Reduction of residual ridges: A major oral disease entity

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Until a disease is recognized as a disease entity, little progress is made in understanding its etiology and in developing adequate treatment and prevention. For example, dental caries had to be acknowledged as a complex disease entity, not simply as cavities in the teeth, before today's massive sophisticated attack could be mounted against this almost universal oral disease.

Similarly, the reduction of the size of residual ridges must be recognized as a complex oral disease with identifiable characteristics and unwanted sequelae afflicting millions of people.

In trying to study a disease entity, whether it be heart disease or dental caries or periodontal disease, it is helpful to study its pathology (the gross and microscopic structural changes of the disease), its pathophysiology (the mechanisms or disordered functions of the disease), its pathogenesis (the life history), and its epidemiology (the worldwide prevalence of the disease and various interrelated factors). The goal of such studies is the better understanding of the etiology of the disease, with such understanding leading to better treatment and ultimately to the prevention or control of the disease.

PATHOLOGY OF RRR

The primary structural change in the reduction of residual ridges (RRR) is the loss of bone. Longitudinal cephalometric studies have provided excellent visualization of the gross patterns of this bone loss. The superimposition of tracings of cephalograms made in such studies clearly shows that reduction of the ridge occurs labially, on the crest, and lingually (Fig. 1). The rate of reduction and the total amount of bone removed in this disease vary from individual to individual, within the same individual at different times, and even at the same time in different parts of the ridge.

In some situations, RRR leaves redundant mucoperiosteum, while in others there...
Fig. 1. Tracings of three lateral cephalographs with the maxillae and mandibles carefully superimposed. Note the changes in the shape of the residual ridges following the extraction of remaining teeth 50 months before. (From Atwood, D. A.: J. PROSTHET. DENT. 13: 811, 1963.)

Fig. 2. Six orders of mandibular anterior residual ridge form: Order I, pre-extraction; Order II, post-extraction; Order III, high well-rounded; Order IV, knife-edge; Order V, low well-rounded; Order VI, depressed. (From Atwood, D. A.: J. PROSTHET. DENT. 13: 817, 1963.)

appears to be well-attached mucoperiosteum with no redundant tissue over the resorbed ridge. Similarly, there may or may not be evidence of inflammation in areas of RRR.

The reduction of the residual ridge leads to a variety of stages of ridge form, including high well-rounded, knife-edge, low well-rounded, and depressed forms (Fig. 2).

PATHOPHYSIOLOGY OF RRR

Microscopic studies have revealed evidence of external osteoclastic activity as the mechanism by which the gross bone loss occurs. Clearly, the amount of bone loss may be greater than the original thickness of the cortical bone (Fig. 3). Yet, there frequently is some semblance of a cortical layer even in the later stages of reduction (Fig. 4). This must mean that, in such patients, new bone is laid down internally while resorption occurs externally. That this bone remodeling process does not always work with equal success is shown in the many patients in whom residual ridge crest has no cortical layer.
Fig. 3. Gross bone loss of residual ridges is revealed by careful superimposition of portions of two cephalometric radiographs made 16 years apart. The actual bone loss in the anterior part of the ridge of the mandible was 13 mm. in height (a 41 per cent reduction) and 60 sq. mm. in cross-sectional area (a 24 per cent reduction).

This process of external resorption and endosteal deposition is not unique to RRR, for it is similar to one phase of bone growth as described by Enlow. Growth of a long bone such as the tibia is much more complicated than the simple periosteal deposition of bone. As long bones grow longer, they are constantly reshaped in three dimensions (Fig. 5). Without going into detail, the diameter of the metaphysis of a long bone, such as the tibia, is reduced, while the bone as a whole becomes longer. This narrowing of a portion of a bone is achieved by external resorption. However, such external resorption does not occur without endosteal deposition (Fig. 6). If no new bone were laid down endosteally, the cortex would become progressively thinner until it completely disappears. This clearly does not occur in bone growth.

The structural product of this inward growth is called “endosteal bone” and is characterized either by a convoluted whorled appearance (when growth occurs into a trabecular area) or by a zone of even, regular, uninterrupted circumferential lamellae (when bone is laid down in layers on the endosteal side of smooth cortical bone). In each instance, the configuration of the new bone is dependent upon the configuration of the bony surfaces on which the deposition occurs.

Evidence of both types of inward growth has been found in microradiographs of residual ridges, with no evidence of external growth on the crest on the ridge. The type of bone commonly found on the crest of the ridge appears to be endosteal bone of the whorled convoluted type due to the compacting of the trabecular bone by the-
deposition of layers of new bone on old trabeculae (Fig. 7). As the endosteal bone becomes compacted, it is invaded by resorption spaces and new haversian systems are formed within the compacted bone. One can find examples of both convoluted and lamellar endosteal bone in the same specimen (Fig. 8).

The reduction of the metaphysis of a long bone, as described in detail by Enlow, has been illustrated diagrammatically by the Principle of the "V" with the minus symbols representing periosteal resorption and the plus symbols standing for endosteal deposition (Fig. 9).

A modified version of this principle of bone remodeling can illustrate diagrammatically and dramatically the mechanism of the reduction of the mandibular residual ridge by external resorption accompanied by endosteal deposition—the Principle of the Inverted "V" (Fig. 10).

**PATHOGENESIS OF RRR**

After the extraction of the tooth, any sharp edges remaining (Order II) are rounded off by external resorption leaving a high well-rounded ridge (Order III). As resorption continues from the labial and lingual aspects, the ridge becomes increasingly narrow, ultimately becoming knife-edged (Order IV). As the process con-
Fig. 5. Area relocation. Level $AA'$ becomes repositioned into level $BB'$ as a result of the increased growth in length of the entire bone. The relative level at $AA'$ in the larger bone is indicated by an $X$. As a result of the relocation of $AA'$ to $BB'$, note that (1) the sectional shape and (2) the sectional diameter have changed. Note also that the point indicated by the black arrow has been relocated from the inner side of the cortex in $AA'$ to the outer side of the cortex in $BB'$. All of these changes involve structural remodeling by the processes of resorption and appositional deposition. (From Enlow, D. H.: Principles of Bone Remodeling, Springfield, Ill., 1963, Charles C. Thomas, Publisher.)

Continues further, the knife-edge ridge becomes shorter and eventually disappears, leaving a low well-rounded or flat ridge (Order V). Eventually, this too resorbs leaving a depressed ridge (Order VI) (Figs. 2 and 4).

The reduction of residual ridges is chronic, progressive, irreversible, and cumulative. The reduction of residual ridges usually proceeds slowly over a long period of time from one stage to the next. Autonomous regrowth of residual ridges has not been reported. The annual increments of bone loss have a cumulative effect, leaving less and less residual ridge.

In general, the rate of RRR varies between different individuals (Fig. 11). Within a given individual the rate is usually most rapid in the first 6 months following extraction. An interesting history of one patient shows a rapid resorption rate in the early months in both the upper and lower anterior ridge height (Fig. 12). Whereas, the upper ridge showed no measurable change after the first 3 years, the lower ridge showed a continuing RRR at a steady rate (0.4 mm. per year) over a 15-year period. The vertical bone loss of the anterior part of the ridge in 19 years was 3 mm. in the maxillae and 14.5 mm. in the mandible. This report illustrates basic principles of variation in rate of RRR within a given individual.

The reduction of residual ridges seems to be potentially unlimited. Both trabecu-
Reduction of residual ridges

Fig. 6. Inward growth is accomplished by periosteal resorption together with endosteal deposition. On side A, endosteal or inward growth is proceeding (1) into an area of the metaphysis already occupied by cancellous bone and (2) into the medulla of the shaft which does not contain cancellous trabeculae. Two different patterns of compact bone structure result from these circumstances. Illustrated on side B of the diagram, the compaction of cancellous bone produced during 1 as designated above results in a convoluted whorled arrangement of cortical bone (Y). The growth during 2 produces an inner layer or zone of even, regular, uninterrupted circumferential lamellae (X). (From Enlow, D. H.: Principles of Bone Remodeling, Springfield, Ill., 1963, Charles C Thomas, Publisher.)

lar bone and cancellous bone can be resorbed no matter how well they are calcified. RRR can go on for years, leaving grossly mutilated jawbones. RRR can go below the mucobuccal fold, the muscle attachments, the genial tubercles, the mylohyoid line, and the level of the periapical bone.

Because RRR is chronic and progressive, it results in repeated mucosal, functional, psychologic, esthetic, and economic problems for denture patients. Because it is cumulative, the patient with this disease becomes more and more dentally handicapped, ultimately a "dental cripple."

EPIDEMIOLOGY OF RESIDUAL RIDGE REDUCTION

How prevalent is RRR? Cephalometric studies have shown the presence of RRR in Finland, Sweden, Great Britain, Massachusetts, District of Columbia, Pennsylvania, Ohio, Michigan, Illinois, Kentucky, Washington, Australia, Japan, and many other places throughout the world.

However, there just are not enough vital statistics on the incidence of RRR in relation to such factors as geography, age, sex, race, body size, facial morphology, and other identifiable statistics.
Figs. 7 and 8. For legend see opposite page.
Fig. 9. The Principle of the "V". Progressive growth of a long bone in the direction of the arrow is accomplished by additions on the inside surface (+) and removal from the outer surface (−). (From Enlow, D. H.: Principles of Bone Remodeling, Springfield, Ill., 1963, Charles C Thomas, Publisher.)

Fig. 10. The Principle of the Inverted "V". A modified version of Enlow's Principle of the "V" illustrates diagrammatically the mechanism of the reduction of mandibular residual ridge by external resorption (−) accompanied by endosteal deposition (+).

Fig. 7. A microradiograph of compacted bone on the crest of a low well-rounded residual ridge showing a whorled convoluted type of endosteal bone. As the bone becomes compact, it is invaded by resorption spaces which, in turn, result in a network of haversian systems throughout the compact bone. No circumferential lamellae are seen over the ridge crest on the periosteal side.

Fig. 8. A microradiograph of compacted bone on the crest of a knife-edge residual ridge showing both types of endosteal bone. Toward the crest and on the lingual side (left), there is a whorled convoluted type of compact bone with secondary haversian systems. On the labial aspect (lower right), there is inward growth of the even, regular, uninterrupted circumferential lamellae. (From Atwood, D. A.: J. PROSTHET. DENT. 13: 820, 1963.)
Fig. 11. Two examples of RRR (reduction of residual ridge) curves illustrate variations in the rate and total amount between two subjects in the first postextraction year and variations in the rate within each subject.

Much of what has been learned about the etiology and prevention of dental caries has been learned from epidemiologic studies. Without the DMF count and the discovery of an easy laboratory method for measuring fluoride in water, the pioneers in caries research would never have found the relation between fluoride and dental caries. Since the breakthrough on measuring fluoride and with the application of epidemiologic studies, giant strides have been made in caries research. This has made possible an infinite variety of different, previously unthought-of approaches to caries research.

Research into RRR is about 30 years behind dental caries research. Yet by studying their methods and their development, we can perhaps come up with dramatic breakthroughs which will allow us to catch up quickly.

Epidemiologic studies are useful in trend-finding investigations of multifactorial diseases. It is entirely possible that RRR is a multifactorial disease and that the rate of RRR depends not on one single factor but on the concurrence of two or more factors, which may be called cofactors.

Some years ago, it was suggested that, for convenience, possible factors could be divided into four categories: anatomic, metabolic, functional, and prosthetic. The anatomic factors include such things as the size and shape of the ridge, the type of bone, and the type of mucoperiosteum.

The metabolic factors include such things as age, sex, hormonal balance, osteoporosis, etc.

The functional factors include the frequency, direction, and amount of force applied to the ridge.
Fig. 12. The RRR curves in one subject studied over a 19 year period illustrate various principles of variation within a given subject. The anterior vertical RRR in the maxillae was 3 mm. during the first three years and immeasurable thereafter; while the mandible after a dramatic early bone loss continues to show a steady reduction rate (0.4 mm. per year) to a total of 14.5 mm. in 19 years.

The prosthetic factors include the type of denture base, the form and type of teeth, the interocclusal distance, and the like.

For further convenience, since the functional factors must function through the prosthetic factors, they may be grouped together as mechanical factors.

This gives us three groups of cofactors—anatomic, biologic, and mechanical (Fig. 13).

A triad of cofactors has been described in dental caries—namely, host, substrate, and bacteria. Host factors include all the factors in the subject which contribute to host resistance or susceptibility to the disease. Substrate includes primarily sugar. Bacteria are essential to caries as has been shown by germ-free studies, and although bacteria might be termed the etiologic agent, the incidence of caries depends also on the cofactors—substrate and host. When all three are present in large amounts, one observes rampant dental caries.

Similarly, it may well be that when many anatomic, biologic, and mechanical factors coexist, the rate of RRR will be high. Whereas, if certain cofactors are absent, even if some cofactors are present to a large degree, the rate of RRR may be...
little or none. We have all seen patients with very bad dentures and little resorption and patients with good dentures and a high rate of reduction of the residual ridge. However, more often, we see bad dentures with a high rate of RRR.

**DIFFICULTIES IN STUDYING RRR**

Studying the reduction of the residual ridge is very similar to studying dental caries, and it has many of the same problems.

1. The disease is almost universal, but there are variations in amount and rate between individuals.
2. The amount is cumulative so that a single examination does not reveal the present rate (DMF count and Ridge Order).
3. The rate is slow. Therefore, lengthy longitudinal studies are required to determine the rate.
4. The rate may vary at different times and in different sites within an individual. Therefore, repeated readings at intervals are needed to reveal changes in rate.
5. The rate is very likely to be dependent, not on a single factor, but on the coexistence of several factors. Therefore, any attempt to correlate this complex problem with a single factor, such as free-way space (interocclusal distance) or age, is likely to be inconclusive.
6. Not all cofactors are easily measured. Hence, new research methods must be developed.
7. It is possible that not all cofactors are even being considered. It is entirely possible that the presence or absence of hormone “X,” or vitamin “Y,” or mineral “Z” is extremely important in the rate of residual ridge reduction.
SUMMARY

1. Reduction of residual ridges (RRR) needs to be recognized for what it is: a major unsolved oral disease which causes physical, psychologic, and economic problems for millions of people all over the world.

2. RRR is a chronic, progressive, irreversible, and disabling disease, probably of multifactorial origin. At the present time, the relative importance of various cofactors is not known.

3. Much is known about the pathology and the pathophysiology of this oral disease, but we need to know much more about its pathogenesis, epidemiology, and etiology.

4. The ultimate goal of research of RRR is to find better methods of prevention or control of the disease.

5. Over 25 million Americans are estimated to be totally edentulous. The need for the delivery of more prosthodontic care in this country alone is staggering.

6. More research in RRR with new methods and new thinking are badly needed in order to provide the best possible oral health care for millions of edentulous patients.

References

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