Residual Ridge Resorption (RRR)

Residual bone: that component of maxillary or mandibular bone that remains after the teeth are lost.
Residual ridge: the portion of the residual bone and its soft tissue covering that remains after the removal of teeth
Residual ridge crest: the most prominent continuous surface of the residual ridge, not necessarily coincident with the center of the ridge.
Residual ridge resorption: is a term used for the diminishing quantity and quality of residual ridge after teeth are extracted. It is a chronic, progressive and irreversible process with the rate being fastest in the first 6 months after extraction. The size of the residual ridge is reduced most rapidly in the first six months, but the bone resorption activity of the residual ridge continues throughout life at a slower rate, resulting in removal of a large amount of jaw structure. This unique phenomenon has been described as residual ridge reduction.
Post tooth extraction, a cascade of inflammatory reactions is immediately activated, and the extraction socket is temporarily sealed by blood clotting. Epithelial tissues begin its proliferation and migration within the first week and the disrupted tissue integrity is quickly restored. Histologic evidence of active bone formation in the bottom of the socket is seen as early as 2weeks after the extraction and the socket is progressively filled with newly formed bone in about 6 months. The most striking feature of the extraction wound healing is that even after the healing of wounds, the residual ridge alveolar bone undergoes a lifelong catabolic remodeling.
The rate of RRR is different among persons and even at different times and sites in the same person.

A basic concept of bone structure and its functional elements must be clear before bone resorption can be understood. The structural elements of bone are:
a. Osteocytes: These are cells responsible for metabolic activity of bone.

b. intercellular substance or bone matrix consisting of fibrils or called Calcified cementing substance: The calcified cementing substance consists mainly of polymerized glycoprotein. Mineral salts namely calcium carbonate and phosphates are bound to these protein substances.

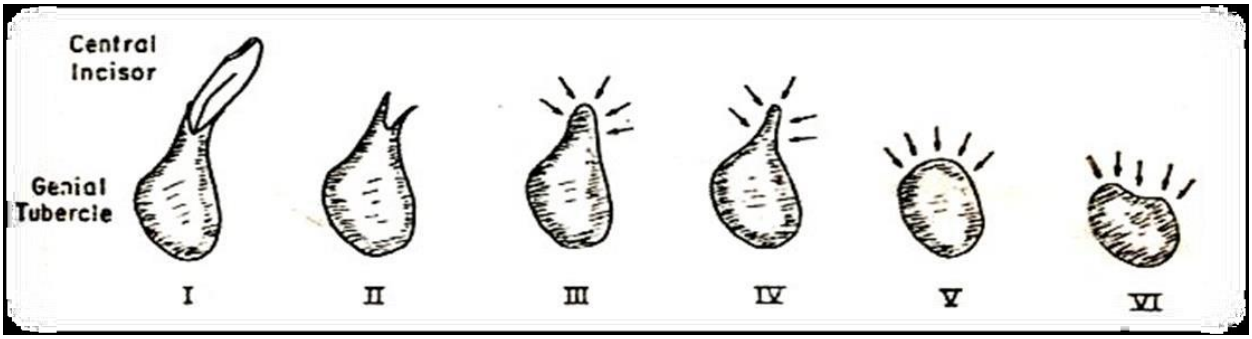
c. Osteoblasts: Osteoblasts, by their function of forming and calcifying the intercellular substance, are the active bone forming cells. The osteoblasts surround the bone in a continuous layer. In the course of bone formation, some osteoblasts get engulfed in the intercellular substance and become osteocytes.

d. Osteoclasts: Osteoclasts are the cellular components of bone that are responsible for bone resorption. Bone resorption always requires the simultaneous elimination of the organic and inorganic components of the intercellular substance.
Alveolar bone has two structural characteristics. A hard compact outer layer is superimposed on a spongy somewhat resilient substructure. A healthy and thoroughly healed alveolar process has a layer of wear resistant compact bone of varying thickness. Beneath the compact bone is the spongy bone. The spaces between the trabeculae communicate throughout the spongy bone. Bone is constantly undergoing changes in response to replacement and functional demands.

Pathology of RRR

1.Gross Pathology:

A frequently expression for RRR is “My gums have shrunk”. Actually the basic change in RRR is a reduction in the size of the bony ridge under the mucoperiosteum. It is primarily a localized of bone structure. Sometimes it may leave the overlying mucoperiosteum excessive and redundant.
There exists a wide variety of shapes and sizes of residual ridges.
They are categorized into common residual ridge configuration in a system of six orders given by Atwood Order
Order 1 : Pre-extraction
Order 2 : Post extraction
Order 3 : High, well rounded
Order 4 : Knife-edge
Order 5 : Low, well rounded
Order 6 : Depressed



RRR does not stop with residual ridge, but may go well below where apices of teeth were, sometimes leaving only a thin cortical plate on the inferior border of the mandible or virtually no maxillary alveolar process of the upper jaw. In clinical examination usually one can visually judge the residual ridge form. However, sometimes a knife-edge ridge may be masked by redundant or inflamed soft tissues.

2.Microscopic Pathology: Microscopic studies have revealed osteoclastic activity on the external surface of the crest of residual ridges. The scalloped margins of Howships lacunae sometimes contain visible osteoclasts which cause bone resorption. There exists a wide variation in the configuration, density and porosity of the residual ridges, sometimes even with evidence of osteoporosis.

Studies have shown the presence of new bone and reversal lines inside the residual ridge and minute areas of bony repair on the periosteal side in some specimens. The mucoperiosteum shows varying degrees of keratinization, acanthosis, edema and architectural pattern of mucosal epithelium in the same mouth and between subjects. Similarly, varying degrees of inflammatory cells are found in areas that appear from clinically normal to frankly inflamed in edentulous patients or who were denture or non-denture wearers. Inflammatory cells include lymphocytes and plasma cells. There exists proximity of small blood vessels to area of bone resorption.

Pathogenesis of RRR:

Immediately following the extraction (order II), any sharp edges remaining are rounded off by external osteoclastic resorption, leaving a high well rounded residual ridge (order III). As resorption continues from the labial and lingual aspects, the crest of the ridge becomes increasingly narrow ultimately becoming knife-edged (order IV). As the process continues, the knife-edge becomes shorter and even eventually disappears, leaving a low well rounded or flat ridge (order V).

Eventually, this too resorbs, leaving a depressed ridge (order VI).RRR is chronic, progressive, irreversible and cumulative.

According to the American college of prosthodontists: Based on Bone Height (Mandible only)

Type I : Residual bone height of 21 mm or greater measured at the least vertical height of the mandible.
Type II : Residual bone height of 16 - 20 mm measured at least vertical height of the mandible.
Type III : Residual alveolar bone height of 11 - 15 mm measured at the least vertical height of the mandible.
Type IV : Residual vertical bone height of 10 mm or less measured at the least vertical height of the mandible.

Direction of Bone Resorption

* **Maxilla resorbs upward and inward** to become **progressively smaller** because of the direction and inclination of the roots of the teeth and the alveolar process.
*The opposite is true of **the mandible, which inclines outward** and becomes **progressively wider**.
* This progressive change of the edentulous mandible and maxilla makes many patients appear prognathic.
Thus, RRR is centripetal in maxilla and centrifugal in mandible.

Patterns of bone resorption

In the **Mandible**, large proportions of bone loss occur in the
- labial side of anterior residual ridge,
- equally on the buccal and lingual side in premolar region and
- lingually in the posterior or molar region.

In the **Maxilla** bone loss primarily occurs on the labial or buccal aspect.
Therefore, while teeth arrangement we should try to restore the natural position of the teeth before they were lost.
- Teeth in the maxillary arch are arranged slightly labially and buccally.
- While in the mandible, teeth in the anterior region are arranged labially, on the center of the ridge in the premolar region and slightly lingually in the molar region.

Maxilla V/s Mandible

- It is a clinically acknowledged fact that the anterior mandible resorbs 4 times faster than the anterior maxilla.
- Woelfel et al have cited the projected **maxillary denture area to be 4.2 sq inch and 2.3 sq in for the mandible**; which is in the ratio of 1.8:1.
- If a patient bites with a pressure of 50 lbs, this is calculated to be **12 lbs/sq inch under the maxillary denture and 21 lbs/sq inch under the mandibular denture**.

The significant difference in the two forces may be a causative factor to cause a difference in the rates of resorption.

- Cancellous bone is ideally designed to absorb and dissipate the forces it is subjected to.
- The maxillary residual ridge is often broader, flatter, and more cancellous than the mandibular ridge.
- Trabeculae in maxilla are oriented parallel to the direction of compression deformation, allowing for maximal resistance to deformation.
- The stronger these trabeculae are, the greater is the resistance

Consequences of RRR:

a. There is apparent loss of sulcus width and depth.
b. Muscle attachments are displaced closer to the crest of the residual ridge. Due to loss of VDO lower face height is reduced and mandible is rotated anteriorly.
c. Patient may develop habitual prognathic appearance.
d. Inter-alveolar ridge relationship is altered.
e. Morphological changes in residual ridge may appear such as sharp, spiny, uneven residual ridges.
f. Resorption of the mandibular canal wall and exposure of the mandibular nerve.
g. Location of the mental foramina close to the top of the mandibular residual ridge. This provides serious problems to the clinician on how to provide adequate support, stability and retention of the denture.

Etiology of RRR

In equilibrium the two antagonistic actions (of osteoblasts and osteoclasts) are in balance. Ingrowth, although resorption is constantly taking place in the remodeling of bones as they grow, increased osteoblastic activity more than makes up for the bone destruction. Whereas in osteoporosis, osteoblasts are hypoactive, and, in the resorption related to hyperparathyroidism, increased osteoblastic activity is unable to keep up with the increased osteoclastic activity.

Ridge resorption varies directly with some systemic or localized bone resorptive factors and inversely with some bone formation factors

$$RRR \alpha \frac{\text{bone resorption factors}}{\text{bone formation factors}}$$

The Systemic factors that influence the balance between the normal bone formation and bone resorption. These factors create a natural resistance to unfavorable local factors. They are:

1. Estrogen.
2. Thyroxin.
3. Growth hormone.
4. Androgens.
5. Calcium.
6. Phosphorus.
7. Vitamin D.
8. Protein.
9. Fluoride.

Some local biochemical factors in relation to periodontal disease which affects the ridge resorption-

1. Endotoxins from dental plaque on unclear dentures.
2. Osteoclast activating factor (OAF).
3. Prostaglandins.
4. Human gingival bone resorption stimulating factors.
5. Heparin acts as a cofactor in bone resorption which is produced from mast cells

RRR is a multi-factorial, biomechanical disease that results from a combination of anatomic, metabolic and mechanical determinants (functional and prosthetic).

1- Anatomic Factors: these factors includes amount of bone and quality of bone.

Amount of bone: When we clinically examine a completely edentulous foundation, we tend to gauge the residual ridge on the basis of it being high/low, broad/narrow, rounded/spiny, covered by thick/thin mucoperiosteum.

the rate of vertical bone loss in broad, high ridge may actually be slower than that of a narrow ridge because there is more bone to be resorbed per unit of time and because the rate of resorption also depends on the density of the bone.

Quality of bone*:* On theoretic grounds, the denser the bone, the slower the rate of resorption because there is more bone to be resorbed per unit of time.

2. Metabolic factors: it includes both nutritional disturbances and hormonal causative factors.

a.Hormonal Factors

1.Pituitary Glands and Hypophysis

The hypophysis is the master gland of the endocrine system. The control of the hypophysis over the endocrine system is complex and problems of dysfunction require the analysis of an endocrinologist. Such findings are of importance to the dentist because they involve the general health of the patient, which is reflected in the oral cavity.

2. ***Thyroid Glands***

They are responsible for the regulation of the rate of metabolism. Hyperthyroidism increases the metabolic rate leading to negative nitrogen balance. Such a balance is equivalent to protein deficiency, which can be a direct cause of osteoporosis. Thyroxin also has a direct influence on the kidneys, causing an increased excretion of calcium and phosphorus. This depletion of calcium and phosphorus results in decreased bone apposition and increased osteoclastic activity.

3.Parathyroid Glands

Parathormone maintains blood calcium by mobilizing it from the bones through osteoclastic activity.

4.Islets of Langerhans

The failure of these glands to produce sufficient insulin for proper utilization of glucose causes diabetes mellitus. The syndrome of poor healing, low tissue tolerance and rapid resorption of bone is associated with the diabetic patient. In the absence of insulin, a relative nitrogen starvation occurs from increased gluconeogenesis with the amino acids being diverted from protein synthesis. A diabetic controlled by either insulin or diet is not affected by this mechanism. Since perfect control is rarely possible, a word of caution and explanation to diabetic patients is necessary so that they can appreciate their prosthetic difficulties.

5.Suprarenal Glands

The adrenal cortex produces steroid hormones called corticoids. Cortisone and related steroids are antianabolic. It may induce the formation of glucose from carbohydrates and may increase the calcium loss by direct effect on calcium

excretion. The prolonged use and administration of such steroids are considered very dangerous to bone tissue. However, one of the beneficial effects of corticoids is to control the defense mechanism of inflammation.

6. Gonads

In general, the sex hormone (*androgens and estrogens*) promotes a protein anabolic action on all tissues including bone. A moderate amount of osteoporosis accompanies senescence because of the increased catabolic action reflected by atrophic and degenerative changes throughout the body. The ageing person produces decreased amount of androgens and estrogens, which results in faulty protein metabolism for tissue repair. The bone matrix suffers and normal bone loss cannot be compensated.

b. Dietary Factors

Food is classified as proteins, carbohydrates, fats, vitamins and inorganic elements.

1.Protein

Protein is necessary to build and maintain tissue and to supply energy. The synthesis of osteoid tissue in protein starved people is compromised and calcification is decreased since the protein matrix is embarrassed.

Protein may not be available because of inadequate intake, improper assimilation or excessive loss as in nephrosis or because it is utilized as calorie requirements because of hyperthyroidism/ uncontrolled diabetes.

Inadequate incorporation of protein in diet (3 ounces/ day) will cause slow growth of bone. Bone apposition cannot keep up with normal osteoclastic activity and a negative bone factor exists.

Vitamin C: Lack of vitamin causes decalcification of the bone and has been held responsible for diffuse alveolar atrophy. The apposition of new bone slows down dramatically because osteoblastic activity is impaired. The collagen content of bones is also reduced in vitamin deficiency. The periosteum thickness and the cells appear immature and resemble

2.Vitamins

The action of vitamins in many respects is said to be same as that of hormones. The relationship of vitamins and hormones can be explained on the basis that the endocrine glands produce intrinsic hormones and the vitamins are extrinsic hormones.

Vitamin A: A deficiency of vitamin A may result in poor development and calcification of bone. Prolonged deficiency of vitamin A causes renal damage by hornification of tubules, which then lose the capacity to reabsorb phosphorus. The imbalance of the calcium: phosphorus ratio leads to osteoporosis.

Vitamin B complex: The total effect of vitamin B complex is of a regulatory nature. Hypovitaminosis B results in loss of appetite, dietary insufficiency, increase in nervous irritability resulting in lowered resistance to stress and emotional tension. The total well-being of the individual is impaired.

Vitamin C: Lack of vitamin causes decalcification of the bone and has been held responsible for diffuse alveolar atrophy. The apposition of new bone slows down dramatically because osteoblastic activity is impaired. The collagen content of bones is also reduced in vitamin deficiency. The periosteum thickness and the cells appear immature and resemble fibroblasts. This condition may make the periosteum easily prone to injury by the denture base. Osteophytes appear as a result of avitaminosis C. The rapid loss of bone and the increased inflammation of the mucoperiosteum cause the development of these bony outgrowths.

Vitamin D: It is necessary for the calcium phosphorus balance to remain within tolerable limits. Vitamin D would be unnecessary if the exact required ratio of calcium and phosphorus were available in the diet. When bone loses its ability to calcify the matrix, administration of vitamin D will cause calcification and denser bone. Moderate overdosage causes excessively mineralized bone, but gross overdosage causes bone resorption. Many drugs act as vitamin antagonists. These drugs act largely on vitamin C and B complex and their excessive use may cause a marked vitamin deficiency.
Some of the common vitamin inhibitors are nicotine, alcohol, barbiturates, morphine, some of the sulfa drugs and some of the antibiotics such as streptomycin and penicillin.

3.Carbohydrates (Starch and Sugars)

They provide the chief source of energy. They are related only indirectly to bone resorption through association with diabetes and by substitution for more favorable foods.

4.Fats and Organic Substances

They are those, which yield heat and energy and only secondarily build/repair tissue.

5.Inorganic Elements

Calcium salts (calcium carbonate and calcium phosphate)
form the rigid supporting structure of bones. Phosphorus in the form of calcium and magnesium phosphate, gives hardness to bone. Abnormalities of the calcium

phosphorus elements of the blood stream may be associated with alveolar resorption or rarefaction.

The body requires 0.7 gm of calcium/day, which can be obtained from 1 quart of milk. Other sources of calcium are dairy products, spinach, oranges, celery, chard, carrots and lettuce. The phosphorus need is about1.5 to 3 gm daily dependent upon the form. Dry beans, milk, cheese, leafy vegetables, celery and carrots may fulfill these requirements.

Edentulous patients should follow a prescribed dietary regime. This diet should be low in carbohydrates and high in protein intake. The diet should include at least a quart of milk or substitute dairy products, vegetables, fruits and a multiple vitamin supplement. The normal equilibrium may be upset and pathologic bone loss may occur if either bone resorption is increased or bone formation is decreased, or if both occur.

- Since bone metabolism is dependent on cell metabolism, anything that influences cell metabolism of osteoblasts and osteoclasts is important.
- The thyroid hormone affects the rate of metabolism of cells in general and hence the activity of both, the osteoblasts and osteoclasts.
- Parathyroid hormone influences the excretion of phosphorous in the kidney and also directly influences osteoclasts.
- The degree of absorption of calcium, phosphorous and proteins determines the amount of building blocks available for the growth and maintenance of bone.
- Vit C aids in bone matrix formation.
- Vit D acts through its influence on the rate of absorption of calcium in the intestines and on the citric acid content of bone.
- Various members of Vit B complex is necessary for bone cell metabolism.
- In general terms, anabolism exceeds catabolism during growth and convalescence, levels off during most of adult life and is exceeded by catabolism during disease and old age. Bone has its own specific metabolism and undergoes equivalent changes. At no time during life is bone static, but rather it is constantly rebuilding, resorbing and remodeling subject to functional and metabolic stresses.