

**Post-extraction Remodeling of the Adult Mandible**

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**ABSTRACT:** Following tooth loss, the mandible shows an extensive loss of bone in some individuals. This may pose a significant problem in the prosthodontic restoration of function and esthetics. The many factors which have been proposed as being responsible for the inter-individual variation in post-extraction remodeling mean that a perfunctory analysis of the literature, in which well-controlled, relevant studies are scarce, may not provide the whole story. This article reviews the local and systemic factors which may play a role in the post-extraction remodeling of the mandible. Since severe residual ridge resorption may occur even when the bone status in the rest of the skeleton is good and *vice versa*, it is concluded that local functional factors are of paramount significance. It is now essential to determine how they can be modified and applied to help maintain ridge height and quality in our aging, edentulous population.

**Key words.** Oral bone loss, osteoporosis, bone turnover, mandible.

**1 Introduction**

The adult human mandible is a bone which exhibits a large degree of anatomical variability (Humble, 1936; Kuznetsova et al., 1972; Tallgren, 1972; Enlow et al., 1976; von Wowern and Stoltze, 1977; Jacobsen and Krol, 1983; Poiriot et al., 1986; Carter et al., 1991; Kingsmill and Boyde, 1998a). This variation occurs not only between subjects or as a result of aging, but also between the right and left sides in an individual. After growth has ceased, the single most important factor governing the gross morphological shape of the bone is related to the presence or absence of the teeth. After tooth extraction, there follows a phase of remodeling which may result in an extensive loss in the height of the jaws, particularly the mandible (Carlsson and Persson, 1967; Tallgren, 1972). Historically, this phenomenon is of importance, since it was the observation of the ‘waste of the sockets of the teeth’ by John Hunter in the 1750s that prompted him to consider bone as a material capable of remodeling, rather than the immutable and permanent structure it had previously been thought to be (in Cohen, 1993).

**1.1 What happens post-extraction**

Both internal and external changes occur in the mandible after the teeth are lost (Walkhoff, 1901; Neufeld, 1958; Atwood, 1963). During the initial healing phase, the sockets are filled with blood clot. Osteoprogenitor cells from the ruptured periodontal ligament differentiate into osteoblasts, invade the coagulum, and form woven bone (Roberts and Chase, 1981; McCulloch and Melcher, 1983; Lin et al., 1994), later to be replaced by coarse cancellous bone. At the same time, new bone formation is seen deep to and some distance from the socket surrounding the inferior dental canal (Boyne, 1982). The crest of the residual ridge narrows and the sharp edges of the alveolar processes are reduced (Atwood, 1963; Pietrokovski and Massler, 1967; Enlow et al., 1976). As the bone is reduced in height by perioral osteoclastic resorption, there is an accompanying endosteal apposition (Pudwill and Wentz, 1975), but at no time is new bone formation seen on the perioral surface of the residual ridge, which remains porous, never developing a complete cortical layer (Neufeld, 1958; Atwood, 1963; Pudwill and Wentz, 1975). Further internal remodeling results in a loss of organization and a thinning of the trabeculae (Neufeld, 1958) as well as disruption in the arrangement of the lamellar and Haversian systems (Seipel, 1948).

**1.2 Pattern of bone loss**

Most longitudinal studies of the changes in the external form of the bone have been carried out by measurements either from serial study casts (Pietrokovski and Massler, 1967; Likeman and Watt, 1974; Brehm and Abadi, 1980) or from radiographs (Lönberg, 1951; Atwood, 1957, 1962; Tallgren, 1957, 1966, 1967, 1969; Carlsson and Persson, 1967; Woelfel et al., 1976; Wright and Watson, 1998). These studies have shown that the loss in vertical height is greatest anteriorly (Lönberg, 1951; Carlsson and Persson, 1967). Little change is thought to occur in the region of the superior genial tubercles or the mylohyoid
and external oblique ridges, which become increasingly prominent (Neufeld, 1958; Osborne, 1963) and, in extreme cases, may require surgical reduction for the provision of dentures. The stability of these regions has been attributed to their being composed of cortical bone (Devlin and Ferguson, 1991), though functional factors are likely to have a greater influence (Klemetti et al., 1994d). In the horizontal plane, the majority of the bone loss occurs from the buccal aspect in the upper jaw, resulting in a reduction in palatal width and length, as well as height (Fish, 1947; Likeman and Watt, 1974). The situation is more complex in the mandible, with the majority of loss occurring from the labial aspect anteriorly and from the lingual aspect posteriorly (Fish, 1947; Watt and MacGregor, 1986), thus matching the regions where the cortex is inherently more porous (Atkinson and Woodhead, 1968), and where the rate of bone turnover is higher (Kingsmill and Boyd, 1998b). Some resorption is also seen buccally (Pietrokovski and Massler, 1967; Pietrokovski, 1975; Enlow et al., 1976; Wang, 1989), but the arch width of the edentulous mandible is, on average, 7 mm greater than that of the edentulous maxilla (measured immediately anterior to the retromolar maxilla, measured immediately anterior to the retromolar pads and tuberosities, respectively; Parkinson, 1978).

A cross-sectional histological study of 15 mandibles exhibiting various stages of tooth loss (Enlow et al., 1976) described variations in the areas of surface resorption and apposition in the different mandibles. This study concluded that the resorptive and depository areas are similar to those present during growth. This agrees with the findings of a longitudinal radiographic study spanning a period of 25 years (Tallgren, 1972) which showed that the intra-individual pattern of bone loss remains fairly constant, yet that there is inter-individual variation in both the pattern and rate of loss. In the subjects in this latter study, between 20 and 120 mm² of bone was lost from the lateral projection of the mandibular midline over a 13.5-year period of denture wear (see Fig.).

(1.3) Rate and duration of loss

Most of the bone loss occurs in the first year after extraction, with the highest rate being in the first few months (Atwood, 1957; Tallgren, 1966; Carlsson and Persson, 1967; Likeman and Watt, 1974). However, continued bone loss from the mandible can still be detected up to 25 years post-extraction, reducing much sooner (or even ceasing in some individuals) in the maxilla (Brehm and Abadi, 1980). The maxilla shows, on average, one-quarter of the reduction of the mandible after a period of seven years (Tallgren, 1966; Kalk and de Baat, 1989).

It has been suggested that the reduction in the rate of bone loss with time was due to the resorbing ridge losing contact with the base of the denture (Devlin and Ferguson, 1991), which would therefore no longer be subjected to unfavorable loading. This seems unlikely: Not only did Tallgren (1972) show this pattern of loss in patients with well-maintained dentures, but also even poorly fitting dentures will make contact with the underlying tissues in some regions.

(1.4) Extent of loss

Many texts loosely describe the bone that is lost after tooth extraction as being that which formerly belonged to the alveolar processes (Roth and Calmes, 1981), but since the alveolar bone is arbitrarily taken as ending at the root apices (Berkovitz et al., 1992), its delineation is lost once the teeth are removed. In 1928, Brash stated that 'there is...no essential difference between alveolar bone and the bone of the base, because the former becomes progressively transformed into the latter'. However, confusion has arisen in connection with the terminology of the bone surrounding the teeth. The alveolar bone proper (the very thin cribiform plate which immediately abuts the periodontal ligament and gives rise to the radiographic appearance of the lamina dura),

Figure. Mandibular sections from an 83-year-old female and a 69-year-old male illustrating the large differences that may be seen between individuals. In both cases, the individuals were edentulous, and the sections were taken from comparable sites, just posterior to the mental foramen. Neither mandible showed any evidence of healing of extraction sockets, indicating that tooth loss had not been a recent event. Scale marker = 1 cm.
### TABLE
Local and Systemic Factors Which Have Been Suggested as Influencing Residual Ridge Resorption

| Functional | lack of mechanical stress |
|           | absence of dentures       |
|           | presence of dentures      |
|           | nature and magnitude of   |
|           | applied force             |
|           | period of daily denture   |
|           | wear number of years of   |
|           | denture use               |
|           | number of sets of         |
|           | dentures                 |
|           | denture tooth selection   |
|           | soft linings             |
|           | muscle tone              |
| Anatomical | facial form              |
|           | original size of mandible|
|           | original depth of tooth   |
|           | sockets local bone       |
|           | quality proportion of    |
|           | extrinsic fibers         |
|           | age/availability of bone  |
|           | cells quality of the soft |
|           | tissues blood supply     |
|           | muscle attachments       |
| Inflammatory | trauma inflicted at      |
|             | extraction               |
|             | pre-existing/residual    |
|             | infection periodontal    |
|             | disease mucosal          |
|             | inflammation local       |
|             | inflammatory mediators   |
|             | denture hygiene          |
| Systemic   | age and gender           |
|           | skeletal status          |
|           | bone regulatory hormones |
|           | and dietary calcium      |

| Manson, 1976; von Wowern et al., 1979; Elovic et al., 1995 |
| Mauley and Stuart, 1937 |
| Atwood, 1979; Devlin and Ferguson, 1991 |
| Applegate, 1958; Sobolik, 1960, Ortman, 1962; Winter et al., 1974 |
| Bergman et al., 1971; Harrison, 1972 |
| Harrison, 1972 |
| de Van, 1935; Winter et al., 1974 |
| Wright, 1994 |
| Watt and MacGregor, 1986; Klemetti et al., 1994d |
| Tallgren, 1972; Mercier and Lafontant, 1979 |
| Atwood, 1963; Carlsson and Persson, 1967 |
| Atwood, 1963 |
| de Van, 1935; Baxter, 1987 |
| Landini, 1991 |
| Tonna, 1976; Atwood, 1979 |
| Atwood, 1979 |
| Ortman, 1962 |
| Watt and MacGregor, 1986 |
| Sobolik, 1960 |
| Humble, 1936; Sobolik, 1960 |
| Atwood, 1979 |
| Pudwill and Wentz, 1975; Penhall, 1980 |
| Atwood, 1979; Mundy, 1993; Klemetti, 1996 |
| Penhall, 1980 |
| Bergman et al., 1971; Harrison, 1972 |
| Henrikson and Wallenius, 1974; von Wowern & Stoltze, 1979; Ortman et al., 1989; Mohajery and Brooks, 1992; von Wowern and Kollerup, 1992 |
| Ortman, 1962; Atwood, 1979; Kribbs, 1992; Hsieh et al., 1995; Wical and Swoope, 1974b; Wical and Brussee, 1979 |

which *does* disappear completely shortly after tooth extraction (de Van, 1935), is not always distinguished clearly from the rest of the bone of the alveolar process (alveolar bone) (Becks and Grimm, 1945). To help overcome the problems in terminology, the phrase 'residual ridge resorption' has been coined to encompass all the changes that accompany bone loss after tooth extraction (Edwards, 1954).

In radiographic studies, the division between alveolar and basal bone is usually taken as lying at the level of the mental foramen, being the most readily visible anatomical landmark (Wical and Swoope, 1974a; Ward et al., 1977; Kribbs et al., 1983; Packota et al., 1988; Ortman et al., 1989; Benson et al., 1991; Hirai et al., 1993; Klemetti et al., 1994c; Taguchi et al., 1995). However, the mental foramen has no direct relation to the teeth, and in radiographic projections may lie inferior or superior to the root apices. In addition, identifying the mental foramen and inferior dental canal becomes increasingly difficult in resorbed mandibles (Ulmi, 1989), especially in advanced cases, when the canal may be exposed by the resorptive process (Gabriel, 1958; Gershenson *et al.*, 1986).

It has been claimed that it is possible to see the limit between alveolar and basal bone in the form of 'baseo-alveolar sulci' (Inke, 1972), and that when bones are separated along these lines, the basal parts resemble the shape of a remodelled edentulous mandible and maxilla. Unfortunately, the communication shows only exaggerated diagrams of the sulci, which otherwise are difficult to see. Yet, the sulci may relate to positions of muscle insertions or to the limits of the attached gingiva, both of which have been taken as a functional-anatomical division between alveolar and basal bone (van der Klaauw,
1952; Moss, 1972; Kalk and de Baat, 1989). Of the two, the latter accounts better for the different levels of loss that are seen around the mouth; the former is complicated by the fact that the muscle attachments are not fixed landmarks, with clear changes having been noted for the mentalis and buccinator muscles as the ridge recedes (Lammie, 1956; Osborne, 1963).

Yet why some individuals lose more bone than others is unclear. Many factors, both local and systemic, have been proposed as having an influence on the post-extraction resorption of the mandible (Table). The potential functional, anatomical, physiological, inflammatory, and metabolic causes will be discussed in turn.

(2) Local Factors Influencing Mandibular Bone Loss

(2.1) Functional factors

Since the beginning of the century, many investigators have attributed the changes in the edentulous mandible to the changing function of the bone (Walkhoff, 1901; Lewin, 1913; Seipel, 1948; Ortman, 1962), as did John Hunter in the 18th century, but it is only recently that the relationship of mechanical function to bone architecture and the cellular responses involved are becoming better understood (Rubin and Lanyon, 1984; Skerry and Lanyon, 1993). Neufeld (1958) described the changes that occur in the remodeling of the edentulous mandible as mimicking the effects of 'disuse osteoporosis' (i.e., the changes seen in a bone following a reduction in mechanical stimulation), since some of the features, such as the increase in cortical porosity and the trabecular thinning, are common to both. Disuse osteoporosis, however, is usually accompanied by endosteal resorption with little in the way of change in the external diameter of the bone (Takahashi, 1987; Ruff and Hayes, 1988), and the resorption is self-limiting, ceasing when a new functional equilibrium has been established (Devlin and Ferguson, 1991). In contrast, a resorbing ridge shows bone apposition on the endosteal surface and a continuous overall decrease in size. In addition, in a study of mandibular structural changes with age, no relation between cortical porosity and the presence or absence of the teeth was found (Atkinson and Woodhead, 1968).

It is obvious that many functional changes occur upon the loss of the teeth, both in the source and the magnitude of the applied strains (Sobolik, 1960), as well as in the way they are transmitted to the bone (Ortman, 1962). Bite forces reduce considerably with a reduction in the number of teeth (Helkimo et al., 1976), and patients with dentures can apply only one-eighth to one-sixth of the force that is possible by patients with natural teeth (Sobolik, 1960; Ortman, 1962). Both physical and social factors may reduce customary bite force (Lockington and Bennett, 1994), and a reduction in the activity of the muscles of mastication may be seen within as few as four weeks after the insertion of new dentures, even in individuals experienced in wearing dentures (Raustia et al., 1996). The wearing of dentures has been cited as both the cause (de Van, 1935; Carlsson and Persson, 1967) and, in the means of preventing (Mauley and Stuart, 1937; de Aguiar et al., 1968) residual ridge resorption, although bone loss is observed whether dentures are provided or not (Campbell, 1960). Implants are known to help maintain ridge height, although they may exacerbate bone loss in the opposing jaw (Nemeth et al., 1998).

In the past, lateral shearing loads produced by poorly balanced dentures were thought to have the most detrimental effect upon the underlying residual ridge. Denture design was therefore modified to reduce lateral loading by using teeth with flatter cuspal inclines and by discouraging incision (de Van, 1935; Sobolik, 1960). However, studies have shown the style of denture teeth to have little effect upon subsequent resorption (Brehm and Abadi, 1980), and one five-year study (Winter et al., 1974) showed an effect opposite to that anticipated, with those in which more shearing loads would be expected showing less resorption. This paradox was attributed to the great variation that occurs between individuals (Atwood, 1979). However, it is known from studies of long bones that the applied loads that are the most osteogenetic are those that are applied at high strain rates, are of short duration, and are unusual in their distributions (Lanyon, 1992). These osteogenetic loading characteristics are possibly minimized in the flatter type of occlusal design.

The nature of the application of loading is of importance in the maintenance of bone mass. In 1958, an exercise appliance was described for the edentulous ridges of partially dentate patients (Applegate, 1958). The appliance consisted of a close-fitting, well-extended resin saddle that covered the edentulous areas. The patient placed his or her fingers upon the saddle and bit repeatedly on the fingers for a few minutes each day (the use of the fingers was advocated so that the patient would not bite too hard). After an initial decline which lasted a couple of weeks, it was shown that this procedure increased the radiographic density of the bone in the area, reaching a maximum at around 12 weeks of stimulation (Smith and Applegate, 1961). The data were not subjected to a statistical analysis, but a particularly favorable response was reported for young patients and for those who had experienced only a short period of edentulousness. The differences between younger and older individuals may be due to age changes in the quality of the overlying soft tissues, the bone, and the vasculature, as will be discussed. The changes in radiographic density observed may be due to bone deposition (either periosteally or endosteally) or to an increase in the soft-tissue thickness. However, the ini-
tial decline in radiographic density makes the former more likely, since it may reflect a period of increased osteoclastic activity prior to new bone formation.

In one longitudinal study (Tallgren, 1972), the only factor (of many studied) which was found to correlate with the magnitude of bone loss was how upright the mandibular ramus was with respect to the mandibular base. However, cross-sectional studies have failed to show an association between the size of the gonial angle and the extent to which the residual ridge was resorbed (Raustia and Salonen, 1997). Those individuals with an upright ramus, a more acute gonial angle, and a greater angle from the mandibular base to the condyle measured at the anterior midline (features more frequently associated with patients with a small lower face height) showed a significantly greater amount of resorption than long-faced individuals (Tallgren, 1972). Individuals with different face heights have musculatures of different shapes (Mosolov, 1972), and patients with a small lower face height are capable of generating significantly higher bite forces than are people with longer faces (Moller, 1966), and are more likely to have tooth wear if dentate (Crothers and Sandham, 1993). Masseter size may also have a local effect upon the cortical bone density of the mandible (Klemetti et al., 1994d), and changes in cortical density distribution are seen following tooth loss, possibly due to functional alterations (Klemetti et al., 1994b; Kingsmill and Boyde, 1998b).

Functional loads and the way they are applied play a great role in the reshaping of the mandible after tooth loss, and many factors other than the nature and pattern of loading may affect or interfere with the response of bone to functional strain. However, alterations in functional loading are unlikely to be the sole explanation for the reduction of residual ridges; otherwise, bone changes would not occur at the sites of insertion of the muscles of facial expression (Lammie, 1956). Such sites might be expected to undergo little reduction in function following tooth loss. Other factors that may have an influence upon resorption of the residual ridge or the perception and response to functional strain are discussed below.

(2.2) **ANATOMICAL FACTORS**

(2.2.1) **Bone size**

The original size of the mandible and the depth of the extraction sockets have been suggested to influence resorption (Atwood, 1979), yet neither factor has shown an association with the amount of subsequent resorption in longitudinal studies (Carlsson and Persson, 1967). Finite element analysis of differently shaped mandibles has shown that reduced strains are found along the lower borders of mandibles with increased corpus height (Korioth et al., 1992). If all other factors remained constant, this would imply that as the mandible becomes smaller in cross-section with continued ridge resorption, it must experience lower forces so that the added depth of bone is no longer required. However, with continued resorption of the residual ridge, the mandible has the capacity to become increasingly consolidated (Kingsmill and Boyde, 1998a) and more highly mineralized (Kingsmill and Boyde, 1998b), both of which would improve its resistance to bending.

(2.2.2) **Bone type**

Cortical and cancellous bone are thought to respond differently to local and systemic influences (de Van, 1935; Becks and Grimm, 1945; Baxter, 1987; Buchanan et al., 1988; Lanyon, 1992; Klemetti et al., 1993a). In humans, the number, arrangement, and distribution of trabeculae are highly variable in edentulous mandibles (Becks and Grimm, 1945; Parfitt, 1962; Ulm et al., 1997), and, following tooth loss, the ridge crest never develops a complete cortex (Neufeld, 1958; Atwood, 1963; Pudwill and Wentz, 1975) and may therefore be more prone to age-related or metabolic loss (Baxter, 1987). In addition, the concentration of insulin-like growth factor differs between cortical and cancellous bone, and this difference may affect the rate of turnover of these tissues (Canalis and Agnusdei, 1996). It has been proposed that the thin cortical bone and the relatively large amount of trabecular bone in the maxilla absorb bite impulses more efficiently than does mandibular bone (Klemetti and Vainio, 1994).

(2.2.3) **Bone site/origin**

Bone tissue at different locations in the body varies in its rate of remodeling (lee et al., 1991) as well as in its response to mechanical strain. Alveolar bone may initially develop independently from basal bone in close association with the tooth germs, and there is growing evidence that bone of “dermal” origin (loosely called membrane bone, including most of the bones of the head, plus the mandible) differs in several respects from bone of endoskeletal origin (or endochondral bone, which includes the majority of the post-cranial skeleton; Couly et al., 1993; Smith et al., 1994). For example, the calvaria shows a pattern of age-related bone loss different from that seen at the femoral neck (Karlsson et al., 1995), and experiences strains much lower than those required to maintain bone mass in long bones (Rubin and Lanyon, 1984; Hylander et al., 1991). The mandible itself, however, is subjected to strains more typical of the post-cranial skeleton (Throckmorton et al., 1992). Bone of dermal origin also differs from bone of endoskeletal origin in its behavior as a grafting material (Smith and Abramson, 1974; Zins and Whitaker, 1983; Kusiak et al., 1985; Moskalewski et al., 1988, 1991; Scott and Hightower, 1991; Scott et al., 1994; Sasano et al., 1995) and in the lev-
els of stored growth factors (Baylink et al., 1993). For example, cultured human mandibular osteoblasts produce more fibroblast growth factor and insulin-like growth factor, but less transforming growth factor beta, than human iliac cells (Kasperk et al., 1995). These differences have been attributed to the embryological origin of the bone cells (Recker, 1992).

(2.2.4) Bone composition

Alveolar bone is described as being less brittle than other bone, a feature which may be due to its higher glycosaminoglycan content (Waddington et al., 1989). Alterations in occlusal loading have been shown to alter the proteoglycan distribution and content in rat mandibles (Shore et al., 1996) which may be involved in the strain-memory responses of bone (Davidovitch, 1991) and hence the way it may respond to functional alterations. Not much, however, is known about the effect that this may have upon resorption, although the less highly mineralized a substrate, the more easily it may be resorbed (Jones et al., 1995). The alveolar crest has been found to have a significantly lower mineral content and fewer extrinsic (Sharpey's) fibers than the adjacent bone (Landini, 1991; Kingsmill and Boyd, 1998b), both of which may favor its removal.

Functional loading has been shown to have an effect upon the intrinsic and extrinsic fibers of the alveolus surrounding teeth subjected to periods of non-, hypo-, and hyperfunction (Short and Johnson, 1990). Both non- and hypofunctional groups show a reduction in the level of mineralization of the fibers, which become larger and sparser, compared with the fibers in bone around teeth with normal function. Bone collagen mass may be reduced by as much as 10% at sites of disuse (Akeson et al., 1987).

(2.2.5) Bone cells

Atwood (1979) suggested that the aging of bone cells may contribute to their defective function. However, he failed to mention osteocytes, which probably play an important role in mediating the response of bone to alterations in functional loading. In addition, of the three major bone cells, osteocytes are replaced only when the bone is remodeled, are well-known to exhibit age changes (Tonna, 1976), and may become mineralized (Frost, 1960). The mineralization of osteocytes is particularly evident in the elderly mandible (Kingsmill and Boyd, 1998b).

(2.3) Physiological factors

(2.3.1) Blood supply

Experiments undertaken on animals with continuously erupting teeth have shown that the blood supply to the alveolus is considerably greater than to other parts of the mandible (Indresano and Lundell, 1981). Caution has to be exercised in extrapolating this to the human situation, but in man, alveolar bone does seem to have a high metabolic activity (Waddington et al., 1989). It is less highly mineralized than basal bone (Kingsmill and Boyd, 1998b) and more porous (Atkinson and Woodhead, 1968). Changes in alveolar bone can be the first sign of a metabolic disturbance (Bays and Weinstein, 1982; Baxter, 1987), although this could be due to the fact that changes in the fine bony anatomy are easier to detect here than at other skeletal sites (Bender, 1997).

The pattern of the blood supply in the mandible is thought to change from one that is largely centrifugal in the young dentate individual, to one that is primarily centripetal, with the supply being increasingly dependent upon periosteal and submuscular sources by the time old age is reached (Castelli, 1963; Wallenius and Heyden, 1972; Bradley, 1975, 1981; Poiriot et al., 1986). This is likely to be of immense importance if the mucosa is used to support a denture (Weinmann and Sicher, 1955; Ortman, 1962), since prolonged pressure could occlude the fine periostealplexus of vessels. This could stimulate osteoclastic resorption by altering the local oxygen tension and reducing the pH (Arnett and Dempster, 1990). Pressures of long duration could be expected to have a greater effect, especially since the mucosa is visco-elastic (Picton and Wills, 1978) and is slow to recover in the elderly (Lytle, 1957; Kydd and Daly, 1982). An experimental study which used spring appliances to move rat caudal vertebrae through the surrounding soft tissues showed that bone resorption occurred wherever the tissues were compressed against the bone, and that bone formation occurred on the trailing edge, thereby demonstrating the influence that the status of the surrounding tissues can have on bone remodeling (Feik et al., 1990).

Clinical observation reveals that ridge resorption frequently matches the shape of an underextended denture, but otherwise the importance of pressure on the extent of resorption is less certain. One study of 34 patients (Carlsson and Persson, 1967) found that the daily period of denture wear is related to the rate of residual ridge resorption, with those patients wearing their dentures at night as well as during the daytime experiencing significantly more loss. However, subsequent studies have been unable to find any relationship between the extent of resorption and the frequency of denture use (Bergman et al., 1971; Harrison, 1972). The effects of pressure could account for the differences seen between the resorption of maxillary compared with mandibular bone, the former having nearly twice the area available for support (Woelfel et al., 1976). In a study of 55 edentulous subjects, a significant negative relationship has been found between the size of the projected denture foundation
area on the tentative plane of occlusion and the degree of alveolar ridge resorption in the anterior but not in the posterior part of the mandible (Suenga et al., 1997).

Bone turnover in basal parts of the mandible was thought to cut off existing Haversian systems, further compromising the alveolar blood supply (Atkinson and Hallsworth, 1983). It is now known that the Haversian canals may become occluded with mineralized tissue, particularly in the mandibular bone of elderly individuals (Kingsmill and Boyd, 1998b).

(2.3.2) Bone turnover
The regulation of bone turnover relies upon a complex interplay among osteoblasts, osteoclasts, osteocytes, and many other factors. Osteocytes are thought by some to be the cells responsible for detecting and initiating the cellular responses to changes in bone function, but a large number of osteocytes die following tooth extraction, resulting in many cell-free areas of bone in the initial stages of socket healing (Carlsson et al., 1967). These dead cells may account, in part, for the initial rapid phase of remodeling that is seen after tooth loss, since live osteocytes may release factors that inhibit osteoclastic activity (Maejima-Ikeda et al., 1997; Kingsmill et al., 1999).

In aging bone, viable osteocytes may become fewer to such an extent that the presence of 'over-aged' bone becomes an increasingly frequent finding in the mandible (Pudwill and Wentz, 1975; Kingsmill and Boyd, 1998b). As the turnover reduces, and packets of older bone become more prevalent, patent canalici become reduced in number and diameter, and there is a reduction in the number of osteocyte lacunae per unit volume of bone (Atkinson and Hallsworth, 1983; Mullender et al., 1996). Some areas become completely devoid of canalicular and cellular spaces, probably due to mineralization of the lacunae and their contents (Frost, 1960; Kingsmill and Boyd, 1998b) with, overall, basal bone showing fewer patent lacunae per unit volume than alveolar bone, albeit more evenly distributed (Atkinson and Hallsworth, 1983). This may or may not be of importance in conditions of normal usage, but it is likely that older bone will be less able to respond to changes in function if required. This could account for the younger patients' benefiting more from the exercise therapy prescribed by Smith and Applegate (1961) and could affect the rate of bone responses to implants (von Wowern et al., 1990).

(2.4) Inflammatory factors
Arachidonic acid, released as a result of tissue injury, may be metabolized by two main pathways (i.e., the cyclo-oxygenase pathway, and the lipoxygenase pathway), the resulting products of which (prostaglandins and leukotrienes) have a role in bone resorption. Leukotrienes are involved in osteoclast precursor recruitment and/or differentiation (Franchi-Miller and Saffar, 1995), while prostaglandins are concerned with differentiation and activation. Inhibition of the metabolic pathways with indomethacin or BWA4C reduces post-extraction bone resorption in animal models (Franchi-Miller and Saffar, 1995). However, such effects are probably transient, since, upon cessation of indomethacin administration, resorption is rapidly and completely restored (Leroux and Saffar, 1993). Whether long-term anti-inflammatory medication may affect post-extraction bone loss in humans is unknown.

(2.4.1) Pre-existing periodontal health
In the Western world, teeth are most commonly lost as a direct or indirect (through root caries) result of chronic inflammatory periodontal disease. One of the features of plaque-associated periodontitis is a reduction in the bony support around the teeth due to influences of microbial factors acting either directly or indirectly via the host's inflammatory responses (Jeffcoat, 1993; Henderson and Wilson, 1996). Some have suggested that microbial endotoxins from denture plaque and residual bone-resorbing factors may cause localized resorption of the alveolar ridge in the same way (Humble, 1936; Atwood, 1979; Penhall, 1980). This has been termed 'the local biochemical inheritance from the dentate period' (Kleemetti, 1996). Yet, in one longitudinal study, no association was found between the pre-extraction bony support of the teeth and the amount of subsequent bone loss (Carlsson and Persson, 1967). The latter study based its periodontal assessment on measurements made from lateral cephalograms, which cannot be as reliable an index of periodontal status as the more conventional technique of periodontal probing. On the other hand, the microbial and host interactions that are so crucial to the development of periodontal disease hinge around the fact that the junctional epithelium which links the gingiva with the teeth is highly permeable, a pathway which disappears upon tooth removal.

(2.4.2) Trauma
The amount of bone damage that occurs during the removal of the teeth may affect the subsequent remodeling of the bone (Sobolik, 1960). Mucosal inflammation caused by pressure areas under a denture could cause bone resorption via the generation of arachidonic acid metabolites or interleukins (Hill et al., 1998). Inflammation is more likely to occur in elderly patients with thin, friable, and tender soft tissues (Massler, 1956), and any soreness will probably reduce the mechanical stimulation of the ridge. In one histological study, all individuals studied showed signs of chronic inflammation of the denture-bearing mucosa (Pudwill and Wentz, 1975).
(3) Systemic Factors Influencing Mandibular Bone Loss

(3.1) Nutritional Factors and Calcium Metabolism

Diet can have both a local and a systemic effect on the mandible. In rats, the consistency of the food has been shown to alter mandibular structure, and low-calcium diets result in resorption of mandibular bone (Sones et al., 1986). Orthodontic tooth movement has been found to be faster in beagle dogs that have had parathyroid hormone levels increased by decreasing the dietary calcium:phosphate ratio, compared with controls. The test group also showed less than one-quarter of the amount of trabecular bone (Midgett et al., 1981). Several human studies have suggested that a secondary hyperparathyroidism caused by a lack of dietary calcium may affect mandibular structure (Syriinen and Lampainen, 1983; Habets et al., 1988a,b), and dietary calcium and vitamin supplements may help to maintain ridge size and mass at one year post-extraction (Wical and Swoope, 1974b; Kribbs, 1992). However, the long-term effect is less certain: A retrospective analysis of calcium intake found a weak correlation with mandibular bone density (r = 0.28), but no correlation with mandibular bone mass (Kribbs et al., 1989).

In a study of dental panoramic radiographs from 18 patients with secondary hyperparathyroidism, no correlation was found among serum calcium, inorganic phosphate, alkaline phosphatase, or parathyroid hormone with the radiographic parameters tested, although the patients were judged to have fewer trabeculae compared with controls (Syriinen and Lampainen, 1983). With such a small number of patients and the large subjective bias that panoramic radiographic assessment has (Klemetti et al., 1994c), no firm conclusions can be drawn, yet it is interesting to note that no association has been found between primary hyperparathyroidism and residual ridge reduction (Lekkas, 1989).

In patients whose nutrition is more severely affected, changes in mandibular bone may become apparent. Forty-seven percent of patients with short bowel syndrome receiving parenteral nutrition (for a period ranging from one to 180 months) showed evidence of mandibular osteoporosis (von Wowern et al., 1996) (although this may partly have arisen through functional changes), and patients with renal osteodystrophy showed thinning of the radiographic cortex at the angle of the mandible, the degree of thinning being greatest in those five individuals with the greatest histological severity of disease determined from iliac crest biopsies (Bras et al., 1982b). Cortical thickness at the same site is also reduced in patients with thyroid disease and asthma (Xie et al., 1997).

(3.2) Age

Clinically, it is known that age changes in the mandible may increase the difficulty of orthodontic tooth movement, extractions, or other surgery (Grant and Bernick, 1972). This is probably largely due to an increase in the mineralization density of mandibular bone up until the fifth decade (Kingsmill and Boyde, 1998b), although there may be a concurrent increase in the amount of compact cortical bone (Humle, 1936; Kingsmill and Boyde, 1998a). Radiographic and anatomical cortical thickness measurements have shown both an increase (Benson et al., 1991) and decrease with aging (von Wowern and Stolte, 1979; Bras et al., 1982a; Kribbs, 1990). The cortex also becomes increasingly porous with age (Atkinson and Woodhead, 1968), but porosity tends to decrease initially in early adulthood prior to the increase in older age (Manson and Lucas, 1962). In other sites of the skeleton, bone may also increase in its level of mineralization with age (Boyde et al., 1995), but this is usually accompanied by a loss in bone quantity rather than the gain that may occur in the mandible.

The aging of osteocytes has already been mentioned, and it is possible that their death could slow the rate of response during orthodontic tooth movement by failing to aid in the response to strain. In addition, the mineralization of osteocytes, together with the increasing levels of mineralization of the surrounding bone with age, might retard the resorptive phase of bone turnover. This will be exacerbated in areas where access to the tissue by osteoclasts is hampered by the occlusion of the Haversian canals (Kingsmill and Boyde, 1998b). Levels of insulin-like growth factor in bone are also known to change with age (Hammerman, 1987; Nicolas et al., 1994), and may affect the rate of bone turnover.

(3.3) Gender

The sex of an individual may have some influence on mandibular bone. In edentulous individuals, mandibular height is greater in males than in females, although there is no difference while the teeth are still present (Engstrom et al., 1985; Ortman et al., 1989). This view is supported by the observation that alveolar crestal resorption lacunae are deeper in women than in men (Devlin et al., 1994). However, males have been shown to exhibit more bone loss than females in the posterior mandible, a fact which has been attributed to the males having a greater bite force (Winter et al., 1974); others have found no differences in bone loss between the sexes (Bergman et al., 1971). The porosity of the cortical bone in the femoral neck correlates only to mandibular cortical porosity in females (Dyer and Ball, 1980), and the bone mineral content of the mandible in young dentate women is lower than in young dentate men (von...
Wood, 1988). A study examining cortical thickness from birth up to 69 years reported a reduction after the age of 60 years in females, but no gender difference until this age (Bras et al., 1982a). However, there was no statistical analysis of the data, although another study showed women to have a thinner cortex at the angle of the mandible than did men (Xie et al., 1997). The panoramic mandibular index (PMI, a measure of lower-border cortical thickness in the region of the mental foramen, divided by the distance from inferior border to lower margin of the mental foramen) differs between men and women over 70 years of age (Benson et al., 1991), with males having a greater mean PMI indicative of more cortical bone. Another study showed an increase in mandibular bone density with age in males only; however, a larger age range was represented than for the women (Kingsmill and Boyd, 1998a). No gender difference was found in the level of mineralization of mandibular bone at any age (Kingsmill and Boyd, 1998b).

### 3.4 Relationship with osteoporosis

For over half a century, it has been recognized that generalized constitutional and/or systemic factors may influence mandibular bone (Becks and Grimm, 1945; Massler, 1956; Sobolik, 1960; Ottman, 1962), and this topic has recently been receiving renewed interest. One conference summary devoted to the subject of 'oral' and 'nonoral' bone loss stated that still little is known of the nature of the relationship between mandibular and post-cranial bone (Redford and McGowan, 1993). This may be for two main reasons: (a) As has been discussed, mandibular residual ridge resorption is multifactorial in nature, which severely complicates the construction and interpretation of studies; and (b) clinical studies have to rely on indirect methods of investigating the status of the bone, often giving little regard to the underlying anatomical and structural features of mandibular bone and the changes that occur with aging.

Osteoporosis is the most common systemic bone disease and is characterized by a loss in bone mass with structural changes predisposing to fractures, particularly of the wrist, the lumbar spine, and the femoral neck (Christiansen, 1993). In the dental literature, the term has been used more freely. It has variously been used to describe: a local increase in the diameter of the central canals of cortical Haversian systems (Atkinson and Woodhead, 1968); a reduction in the thickness of the mandibular cortex (Bras et al., 1982a); a sparseness of trabeculae as seen on radiographic examination (Horner and Devlin, 1992); and a bone mineral content value more than two standard deviations below the mean bone mineral content of young normal adults of the same sex (von Wowern et al., 1996) or of one standard deviation below the mean value for the sample (Horner and Devlin, 1998). Spontaneous fractures of the mandible are not a feature of generalized osteoporosis, so there is no convenient endpoint that confirms the diagnosis of osteoporosis in the mandible as there is for the post-cranial skeleton. In addition, since the pathological effect of skeletal osteoporosis in the mandible is unknown, studies have used a range of mandibular variables, measured in a number of different ways, as indicative of systemic disease. These include: altered trabecular patterns, cortical porosity, reduced cortical thickness, bone density, the height of the residual ridge, the number of teeth, and the status of the periodontal tissues.

It should be remembered that the mandible differs in several ways from bones commonly affected by osteoporosis: It has teeth and undergoes a change in shape after their loss; it consists largely of cortical bone with a very variable trabecular element; it may get more compact with age rather than less compact (a very resorbed mandible may consist almost entirely of cortical bone and therefore be extremely dense: Kingsmill and Boyd, 1998a); it is much more highly mineralized (Kingsmill and Boyd, 1998b); it is of a different embryological origin; and sex differences, if genuine, generally become evident at a later age than they do for other bones (Bras et al., 1982a; Benson et al., 1991). Further, techniques such as dual-energy x-ray absorptiometry are one-third as sensitive for the mandible as for the lumbar spine—it would take around two years for the reliable detection of bone loss in the mandible at the rate that it usually occurs post-menopausally in the rest of the skeleton (Corten et al., 1993).

#### 3.4.1 Trabecular pattern

At first sight, a study of the trabecular architecture of the mandible would seem sensible, since, in other bones, such as the lumbar vertebrae, this is where most alteration is seen in osteoporotic states (Jayasinghe et al., 1994). However, the mandible shows great variation in its trabecular architecture between individuals, and varies markedly with aging and upon tooth loss (Humble, 1936; Becks and Grimm, 1945; Parfitt, 1962). Also, the trabecular contribution to structural strength is thought to be negligible in the mandible (Biknevicius and Ruff, 1992), which causes one to question the function of trabeculae in this site. It has been suggested that their only real function in the mandible is as a calcium reserve (Roberts, 1993), but they may help to hold the narrow elements in place, a role that may diminish as the marrow becomes increasingly fibrous with age (Klingsberg and Butcher, 1960). Moreover, accurate and reliable radiographic interpretation of trabecular architecture is not possible, since each radiograph can give only a twodimensional representation of the complex three-dimensional structure, and usually no allowance is made either
for the varying bone and cortical thicknesses, which will influence the medullary volume available for the trabeculae (de Aguiar et al., 1968), or for the inhomogeneity of the tissue. Nevertheless, radiography seems to be the most frequent technique used for mandibular trabecular assessment, sometimes being combined with advanced image processing techniques (Läger et al., 1990; Ruttimann et al., 1992; Law et al., 1996; Southard and Southard, 1996). One study (Horner and Devlin, 1992) has claimed that bone density measured from panoramic radiographs could be used as an indication of mandibular osteoporosis. However, not only was the allocation of the radiographs into each category (osteoporotic or not) entirely subjective, but also the so-called density measurements were based upon similar criteria, so it is not surprising that a correlation was found between the two. It has since been shown that panoramic radiographs cannot be used for the individual assessment of skeletal osteoporosis (Klemetti et al., 1994c), which must, at least partly, be due to the hard- and soft-tissue shadows from the out-of-focus layers in this type of radiograph.

Histological studies in small-animal models have shown that aging, ovariectomy, and disuse all reduce the mandibular total bone area fraction (trabecular and cortical bone) (Elovic et al., 1995), and that the systemic administration of estrogen (Stahl et al., 1950), bisphosphonates (Yaffe et al., 1995), or intermittent parathyroid hormone (Miller et al., 1997) causes increased mandibular bone deposition. However, even animals show an increased variation in tissue morphology with age (Klingsberg and Butcher, 1960), and extreme caution should be exercised in drawing conclusions from human mandibular trabecular architecture in the absence of longitudinal data.

(3.4.2) Cortical thickness/quality

Cortical thickness measurements have been used to assess the effect of osteoporosis in many bones, but even proponents of this method say that it should not be applied to the mandible (Garn et al., 1966). Nevertheless, several workers have shown an age-related decrease in mandibular cortical thickness (von Wovern and Stoltze, 1979; Bras et al., 1982a; Kribbs, 1990), but no association with skeletal bone mass as measured by dual-photon absorptiometry of the lumbar spine or femoral neck (Mohajery and Brooks, 1992). In a study of over 350 patients, Klemetti et al. (1994c) found that there is an association between the height and radiographic quality of the cortex with various degrees of severity of osteoporosis, but that the sensitivity was so low that no indication of osteoporosis risk could be obtained on an individual basis. A further study (Horner and Devlin, 1998) using the same index of cortical quality found a correlation \( r = -0.50 \) with the bone mineral "density" of the mandible (as determined by dual-energy x-ray absorptiometry in which both halves of the mandibular body are superimposed), but there was a limitation in interobserver repeatability.

One study revealed that about half of the patients with severe mandibular atrophy have a thin cortex at gonion (Bras et al., 1983). This was given as evidence of metabolic bone loss, but no measure of metabolic bone loss was made, and so the thinning could equally be due to functional or other changes. In a study of 85 women aged from 50 to 84 years, all having vertebral fractures from osteoporosis, the range in gonial cortical thickness was from 0.3 to 1.6 mm, which is similar to that seen in normal women over 60 years of age (from 0.2 to 1.2 mm; Bras et al., 1982b). Moreover, in some panoramic radiographs, it is not possible to see a distinct cortical layer at gonion (Law et al., 1996), and the radiographic appearance is likely to be affected by the exposure, processing factors, the thickness of the overlying tissues, and the size of the bone. Cortical thickness, especially at gonion, is difficult to measure with precision, even in cleaned dissected mandibles lying directly on a film (Kingsmill, 1996). Even the use of an index of cortical thickness in the region of the mental foramen, where the cortex is more readily identifiable and thicker, failed to find a difference between post-menopausal white women with mild/moderate osteoporosis and a matched control group without osteoporosis (Watson et al., 1995).

(3.4.3) Bone density

The term 'bone density' has been used to refer to a number of aspects of bone quality and quantity, other than just the mass per unit volume of the tissue. The Oxford English Dictionary (Little et al., 1959) defines density as: 'the quality or condition of being dense; thickness; closeness of consistence ... the degree of consistence of a substance, measured by the quantity of matter in a unit of bulk' and 'dense' as: 'having its constituent particles closely compacted together'. Therefore, just as the term 'bone' can be used to describe the organ or the tissue, so can 'bone density' refer to the amount of tissue present in a given volume, or to its constitution at a microstructural level. The use of 'bone mineral content', 'bone mass', and 'bone density' may superficially help to clarify the situation, but when one considers the techniques used for their determination, the division between them is not as clear. For example, two-dimensional radiographic techniques may be affected by the degree of mineralization of the bone tissue (microdensity), the porosity of the tissue, the cortical thickness, the thickness of the part of the bone being examined, and the overlying soft-/hard-tissue shadows (as well as by exposure and processing factors); even quantitative computed tomography is likely to be affected by the first

10(3) 384-404 (1999)
two. The dual energies of x-rays or photons used in absorptiometry techniques eliminate the influence of soft tissues, but do not give true density values (measurements are given in g/mm²), and therefore the depth of bone in the plane being examined may become an important consideration. Both halves of the mandible are often superimposed, which will increase the likelihood of error arising from the patient not being accurately positioned (Horner and Devlin, 1998).

The density of the mandible, as measured by pyknometry, microdensitometry, quantitative computed tomography, and absorptiometry, has been found to correlate with that of other bones (Henrikson and Wallenius, 1974; Rosenquist et al., 1978; Dyer and Ball, 1980; Mercier and Inoue, 1981; Kribbs et al., 1983, 1989; von Wowern, 1988; Kribbs, 1990, 1992; von Wowern and Kollerup, 1992; Klemetti et al., 1993b), but not if the trabecular portion is considered in isolation (Klemetti et al., 1993a; von Wowern and Stoltze, 1979). An uncritical view of the literature, however thorough, may therefore lead one to conclude that osteoporosis could have a significant influence on mandibular bone (Hildebolt, 1997). However, like is often not compared with like: Either there is a problem with the study and control samples—e.g., edentate individuals with severe resorption may have their skeletons compared with those of dentate individuals, rather than those of less severely resorbed edentate individuals (Rosenquist et al., 1978)—or one technique of bone density assessment is used for the mandible, while another technique is applied to the other bones (Kribbs et al., 1989; Klemetti et al., 1993b,c). When these factors are controlled for, and one considers the local levels of mineralization at a resolution in the region of a few cubic microns, no correlation is found between the microdensity of mandibular bone in elderly individuals and that of the iliac crest, the femoral neck, or the fourth lumbar vertebra. However, there is a correlation between the microdensity of mandibular bone and the bone of the cranial vault, as there is among the postcranial bones (Kingsmill and Boyde, 1999).

The relationship between density and quality is less well-defined for the mandible than it is for the rest of the skeleton. Treatment modalities for osteoporosis have traditionally aimed at increasing bone density, but this does not always result in the anticipated reduction in fracture incidence (Riggs et al., 1982; Burckhardt and Burnand, 1993). Bone quality is used to describe the material, architectural, and mechanical characteristics of the tissue (Sherman and Hadley, 1993) and must be considered together with bone density. It is only since the recent upsurge in the use of dental osseointegrated implants that attempts have been made to define mandibular bone quality. The most frequently used classification for the mandible is a descriptive one based upon the intra-operative (or radiographic) arrangement of the bone, in which four types are detailed (Lekholm and Zarb, 1985): quality 1—nearly all compact bone; quality 2—thick layer of compact bone surrounding dense trabecular bone; quality 3—thin layer of compact bone surrounding dense trabecular bone; and quality 4—thin layer of cortical bone with low-density trabecular bone. Qualities 2 and 3 are thought to be the best types for implantation, and failure of osseointegration is most likely in quality 4 bone (Bass and Triplett, 1991). However, even this relatively simple classification may not show a very strong relation to bone mass as determined by dual-energy x-ray absorptiometry (Horner and Devlin, 1998).

One can understand why this area of research may have caused difficulty before there was an agreed consensus as to what constitutes good quality bone, since the effects of disease and the aims of treatment could not be quantified. One example is that some workers thought that a radiographically dense appearance of alveolar bone was the most desirable (Sobolik, 1960), while others argued that less dense bone was to be preferred because it was more 'organic' and hence more conducive to a normal rate of bone maintenance, since its turnover would be facilitated (Smith and Applegate, 1961). Even so, the present classification is more mechanical than biological, with no allowance being made for the cellular status, the blood supply, the age of the individual, or the resilience of the bone tissue.

Mandibular bone has been said to be osteoporotic when the bone mineral content values are more than two standard deviations below the mean bone mineral content of young normal adults of the same sex (as has been used for other sites in the skeleton; Nordin, 1987), but based on this definition, 83% of an osteoporotic group and 64% of a normal group had an osteoporotic mandible (von Wowern et al., 1994). The bone mineral content of the mandible has been found to correlate with the bone mineral content of the forearm (von Wowern et al., 1988) but not with the mineral content of the lumbar spine (von Wowern et al., 1996). This was thought to be due to the relative proportions of cortical bone at these sites, but correlations with the lumbar region may be complicated by the presence of osteophytes and other degenerative age changes (Klemetti and Vainio, 1993).

One study that attempted to correlate total body calcium with mandibular bone status found that, in an osteoporotic population, mandibular density (determined by taking into account the thickness of the mandible from occlusal radiographs and impressions) correlated less well with total body calcium than did mandibular mass (r = 0.31 cf. 0.54; Kribbs et al., 1989). This illustrates that the width of the mandibular body could be a significant factor to be taken into account in
the assessment of mandibular bone, and, as alluded to above, lateral projections of the mandible will not provide information upon true density unless it is. Therefore, any studies of mandibular density which do not take bone thickness into account may just be showing a relationship between the thickness of the mandibular body and the variable tested at other sites. It does not seem unreasonable that an individual with a high total body calcium will have a thicker mandibular body than an individual with a low total body calcium, particularly since a risk factor for generalized osteoporosis is being small and thin. Besides, physical activity has been shown to have a greater influence upon bone size than upon bone density (Brahm et al., 1998). Therefore, the individuals most at risk of developing osteoporosis may also have thinner, smaller mandibles. For example, a study of the bone mineral content in the lower border of atrophic mandibles found a lower level in females than in males (Ulm et al., 1994), but there may have been differences in the mandibular thicknesses between the sexes, especially since males generally have larger bones than females. Another study of nine patients, of unstated age and sex, showed that individuals with the lowest alveolar bone mineral content (determined from bitewing radiographs with no standard) showed the lowest dual-energy x-ray absorptiometry values for the forearm, the femur, and the lumbar spine (Hildebolt et al., 1993). Again, these findings could be attributed to variations in mandibular thickness and the thickness of the overlying soft tissues. A correlation has also been found between mandibular density in the molar region and bone mineral content in post-menopausal women (but not pre-menopausal women), but again the same caveats would apply (Engel et al., 1994). A study using dual-energy x-ray absorptiometry of the mandible has also shown correlations between mandibular bone mineral content and that of all other skeletal sites, although there was a smaller correlation with femoral neck than with spine and forearm (Horner et al., 1996).

The important relationship between muscle strength and bone mass and the role of estrogen have recently been stressed (Schiessl et al., 1998). Not only do masseter size and strength relate to the local mineralization densities in the buccal and lingual cortices distal to the mental foramen, but also individuals with weaker muscles have significantly lower bone mineral density values in their femoral necks and lumbar spine than do those with stronger, more palpable masseters. This trend was the same whether the individuals were partially dentate or edentulous (Klemetti et al., 1994d). Mandibular bone mineral density is usually assessed in the basal parts of the mandible, partly because the alveolar portion may be absent, and may therefore be influenced by muscular activity (Klemetti, 1996). Whole-body bone mass has been shown to correlate well with whole-body muscle strength (Schiessl et al., 1998).

One recent longitudinal study has reported that the radiographic density of the mandible increases in women taking hormone replacement therapy (Iacobs et al., 1996). This led to the conclusion that hormone replacement may have a positive influence on mandibular bone; however, no controls were used in the study.

(3.4.4) Periodontal disease

Several workers have found a weak, sometimes indirect, correlation between skeletal bone status and periodontal disease (Phillips and Ashley, 1973; Manson, 1976; von Wowern et al., 1994). For example, patients who had suffered an osteoporotic fracture were found to have 0.8 mm more attachment loss per site (von Wowern et al., 1994), a small difference to be detected clinically. This increased loss of attachment in osteoporotics may reflect a few teeth having much deeper pockets, rather than an overall increase. However, teeth with a large amount of attachment loss are more likely to be retained in patients with higher skeletal bone densities than in those with lower-density skeletons (Kribbs et al., 1989; Klemetti et al., 1994a). Some other studies have found a relationship between tooth number and skeletal bone density as measured at the radius (Krall and Dawson-Hughes, 1993; Taguchi et al., 1995) or other sites (Kribbs, 1990), although attempts to use tooth number as a predictor for the existence of systemic osteoporosis fall woefully short of being useful (Taguchi et al., 1995).

The bone mineral content of the lumbar spine has been found to be lower in edentulous than in dentate subjects; however, no difference was found after age and menopausal status were corrected for (Elders et al., 1992), and it was concluded that the relationship between periodontitis and skeletal bone mass was not of great importance.

(3.4.5) Ridge height/height of the mandibular body

The majority of studies have found no relationship between the height of the mandible and the skeletal parameters tested, even when a good correlation of the bone density between the sites exists (Ward et al., 1977; Dyer and Ball, 1980; Kribbs et al., 1983; Ortman et al., 1989; von Wowern and Kollerup, 1992; Klemetti et al., 1993a; Taguchi et al., 1995). This is not surprising when one considers that ridge height is not related to the bone mineral density of the mandible (Klemetti and Vainio, 1993), let alone the bone mineral density of other bones. However, Habets and co-workers (1988a,b) found that nearly all iliac crest biopsies from patients with severely resorbed mandibles showed disturbances in mineralization, and that over half of the individuals had a secondary hyperfunction of the parathyroid glands. No information was given on the time of year of examina-
tion, which can affect the incidence of mineral disturbances seen histologically in femoral and mandibular bone (Aaron et al., 1974; Stutzmann et al., 1981).

Total body calcium has been positively correlated with the height of the edentulous mandibular body (Kribbs et al., 1989), but in this study, all patients were osteoporotic. A study of 10 patients with advanced mandibular resorption showed that the patients had low bone densities in the radius as measured by absorptiometry (Bays and Weinstein, 1982). Hirai et al. (1993) also showed the height of the residual ridge to be negatively correlated with the degree of severity of osteoporosis (Hirai et al., 1993). The height of the edentulous alveolar ridge has also been found to be related to the bone mineral density of the femur (Klemetti and Vainio, 1993).

The reasons that most workers have found no correlation between ridge height and skeletal bone status and that only a few have could be due to several factors. Habets and co-workers (1988a,b) and Bays and Weinstein (1982) selected their patients on account of their severely resorbed mandibular ridges. This might pre-select the patients who are more likely to have systemic disease, be more frail than a random selection of edentulous patients, or have poor mandibular function and diet.

The greatest problem in this area, however, stems from the use of the 'fact' (given by Wical and Swoope, 1974a) that the total height of the mandible is 2.9 times greater than the distance from the lower border of the mandible to the mental foramen. It is acceptable for such a ratio to be used in longitudinal studies (Packota et al., 1988), where differences according to size are eliminated, but some have relied upon this ratio to calculate the pre-extraction height of the mandible, and hence to calculate the loss that has subsequently occurred (Wical and Swoope, 1974b; Ward et al., 1977; Taguchi et al., 1995). Such studies tend to find no association between mandibular height and the skeletal variable tested. In the original study (Wical and Swoope, 1974a), the individuals used for calculation of the ratio were selected only if certain criteria were met, such as having a full complement of teeth in a good arrangement, and a clearly visible mental foramen. No mention is made of the age range of the individuals who did fulfill the requirements, and so it is not known whether this ratio can be applied reliably to the aging population, which may experience continued apposition (Whittaker et al., 1990) or loss (Enlow et al., 1976; Ulm, 1989; Xie et al., 1996) on the lower border of the mandible throughout life. If this is true, one would anticipate the ratio of alveolar to basal bone to change with age, even without any changes occurring at the alveolar crest. Another important point is that great variability has been reported in the position of the mental foramen (Tebo and Telford, 1950), which may be located between 9.0 mm and 18.5 mm from the lower border of the mandible. This would imply that the original height of the mandible would range from 26.1 mm to 33.7 mm (a height that must rarely be attained by a human). Moreover, basal bone height has been shown to correlate with alveolar bone height (whether dentate or edentate; Kingsmill and Boyd, 1998a), and this would further affect the reliability of the ratio. The ratio was further tested by Bairam and Miller (1994), but they fail to state the standard deviation and the range found in the individuals tested.

(4) Conclusions

This article has reviewed some factors affecting post-extraction remodeling of the mandible. Many factors, both local and systemic, are likely to play a role in the structure and turnover of the mandible as they do for bone in general. However, studies correlating mandibular variables (such as ridge height, cortical thickness, or bone density) with systemic bone status have come to conflicting conclusions. This is largely because the multifactorial nature of mandibular residual ridge resorption and the variety of mandibular variables which may have to be taken into account have made well-controlled, relevant studies difficult to engineer. Comparable factors are not always considered: Either there is a problem with the study and control samples—for example, individuals with severely resorbed mandibles may have their skeletal status compared with dentate individuals (rather than with individuals with less severely resorbed mandibles)—or one technique of bone assessment is used for the mandible, while another technique is applied to the other bones. A cursory view of the literature may indicate that "density" measures of the mandible have shown a correlation with the density of other skeletal sites. However, the majority of studies have relied upon indirect measures of bone status (e.g., radiography), and the correlations seen may be partly accounted for by variations in bone thickness which are often not considered. On the other hand, direct measures of true bone density (including both the amount of bone per unit volume as well as the level of mineralization of the constituent tissue) have shown no correlation between mandibular and post-cranial sites. Likewise, with regard to measures of residual mandibular height, there seems to be no direct dependence upon the status of other sites in the skeleton.

Importantly, it can be concluded that severe residual ridge resorption may occur even when the bone status in the rest of the skeleton is good and vice versa (Klemetti and Vainio, 1993; Kingsmill, 1998a). Therefore, local functional factors may be of great significance and are particularly important because they may be easier to control than systemic factors. It is now necessary to determine how they can be modified and applied to help maintain ridge height and quality in our aging, edentulous population.
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