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TECHNICAL NOTE

The morphological and clinical relevance of mandibular and maxillary bone structures for implantation

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Tooth loss, which interrupts the biocybernetic feedback circuit of the masticatory system, changes the structures of the jaw bone: such changes are termed "inactivity atrophy". The mandible is subject to vertical atrophy and the maxilla is primarily subject to horizontal atrophy. The mandible possesses more compact bone, the maxilla more spongy; the resorption directions also differ (mandible: towards the oral aspect; maxilla: towards the vestibular). An implant helps to restore the biocybernetic feedback system. The amount of available bone, bone structure, and topographic conditions are crucial factors influencing implant success. Osseointegration is performed at an early stage (which includes bleeding, granulation tissue, foreign-body recognition, interactions) and at a late stage (so-called osseous bridging, development of fibrous and lamellar bone).

Key words: inactivity atrophy, maxilla, mandible, endosteal implant

INTRODUCTION

Given strict indications, careful operative technique and exact prosthetic restoration, implantology today is considered an established treatment option for partially dentate and completely edentulous individuals [1]. Many problems of endosteal implantation for the restoration of masticatory function can be regarded as largely solved. However, the orofacial system must always be taken as an entirety which represents a biocybernetic functional cycle involving the CNS, bones, musculature, temporomandibular joint, teeth, and periodontium. Alterations to these factors lead to craniomandibular imbalances. Implants are a means of restoring balanced function to this feedback system. Thus the various functions of the oral cavity are also restored. These include:

— nutrient uptake and processing;
— function as a sensory organ;
— verbal and non-verbal expression of all kinds.

Implantation areas

The areas of application for implantation in the oral cavity are:

— edentulous maxilla and mandible;
— partially edentulous alveolar arch;
— single-tooth prosthesis;
— complicated, reduced bone tissue;
— aesthetic components.

Of primary concern is the maintenance of bone structures, the support of the prosthesis, the stabilisation of the remaining dentition and psychosocial stability.

No other bony structure in the organism is as subject to functional alterations as the mandible and maxilla. This fact is especially obvious during the processes of ageing as seen in the development of newborn to geriatric skulls (Fig. 1). The edentulous jaw, the main application area for implants in the oral cavity, has anatomical/topographical consequences:
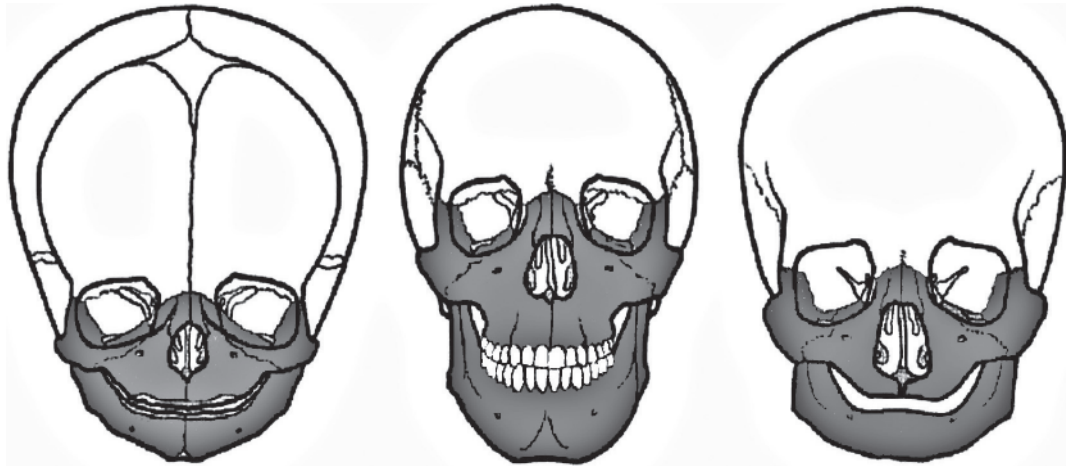
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Figure 1. Ageing of the human skull.

— the *cavum oris proprium* and the vestibule form a common space;
— the tongue volume increases;
— the position of the musculature and therefore also the tonus change.

In addition, structural changes of the bone are observed. These include:
— thinning of the trabecula and corticalis;
— enlargement of the medullary cavities;
— osteoporotic alterations (influence of diet).

These processes lead to secondary changes such as:
— loss of soft-tissue support;
— positional alterations of the joint capsules in the temporomandibular joint (dislocation of the *discus articularis*!).

Causes of jaw atrophy

In cases of inactivity atrophy the loading stimuli to the bone are lacking. Inactivity atrophy is a complex of atrophic and resorptive bone alterations. The resorption of the (edentulous) alveolar ridge is a multi-factorial process. It cannot solely be attributed to the changed loading situation. The factors involved can be classified as follows:

Mechanical factors:
— functional factors;
— prosthetic factors;
— surgical factors;

Biological and metabolic factors:
— age;
— gender;
— alimentary factors;
— hormone status;
— vitamin status;
— mineral deficits;

— medications;
— anaemia;
— blood pressure;

Inflammatory causes:
— periodontal disease;
— local inflammatory processes;

Processes of jaw atrophy

Mandible. The degeneration is primarily a vertical atrophication. The alveolar process is smaller than the mandibular body, which is the constant part (Fig. 2). The geriatric mandible juts more strongly to the anterior. Vertical bone loss is greater here than in the maxilla [our unpublished data] (Table 1). In females these changes are even more pronounced.

The mandibular body is wider than the maxillary body. This difference is compensated for by the slant of the alveolar processes. Thus the alveolar process of the mandible is tilted to the oral and that of the maxilla to the vestibular aspect.
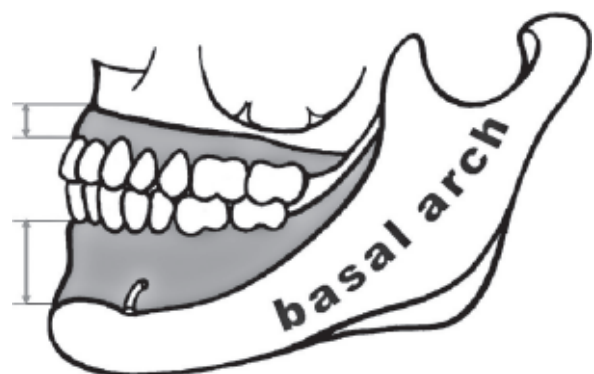

Figure 2. Vertical atrophy of the mandibula.

Table 1. Vertical bone reduction after tooth loss

	Maxilla	Mandibula
0–3 years	1.5 mm	3 mm
Renewal years (average per year)	0.1 mm	0.4 mm

Aside from the mandibular body, which can also exhibit degeneration during ageing, there are structures which are not subject to atrophic alterations [4]: the *linea mylohyoidea*, *linea obliqua*, *spina mentalis*, *tori mandibulares* and *trigona retromolaria*.

Both the mandibular body and the individual alveolar walls are trajectorially oriented.

The atrophy of the alveolar process also leads to an alteration in the gonial angle. In newborns the gonial angle is 150°, and in adults 120°. In old age the angle approaches the values present in childhood and can even attain 160° or more. This alteration of the angle also affects the *trigonum retromolare*, which "sinks" into the atrophied bone, thus moving into a close topographic relationship to the *pars obliqua* of the mandibular canal. Therefore, the *trigonum retromolare* in atrophied bone cannot be used as a site for an implant abutment [3, 4].

Danger zones for implantation in the mandible

A high position of the mandibular canal, particularly in close proximity to the alveoli in the molar area. The canal contains the A./V. *alveolaris inferior* and the N. *alveolaris inferior*; it runs downward in the *ramus mandibulae* and reaches the spongiosa of the mandibular body midway between the lingual and buccal compacta. The canal is surrounded by compacta and is tube-shaped. The distance of the horizontal sections from the floor of the alveoli is 3 to 4 mm in the area of the third molars and approximately 8 mm around the first molars [2, 4]. *Canaliculi alveolares* regularly emerge vertically from the canal up into the alveolus. Between the first and second premolars the canal turns toward the buccal and opens to the exterior as the *foramen mentale*. During ageing atrophy the foramen comes to lie on the alveolar ridge in approximately 25% of cases. A short distance from the opening of the foramen a small curved canal turns into the middle of the spongiosa cranially toward the front (the *canalis incisivus*) with blood vessels and nerves for the canines and incisors:

— accessory canals, such as the Robinson canal and the Serres canal, course through the mandible in 8% of cases [6];
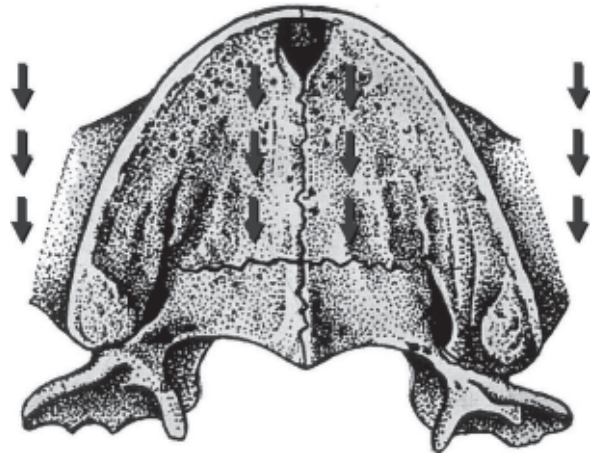

Figure 3. Horizontal atrophy of the maxilla.

— anastomoses exist between the two *canales mandibulae*;
— during ageing the position of the *foramen mandibulae* changes.

Maxilla

The atrophy progresses primarily in the horizontal dimension and only slightly vertically. The alveolar process itself, which only provides limited room to the tooth roots, is larger than the maxillary body. This reduces the maxillary arch more markedly. Through resorption it becomes smaller in relation to the mandible. In the maxilla the middle of the ridge is shifted to the palatal (centripetally) as the atrophy increases. Atrophy of the hard palate proceeds from front to back (Fig. 3). It can become so extreme that the hard palate is perforated, bringing the mucous membrane of the oral cavity into contact with that of the nasal cavity.

The alveolar process ends behind the last molars at the *tuber retromolare*. Up to the age of 7 the tuber exists only in an "embryonic" state. The *canales alveolares* for the Nn. *alveolares superiores posteriores* are present only as *sulci alveolares*. After the age of 20 the tuber is completely formed/differentiated, and the *canales alveolares* are distiguishable. After the age of 50 the tuber begins to regress; the *canales alveolares* open and become *sulci alveolares* again. These changes show that the *tuber retromolare* is not suitable as a site for implant abutments [4]. In the anterior region of the maxilla many canals are found for the accommodation of vessels and nerves; the canals can also be sulci which open to the maxillary sinus [2].

No atrophic alterations are found on the *torus palatinus*, *crista zygomatico alveolaris*, or the *spina nasalis anterior*. The masticatory pressure supports
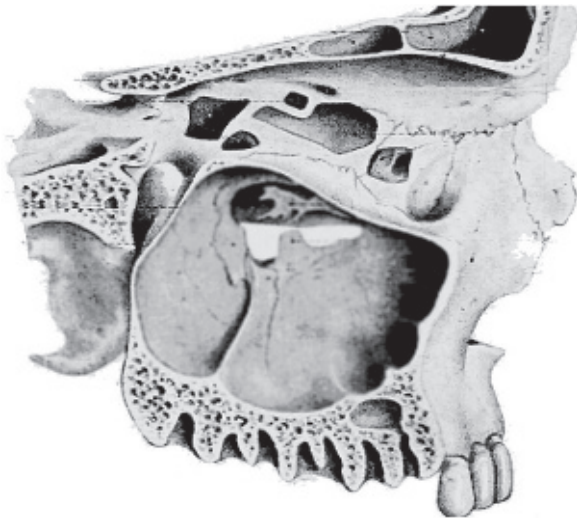
Figure 4. Varying structure of the maxillary sinus [3].

of the maxilla are not subject to atrophic processes and remain intact.

The maxillary sinus represents a danger zone; it lies in the maxillary body as a three-sided pyramid. Because it exhibits great variability, it makes implantation in the maxilla more difficult (Fig. 4).

Many exceptions and peculiarities in form and position are possible [2, 4, 5]:
— the floor of the maxillary sinus lies below the level of the nasal floor in 70% of the cases;
— after tooth loss, not only does the alveolar wall begin to atrophy but the sinus floor also starts to drop, so that after several years of tooth loss only a thin bone lamella separates the floor of the empty alveola from the maxillary sinus. For this reason the forms of the sinus differ between the dentate and edentate;
— the maxillary sinus floor is usually concave and smooth-walled;
— the smallest distance from the maxillary sinus is shown by the root apices of the second and often also of the first molars [our unpublished data] (Table 2); the alveolar wall of these two teeth can bulge out the floor of the sinus, producing a more or less pronounced partitioning and compartmentalisation of the sinus.
— pronounced narrowing of the sinus is also possible; this is a favourable situation;
— variously shaped accessory sinuses can also appear: alveolar sinuses, palatal sinuses, infraorbital sinuses and zygomatic sinuses;
— vessels and nerves lie (though not obligatorily) in wall canals or in half-open "gutters", which are covered with maxillary sinus mucosa.

Table 2. Vertical dimension of alveolar ridge in premolar and molar region

	Distance from apex to sinus floor
First right upper premolar [mm]	6.18 ± 1.6 7.05 ± 1.92
Second right upper premolar [mm]	2.86 ± 0.60
First right upper molar [mm]	1.56 ± 0.77 2.79 ± 1.13 2.82 ± 0.59
Second right upper molar [mm]	0.83 ± 0.49 1.97 ± 1.21 2.04 ± 1.19

Finally, it must be mentioned that the spongiosa of the maxilla has a trajectorial construction as the bone's response to pressure-tension loading during chewing. Intact alveoli are also trajectorially constructed.

General principles of implantation from a morphological perspective

The following points must always be taken into consideration for implantation [4]:
— the bone available;
— bone structure;
— vessel and nerve supply;
— the proximity of nasal cavity and paranasal sinuses;
— properties/condition of the mucous membranes.

The following local contraindications are noteworthy:
— scarcity of bone tissue;
— bone defects;
— cranial shift of the *canalis mandibulae*;
— complicated occlusal and articulation relations;
— macroglossia;
— non-alleviable parafunctions;
— critical oral hygiene.

General contraindications are varied and treatable to a certain extent: systemic bone diseases, rheumatic diseases, heart and kidney diseases, cirrhosis of the liver, haemorrhagic tendencies, allergies, insufficient immune defence and transitory infections.

Histological aspects of bone healing

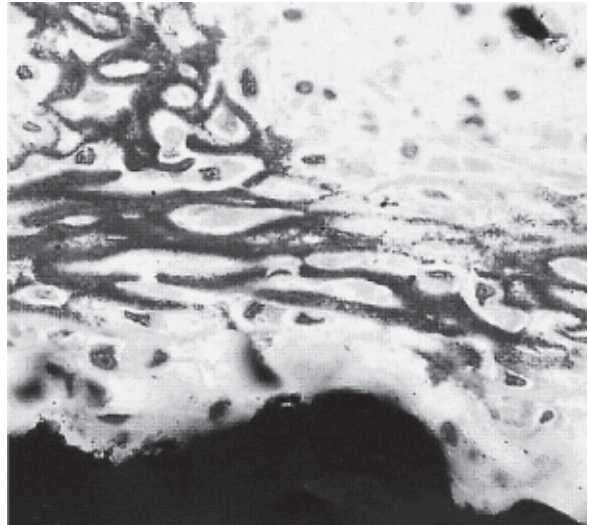A distinction is made between an early and a late phase.

Early phase. As in every healing process bleeding and the formation of a blood coagulum precede later steps. The coagulum forms the "template

tissue" for the reparative tissue (granulation tissue). The superficial adhesion of the blood coagulum on the implant is of great importance for bone healing. The coagulum is organised by ingrowing capillaries and their accompanying pre-osteoblasts, a process termed "centripetal bone growth". In addition to bone formation, the organism recognises the foreign body in this early phase. Macrophages and multi-nucleated giant cells appear. In this phase the interactions between tissue and implant are difficult to evaluate because of the overlapping of acute inflammatory and proliferative wound healing processes.

Late phase. In this period, bony healing per se occurs. A direct bridging of the gap between implant surface and bone takes place via concentrated lamellar bone formation. Larger gaps are bridged within 14 days by fibrous bone growth (Fig. 5). This fibrous bone is restructured into lamellar bone in approximately two months, but in the centre it still contains remainders of fibrous bone. A completely ossified ensheathment of the implant never occurs [5]. In the area of the bone-tissue-free implant surface adipocytes lacking a fibrous separating layer contact the implant.

Figure 5. Osseointegration with first formation of the trabecula (toluidine staining, 320-fold magnification).

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