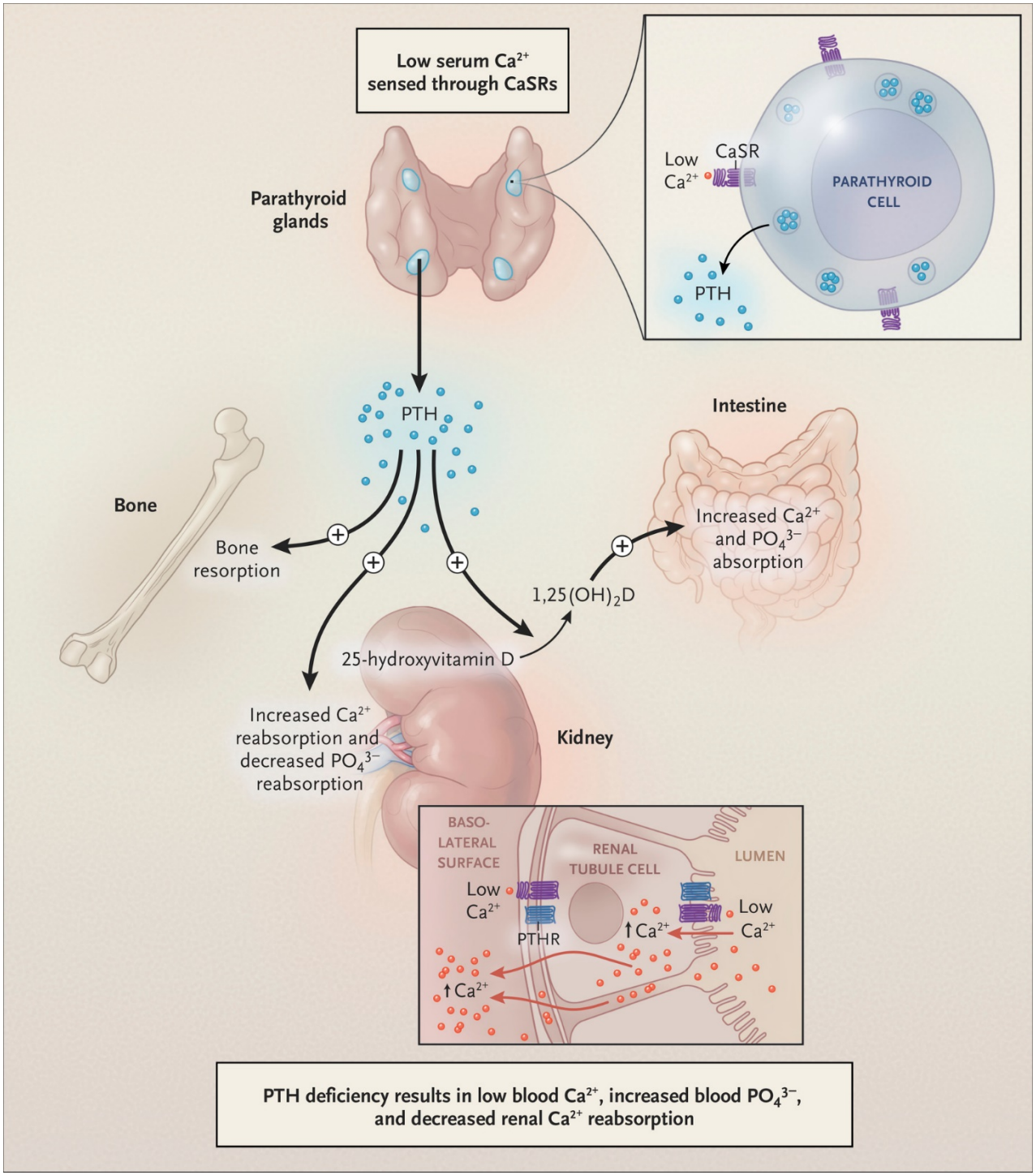
Table 1. Medications for the Treatment of Hypoparathyroidism.*

Medication	Formulation	Route	Administration in Adults	Comments
Calcium				
Calcium gluconate 10%	93 mg of elemental calcium per 10 ml of solution	Intravenous	Bolus: 10–20 ml over 10–15 min; continuous infusion: 1.25 mg of elemental calcium per kilogram of body weight per hour	9.3% elemental calcium; ECG monitoring recommended
Calcium chloride 10%	270 mg of elemental calcium per 10 ml of solution	Central venous catheter only	Bolus: 5–10 ml over 5–10 min	27% elemental calcium; ECG monitoring recommended
Calcium carbonate	Suspension: 100 mg of elemental calcium per 1 ml of solution; tablets and capsules: 160–600 mg of elemental calcium; chews: 500 mg of elemental calcium per chew; and Cal-EZ: 1000 mg of elemental calcium per packet	Oral	0.5–2 g of elemental calcium, divided into 2–4 doses per day	40% elemental calcium; ideally taken with meals for better absorption and to act as a phosphate binder
Calcium citrate	Tablets: 180–760 mg of elemental calcium	Oral	0.5–2 g of elemental calcium, divided into 2–4 doses per day	21% elemental calcium; ideally taken with meals for better absorption and to act as a phosphate binder
Calcium glubionate	115 mg elemental of calcium per 5 ml of solution	Oral	0.5–2 g of elemental calcium, divided into 2–4 doses per day	6.4% elemental calcium; ideally taken with meals
Magnesium				
Magnesium sulfate	492 mg of elemental magnesium per 1 ml of solution	Intravenous	Bolus: 1–2 g over 2–15 min; continuous infusion: 4–8 g (40–81 mg of elemental magnesium) over 24 hr	9.86% elemental magnesium; ECG monitoring recommended
Magnesium oxide	Tablets or capsules: 250–500 mg	Oral	250–1000 mg of elemental magnesium, divided into 2–4 doses per day	60% elemental magnesium
Vitamin D				
Ergocalciferol	Liquid: 8000 IU/ml; capsules: 50,000 IU	Oral	400–4000 IU/day with calcitriol†; 10,000–100,000 IU/day without calcitriol	Target 25-hydroxyvitamin D level: 20–60 ng/ml with calcitriol or >80 ng/ml without calcitriol
Cholecalciferol	Liquid: 400 IU/ml; capsules: 400–50,000 IU	Oral	400–4000 IU/day with calcitriol†; 10,000–100,000 IU/day without calcitriol	Target 25-hydroxyvitamin D level: 20–60 ng/ml with calcitriol or >80 ng/ml without calcitriol
Calcitriol	Liquid: 1 μg/ml; capsules: 0.25 and 0.5 μg	Oral or intravenous for liquid‡; oral for capsules	0.25–3 μg/day, divided into 2 doses per day	Onset of action, 1–2 days; offset of action, 2–3 days; half-life, 5–8 hr
Alfacalcidol	Liquid: 2 μg/ml; capsules: 0.25, 0.5, and 1 μg	Oral or intravenous for liquid‡; oral for capsules	0.25–3 μg/day	Onset and offset of action, within 3 days; half-life, 3–6 hr; not available in the United States
Human recombinant parathyroid hormone				
PTH 1–84§	Pen injector: 25, 50, 75, or 100 μg	Subcutaneous	Start with 50 μg/day; adjust by 25 μg every 4 wk; maximum dose, 100 μg/day	Fixed-dose pen; calcium and calcitriol supplementation may still be appropriate
PTH 1–34	Pen injector: 20 μg	Subcutaneous	NA	Not approved for treatment of hypoparathyroidism

* ECG denotes electrocardiogram, NA not applicable, and PTH parathyroid hormone.
† This dose is used as needed to maintain normal blood levels of vitamin D.
‡ Intravenous therapy is used in patients who are unable to take medications orally.
§ PTH 1–84 is the only medication that has been approved specifically for use in patients with hypoparathyroidism, and it has been approved for use only in adults.



Gafni R.I., N Engl J Med 2019; 380

ecrease in the blood calcium level triggers a cascade of events, primarily mediated through the ac
parathyroid hormone (PTH) and ionized calcium (Ca^{2+}) on the PTH receptor and the calcium-sen
eptor (CaSR), both of which are 7-transmembrane, G-protein–coupled receptors. At the parathy
nd, both the rate and magnitude of change in the blood calcium level are detected by CaSRs
ponse to a decreasing calcium level, PTH secretion is triggered (blue spheres).[4] PTH has d
neral-regulating effects at the bone, where it promotes the release of calcium and phosphate (PO_4^{3-}
ans of bone resorption. At the kidney, it facilitates reabsorption of calcium (orange spheres) from
ate to the blood while concurrently inhibiting phosphate reabsorption from the filtrate, thus promo
sphate excretion into the urine. PTH acts indirectly at the gut through the action of PTH-stimul
al production of 1,25-dihydroxyvitamin D ($1,25[OH]_2D$) to increase calcium and phosphate absorp
m the gut. Much of the fine-tuning of blood and urinary calcium and phosphate levels takes place a
ney through the action of PTH, the CaSR, and fibroblast growth factor 23 (FGF-23; not shown). R
neral homeostasis regulation is complex. A generic renal tubule cell is shown and highlights the
t the PTH receptor and CaSR can be found on both the luminal (urine) and basolateral (blood) sur
cells. Their expression level and action vary along the nephron, depending on the function of
ment. The physiological response to a decrease in the blood calcium level (depicted in the bot
et as low Ca^{2+} on the basolateral side and in the filtrate on the luminal side), is to promote
bsorption of calcium from the filtrate by means of both paracellular and transcellular mechani
licated by the orange arrows) to return the blood calcium level to normal. In the hypoparathyroid s
entire cascade is perturbed, and PTH-mediated calcium and phosphate regulation is disrupted.
od calcium level is decreased, urinary calcium excretion is increased, and the blood phosphate lev
vated. Decreased renal calcium reabsorption can manifest as an inappropriately normal urine calc
el or as an elevated urine calcium level in the context of a low blood calcium level.