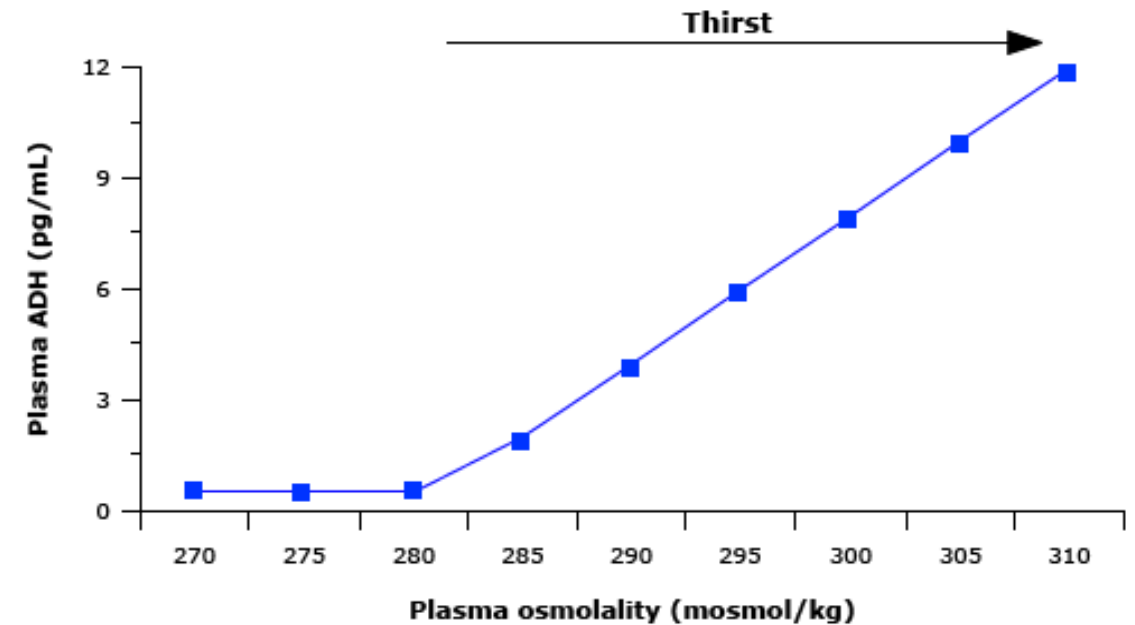


Fig. 2:
Osmotic regulation of ADH release and thirst



Hypotonic hyponatremia

- $[\text{Na}] < 135 \text{ mEq/L}$

130 to 134 mEq/L = mild

120 to 129 mEq/L = moderate

<120 mEq/L = severe

- Chronic hypoNa in PPD
 - 'Asymptomatic'
 - ADH regulation + kidney function intact



Water intoxication

- Acute
 - 'Water poisoning', 'water toxemia'
- Pathophysiology:
 - Osmotic intracellular shift --> cellular swelling
 - ICP increased: first observable symptoms
 - Respiratory distress, abnl VS
 - Cerebral edema, CNS dysfunction
 - Seizures, brain damage, death



Water intoxication

- NI ADH-mediated kidney function:
 - Up to 400-600ml urine excreted/ hr
 - Maximally dilute urine to between 40-100mosml/kg
- Symptomatic hypoNa can be induced with an acute 3-4L water load
 - Non- PPD examples
 - (un)intentional OD
 - PPD with acute psychosis



USG = 1.004
Uosm = 105
Ucolor = 5



USG = 1.016
Uosm = 522
Ucolor = 4



USG = 1.035
Uosm = 1257
Ucolor = 7



Water intoxication

- Psychotic pts have altered H₂O metabolism
 - Transient stimulation of ADH --> SIADH
 - Increase in net renal response to ADH
 - Iatrogenic drug adverse effects
 - Increase ADH effect: AP, AD, AED, Li
 - Xerostomia
- **Hospital referral**
 - Na <120 mEq/L
 - Severe symptoms





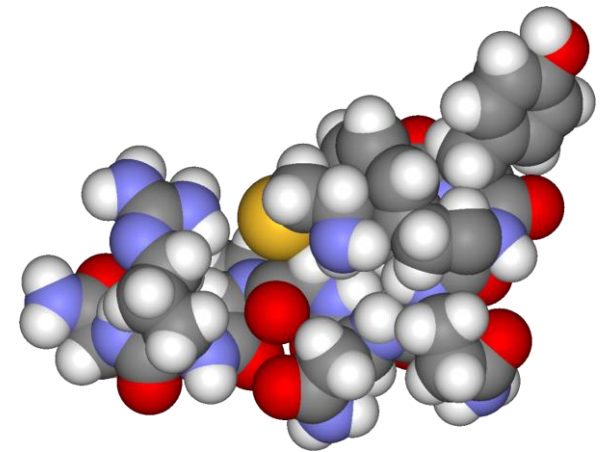
Primary Polydipsia: DDx





PPD DDx

- **Polyuria + NI Na**
 - Distinguish between osmotic vs water diuresis
 - 24hr urine volume collection
- Osmotic (solute) diuresis
 - $U_{osm} > 600$ osmol/kg
 - Glucosuria
- Water diuresis
 - $U_{osm} < 300$ osmol/kg
 - **PPD** vs. central or nephrogenic DI
 - H₂O restriction +/- desmopressin challenge



Spacefilling model of ADH

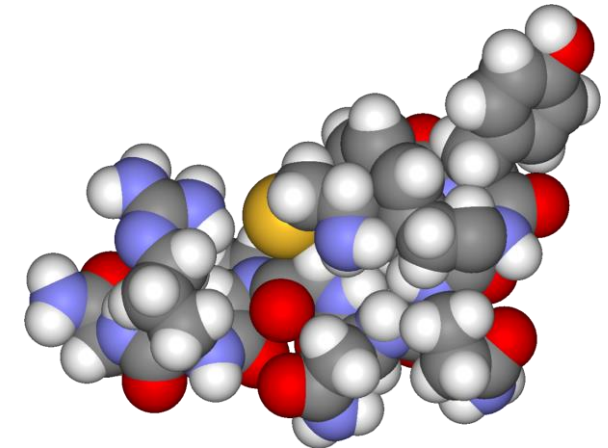


PPD DDx

- Hyponatremia $[\text{Na}] < 135\text{mEq/L}$:
 - SIADH
 - CNS disturbances
 - Malignancies
 - Drugs

Testing:

CMP
Uosm
Urine Na
CBC



Spacefilling model of ADH



Psychotropics and SIADH

- Mood stabilizers
 - Carbamazepine, oxcarbazepine, VPA, lamotrigine
 - 5% -40% incidence rate
- AD
 - SSRIs, SNRIs, TCAs
 - Serotonin effects 5-HT₂/ 5-HT_{1C} increase ADH
- AP
 - Haloperidol, thioridazine vs. SGA



PPD vs SIADH

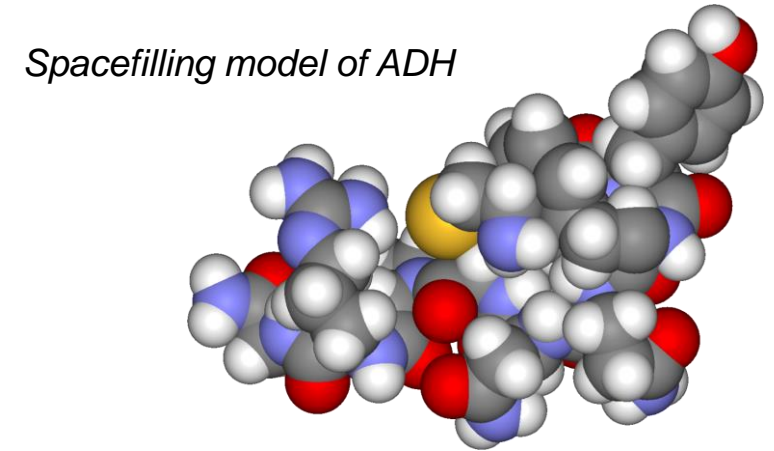
Disease	Signs/symptoms	Lab values/notes
Hyponatremia from polydipsia	Lethargy Confusion Seizures Ataxia	Serum osmolality: <280 mOsm/kg Urine osmolality: <100 mOsm/kg Urine sodium: ≤ 20 mmol/L Serum sodium: <135 mmol/L
SIADH	Lethargy Confusion Seizures Ataxia	Serum osmolality: <280 mOsm/kg Urine sodium: >40 mmol/L Patient is receiving a medication that can cause SIADH (antidepressants, antipsychotics, mood stabilizers)

Diagnosis of SIADH vs psychogenic polydipsia



PPD DDx

- Polyuria + nl Na:
 - Osmotic diuresis
 - Glucosuria
 - Water diuresis
 - **PPD**
 - Central DI
 - Nephrogenic DI
- Hyponatremia:
 - SIADH
 - CNS disturbances
 - Malignancies
 - Drugs



Testing:

CMP
Uosm
Urine Na
CBC



PPD Management





General strategies for the psychiatrist

- Develop rapport/ psychoeducate
- Inquire about underlying psychiatric symptoms
- Review psychotropic medications
- Consult with medical service
- Referral to a higher level of care
- Encourage communication amongst treatment/ custodial team members



Inpatient units/ infirmarium-level care

- Restrict fluids
 - 2-3L day + 'token economy'
- Monitor adherence
 - Twice daily wt check
 - Check Na levels frequently
- Communication and collaboration!



Literature review

- Limited evidence indicating efficacy in decreasing polydiptic behavior
 - Clozaril
 - Phenytoin
 - Bupropion
 - Inderal
 - Demeclocycline



Conclusions





Learning objectives revisited

- **LO1:** Review the epidemiology and pathophysiology of primary polydipsia
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References

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Questions?



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