Parathyroid Hormone Response to Exercise

1. Parathyroid hormone (PTH) is the major hormone:

- Regulating calcium metabolism
- Involved in both catabolic and anabolic actions on bone

2. Intermittent PTH exposure stimulates bone formation and bone mass when PTH injected.

3. In contrast, continuous infusion of PTH stimulates bone resorption.

4. PTH concentration is affected by physical exercise.

5. The variation in PTH concentration appears to be influenced by both:

- Exercise duration
- Intensity

6. There probably exists a stimulation threshold of exercise to alter PTH.

7. PTH regulation is also influenced by the:

- Initial bone mineral content
- Age
- Gender
- Training state
- Other hormonal and metabolic factors (catecholamine, Lactic acid and Calcium concentrations)

8. Physical exercise has frequently been shown to induce bone mass gain, especially in load-bearing bone sites (Maïmoun et al., 2003). Exercise thus be an important factor in preventing osteoporosis (Dalsky, 1987), by either increasing the peak bone mass during childhood growth (Bradney et al., 1998) or decreasing the rate of bone loss in the elderly (Lane et al., 1990).

9. PTH being the major regulator of bone metabolism functions to maintain the calcium-ion concentration of the extracellular fluids within physiological limits.

10. PTH is also a primary determinant of intracellular calcium homeostasis.

11. The principal target organs for PTH are the kidney (increasing proximal tubular resorption of calcium, phosphate excretion and 1,25 Dihydroxy vitamin D formation) and the skeleton. An indirect effect, increasing intestinal calcium absorption, is mediated by the increase in 1,25 Dihydroxy vitamin D formation in the kidney.

12. PTH has biphasic effects on bone:

- Continuous treatment is catabolic
- Intermittent treatment is anabolic

13. Several investigations showed that graded exercise until exhaustion (Brahm et al., 1997a) and continuous (2 exercises of 21 minutes each at respectively 70 and 85% of VO_2max) or intermittent (2 exercises of 21 minutes each at respectively 70 and 85% of VO_2max separated by 40 minutes recovery) sub-maximal exercise (Bouassida et al., 2003) enhances PTH concentrations.

14. Although the effects of PTH on bone metabolism have been intensively studied, there is a paucity of literature relating the effect of physical exercise on PTH concentrations.

15. The potential contribution of physical activity to increase bone mass is important in children and adolescents since BMD reaches about 90% of its peak by the end of the second decade (Glastre et al., 1990) and because about one quarter of adult bone is accumulated during the two years that surround the peak bone velocity (Baily, 1997). This supports the idea that patterns of physical activity during childhood and adolescence can act to prevent bone disorders (like osteoporosis) later in life.

16. Biological Effects of Parathyroid Hormone on Bone

1. A key factor in the control of bone remodeling is parathyroid hormone,

2. The principal regulator of calcium homeostasis.

3. Calcitonin and Catecholamine also modify PTH secretion.

4. Elevated levels of PTH increase bone turnover, leading to either anabolic or catabolic effects on the skeleton depending upon the pattern and duration of elevation.

5. The normal reference range of PTH concentrations is 0.5-5.0 $pmol^{-1}$ in young adults and is 0.40-1.08 IU in males below the age of 50.

Anabolic Effects of PTH on Bone:

1. The mechanisms responsible and implied in these two mechanisms are not yet entirely elucidated.

2. It has been proposed that intermittent PTH injection exerts its anabolic effects at three steps of bone formation:

(1) Stimulating the proliferation of pre-osteoblasts

(2) Promoting the differentiation of pre-osteoblasts and osteoblasts

(3) Inhibiting osteoblast apoptosis

Catabolic Effects of PTH on Bone:

1. The catabolic effects of PTH result from pathological conditions in which one or more parathyroid glands secrete too much hormone continuously at a sustained level. Such continuous secretion of PTH (as occurs in chronic renal disease and primary hyperparathyroidism) can lead to bone destruction.

Less is known of the mechanisms whereby continuous PTH is catabolic to the bone.

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