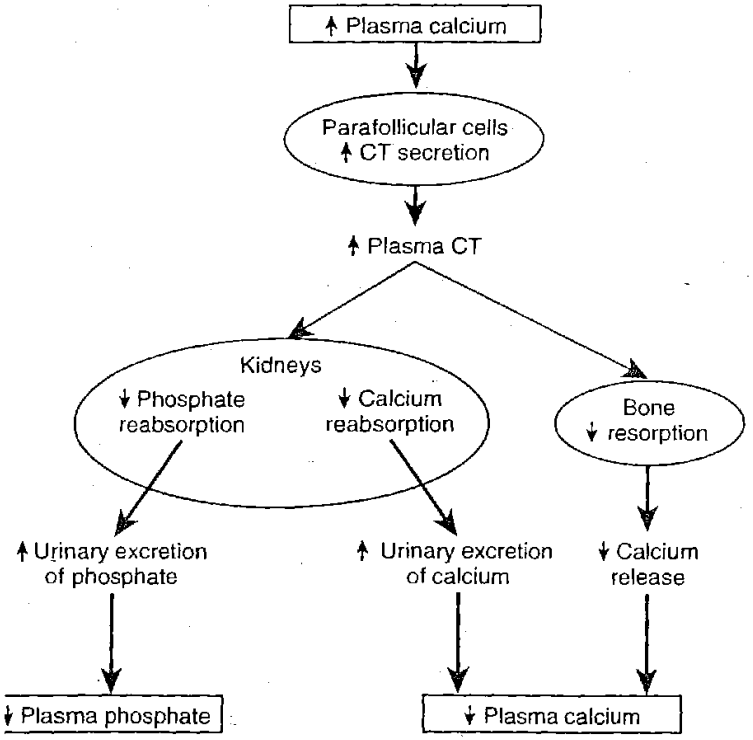

FIGURE 36.8 **Effects of calcitonin (CT) on calcium and phosphate metabolism.**

Table 27–1
Some of the Physiological Actions of Calcium

1. Required for the maintenance of normal sodium permeability in nerves
2. Involved in triggering the release of acetylcholine from nerve endings at the neuromuscular junction
3. Involved in excitation-contraction coupling in muscle cells
4. Serves as an intracellular signal for some hormones
5. Required by some enzymes for normal activity
6. Required for blood clotting to occur normally
7. Required for protein secretion
8. Constituent of bone

Table 27–4
Major Inorganic Constituents of Bone

Constituent	Total Body Content Present in Bone (%)
Calcium	99
Phosphate	85
Carbonate	80
Magnesium	50
Sodium	35
Water	9

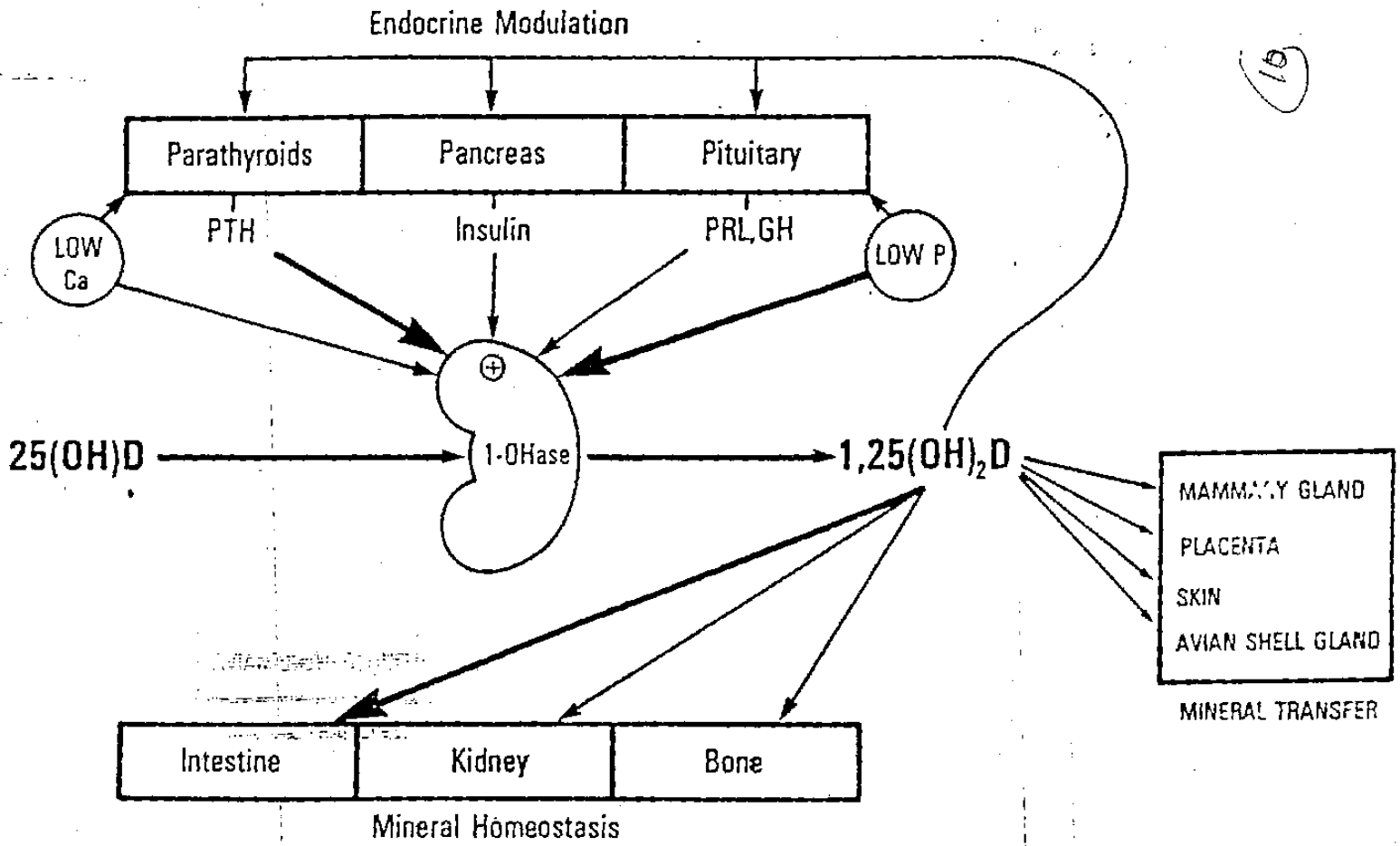

Figure 8.41. Function and regulation of 1,25-$(OH)_2D$. (From Haussler and McCain, 1977.)

Table 21–1. Distribution (mmol/L) of calcium in normal human plasma.

Diffusible		1.34
Ionized (Ca^{2+})	1.18	
Complexed to HCO_3^-, citrate, etc	0.16	
Nondiffusible (protein-bound)		1.16
Bound to albumin	0.92	
Bound to globulin	0.24	
Total plasma calcium		2.50

⊛ Ionized Ca^{++} concentration, depends on blood pH. Alkalosis increases the protein-bou and decreases the ionized Ca^{++} concentration, whereas acidosis has the opposite eff. ect.

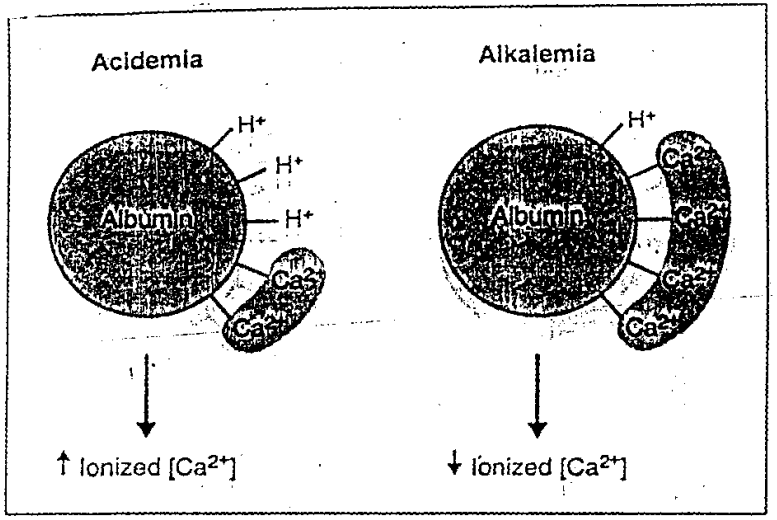

FIGURE 9–32. Effects of acid-base disturbances on plasma protein-binding of Ca^{2+} and the ionized Ca^{2+} concentration in blood.

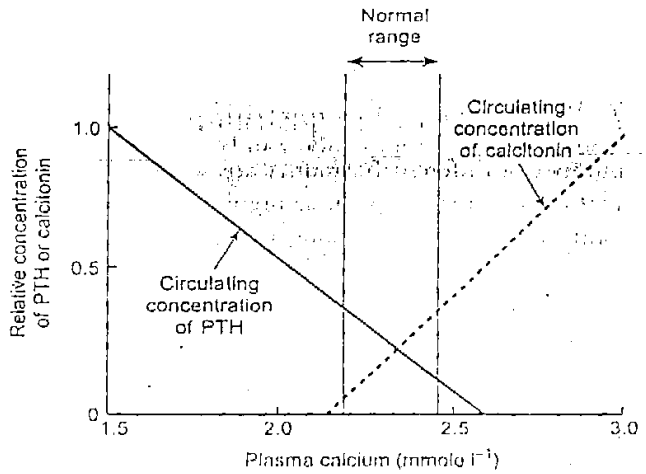

Fig. 12.25 The relationship between the plasma calcium concentration and the secretion of both parathyroid hormone and calcitonin. As calcium rises, the secretion of parathyroid hormone falls while that of calcitonin rises.

28

Calcitonin

1. Calcitonin, a straight-chain peptide of 32 amino acids, has a molecular weight of 3400.
2. The biologically active core of the molecule probably resides in its central region.
3. Calcitonin is secreted by thyroid parafollicular cells known as "C" cells.
4. Calcitonin, (CT), decreases plasma calcium levels by antagonizing the actions of PTH on bone.
5. Calcitonin is also present in nervous tissue, where it may function as a neuromodulator.
6. The major stimulus to CT secretion is a rise in plasma calcium concentration.
7. The hypocalcemic action is caused by inhibition both of osteocytic osteolysis & osteoclastic bone resorption particularly when these are stimulated by PTH.
8. However, with respect to phosphate, it has the same net effect as PTH; that is, CT decreases plasma phosphate concentration & increases urinary phosphate excretion slightly.
9. The importance of CT in humans is controversial CT deficiency does not lead to hypercalcemia & CT hypersecretion does not produce hypocalcemia. It may be that abnormal CT secretion is easily compensated for by adjustment in PTH & vitamin D levels.
10. Is degraded within the liver & kidney, after half-life of 30-60 minutes.

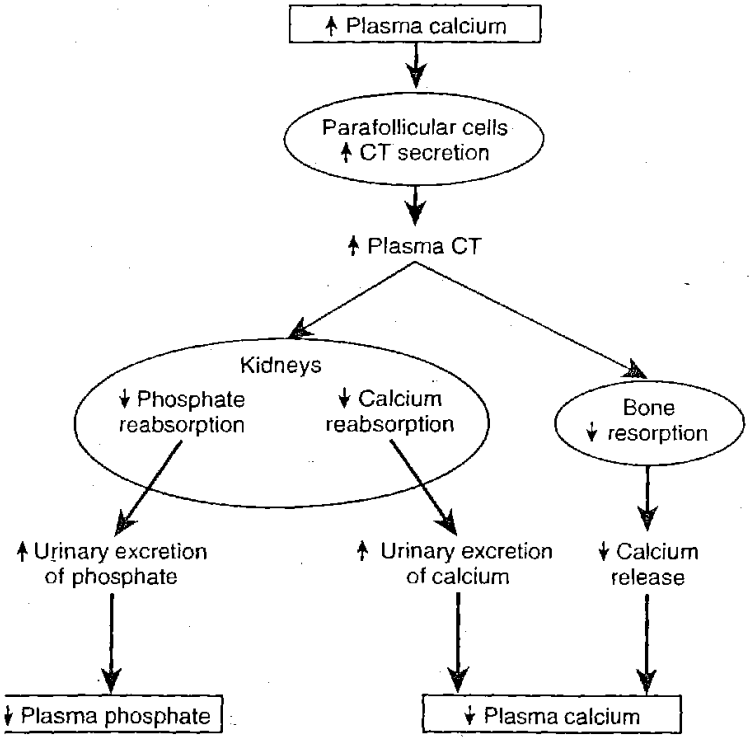

FIGURE 36.8 **Effects of calcitonin (CT) on calcium and phosphate metabolism.**

Table 27-2
Some of the Physiological Actions of Phosphate

1. Functions as part of the intracellular buffer system
2. Important constituent of a variety of macromolecules, such as nucleic acids, phospholipids, metabolic intermediates, and phosphoproteins
3. Constituent of bone

① Ca^{++}, PO_4^{---} and Mg^{++} homeostasis are essential for health and life. A complex system acts to maintain normal body contents and ECF levels of these minerals in the face of environmental (e.g., diet) and internal (e.g., pregnancy) changes.

② The key elements in the system are:-
Ⓐ- vit. D. Ⓑ- PTH. Ⓒ- calcitonin. Ⓓ- other hormones

③ The G.I.T, the kidneys, the skeleton, the skin and the liver are involved in the homeostatic regulation.

RICKETS

Rickets occurs mainly in children as a result of calcium or phosphate deficiency in the extracellular fluid. Yet, ordinarily rickets is due to lack of vitamin D, rather than a dietary lack of calcium or phosphate. If the child is properly exposed to sunlight, the 7-dehydrocholesterol in the skin becomes activated by the ultraviolet rays and forms vitamin D_3, which prevents rickets by promoting calcium and phosphate absorption from the intestines, as discussed earlier in the chapter.

Children who remain indoors through the winter in general do not receive adequate quantities of vitamin D without some supplementary therapy in the diet. Rickets tends to occur especially in the spring months because vitamin D formed during the preceding summer is stored in the liver and is still available for use during the early winter months. Also, calcium and phosphate absorption from the bones can prevent clinical signs of rickets for the first few months of vitamin D deficiency.

Osteomalacia

Osteomalacia is rickets in adults and is frequently called "adult rickets."

Normal adults rarely have a serious *dietary* deficiency of vitamin D or calcium because large quantities of calcium are not needed for bone growth as in children. However, a serious deficiency of both vitamin D and calcium occasionally occurs as a result of steatorrhea (failure to absorb fat), for vitamin D is fat-soluble, and calcium tends to form insoluble soaps with fat; consequently, in steatorrhea both vitamin D and calcium tend to pass into the feces. Under these conditions an adult occasionally has such poor calcium and phosphate absorption that adult rickets can occur, though this almost never proceeds to the stage of tetany—but very often is a cause of severe bone disability.

OSTEOPOROSIS

Osteoporosis, the most common of all bone diseases in adults and especially in old age, is a different disease from osteomalacia and rickets, for it results from diminished organic matrix rather than abnormal bone calcification. Usually, in osteoporosis the osteoblastic activity in the bone is less than normal, and consequently the rate of bone deposition is depressed. But occasionally, as in hyperparathyroidism, the cause of the diminished bone is excess osteoclastic activity.

CAUSES OF OSTEOPOROSIS ARE:

1) Lack of physical stress on the bones because of inactivity.
2) Malnutrition to the extent that sufficient protein matrix cannot be formed.
3) Lack of vitamin C,
4) Postmenopausal lack of estrogen secretion.
5) Old age, in which many of the protein anabolic functions are poor .
6) Cushing's disease, because massive quantities of glucocorticoids cause decreased deposition of protein.
7) Acromegaly, possibly because of lack of sec hormones, excess of adrenocortical hormnes, and often lack of insulin because of the diabetogenic effect of growth hormone.