Ridge augmentation for an atrophied posterior mandible using NanoBone block

Part II: Treatment outcome of clinical complications

Authors_Dr Omar Soliman & Prof. Dr Dr Mohamed Nassar, Egypt

_In the previous issues of implants **international magazine of** oral implantology (implants 1/2014) the authors gave a detailed introduction to their topic. In this issue, their report is completed.

At the time of implant placement, usually four months after the grafting procedure, the remodelling process is still underway. Even seven months after grafting, significant amounts of non-vital bone can be found.[8] Certain factors may influence the efficacy of the regeneration process. Revascularisation of the graft is crucial to tissue nutrition and regeneration. Revascularisation of a cancellous bone graft is tenfold faster than that of a cortical bone graft.[9] The regenerative potential of the residual ridge is also an important factor. Highly atrophied ridges usually consist of cortical bone that is not well vascularised and does not provide many cells.[10] These factors can influence the time needed for remodelling of the graft. Clinically, poor bone regeneration can be visually established from poor bleeding because of an inadequate blood supply, or from an inhomogeneous structure. Sometimes, even a clear border between the grafted bone and residual ridge can be observed.[5] In most cases, the screw has to be removed before implants can be placed after bone grafting. If the graft is not properly integrated, implant placement can loosen the graft. Mechanical stability of the graft is an important factor for proper bone regeneration and integration. It is well known that osteoblasts differentiate into fibroblasts under mechanical overload.[10] If mobility of the graft is observed, soft tissue has to be removed, bleeding should be provoked and mobile fragments have to be recured with the screws to allow the tissue to heal for another three or four months.[5] Another reason for insufficient integration


Fig. 1a

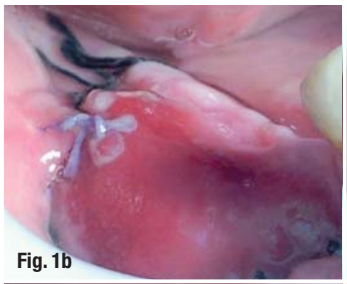
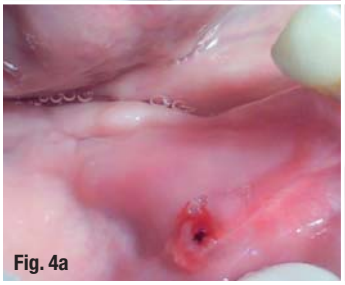


Fig. 1b


Fig. 2


Fig. 3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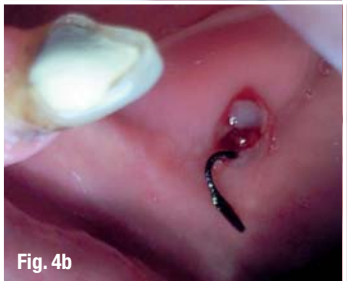
Fig. 4b


Fig. 5a

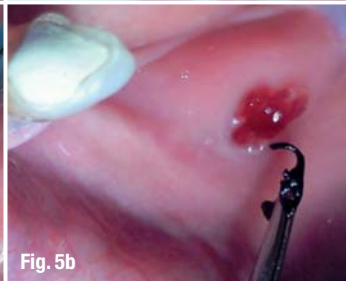
Fig. 5b

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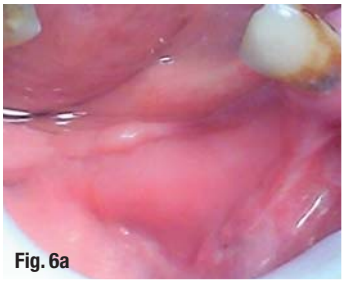
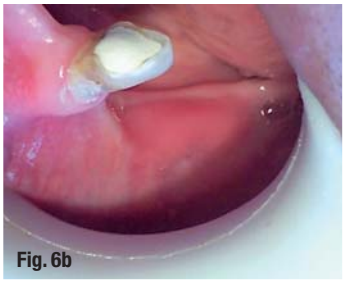


Fig. 6a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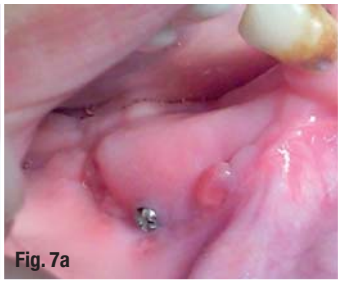
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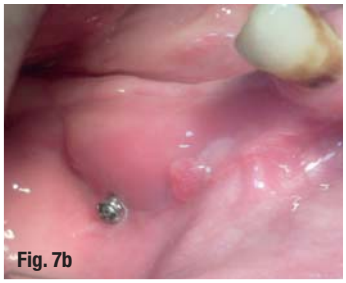
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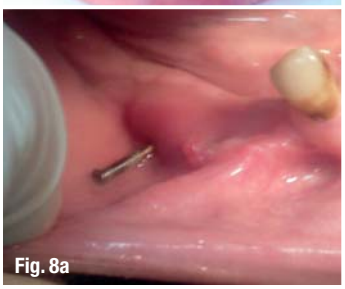
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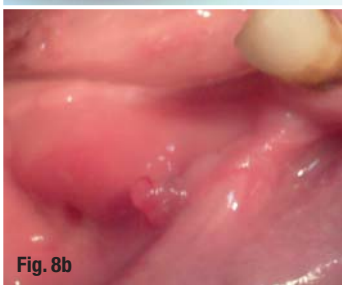
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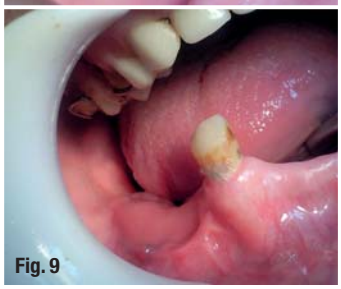
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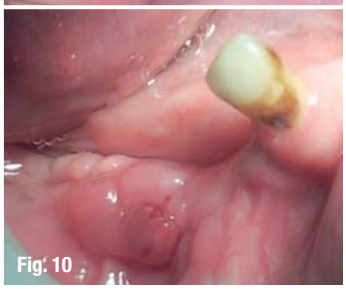
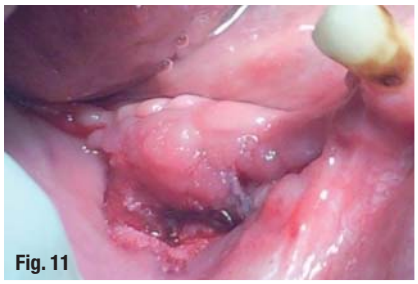


Fig. 10

of bone grafts is the migration of soft tissue, such as connective tissue, between the grafts, or between the graft and the residual ridge.[4] There should be proper adaptation of the graft to the defect. All the gaps between the block graft and the residual ridge must be filled with bone chips to prevent ingrowth of the connective tissue. If all of the gaps are sealed with particulate bone, no membrane is needed to prevent the ingrowth of soft tissue, but titanium mesh can be useful in some cases to stabilise the grafted material and to hold it in place. If fibrous or granulation tissue is present in the grafted area, it should be removed before grafting.[5] Resorption may be identified by the appearance of the fixation screw through the tissue as the soft tissue follows the underlying bone. Regeneration is commonly observed in block grafts.[12–14] Dehiscence leads to a higher percentage of resorption. Combining a membrane with a block graft has been reported to achieve less bone resorption.[15] However, a high complication rate, such as dehiscence of up to 14 %[16] or 18 %[17] with resulting infection or resorption, has been reported in connection with non-resorbable membranes, making this approach less attractive. Titanium mesh may be useful for avoiding resorption and appears to cause less dehiscence than do other non-resorbable materials.[18]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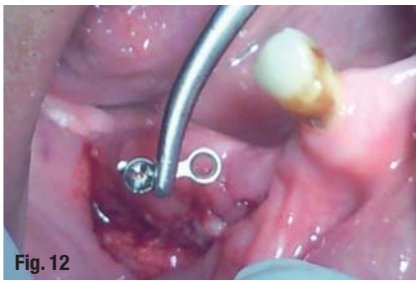
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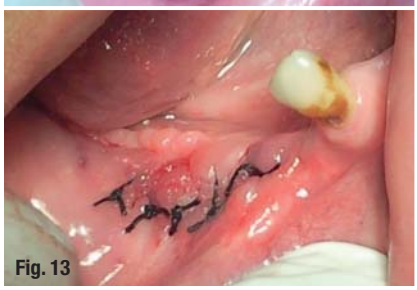
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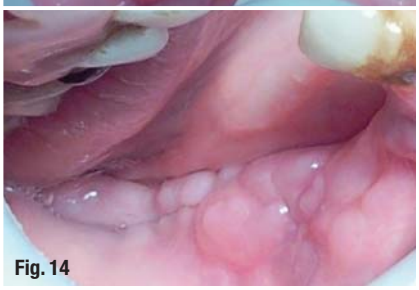
Fig. 14

_Bone remodelling and resorption after grafting

Parallel to the healing of transplanted bone, including revascularisation and remodelling, the volume of the grafted area is reduced in the first few months after the surgical procedure. Bone resorption of different forms and intensity is a typical phenomenon after the transplantation of a free bone graft.[19] There are different reasons for this bone resorption, depending on the graft technique, localisation, the type of surgery, soft-tissue pressure and muscle function. The bone quality of the graft and of the recipient site, the amount of revascularisation and revitalisation, and some genetic parameters influence the intensity of this bone resorption.

Resorption of the grafted bone has been found to be influenced by the following parameters:[6]

1) Bone blocks inside the contours of the alveolar crest for reconstruction of a failed bone wall showed significantly greater and faster resorption than those grafted inside the contours. This resorption of the grafted bone outside the contours can be influenced by implant insertion into the grafted area, thereby moderating resorption.
2) Functional loading of the grafted bone with an implant reduces the amount of bone resorption. When implants had not been inserted in the grafted area, the majority of the bone

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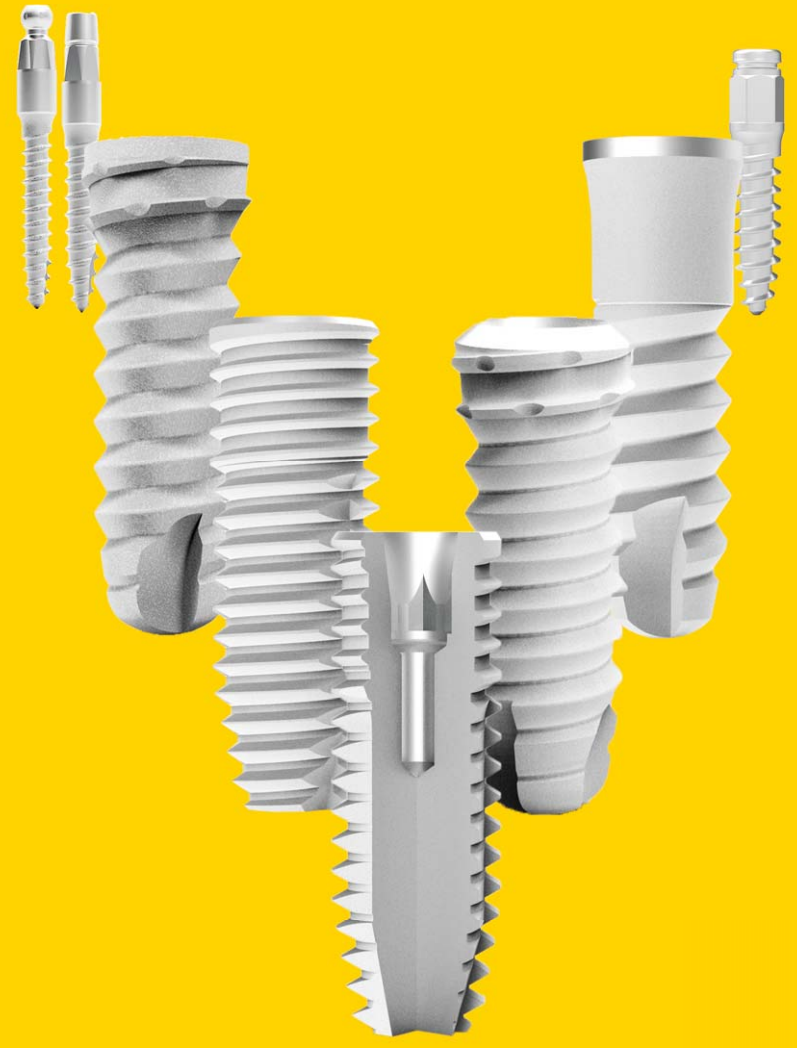

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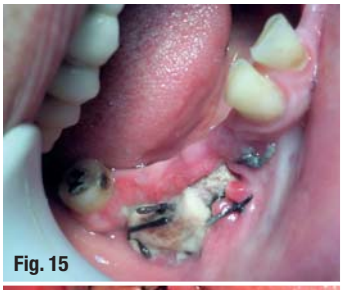
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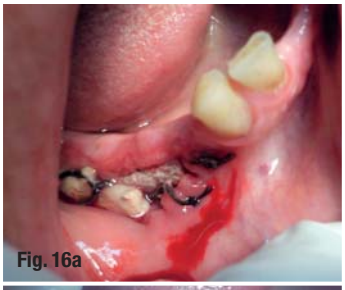
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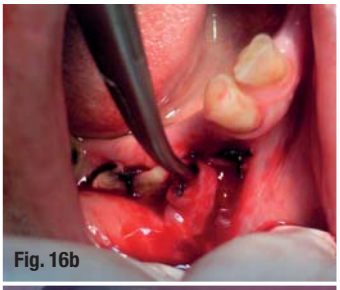
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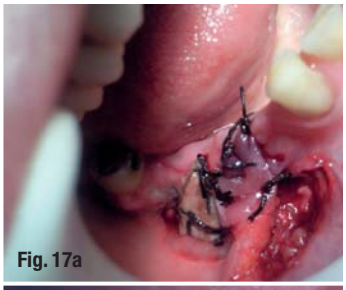
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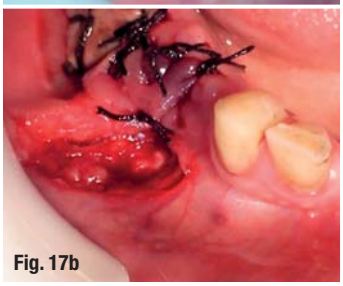
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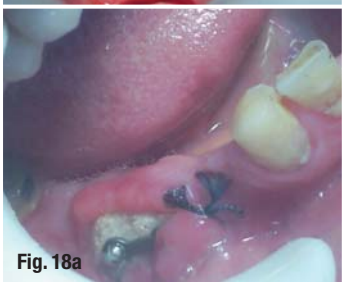
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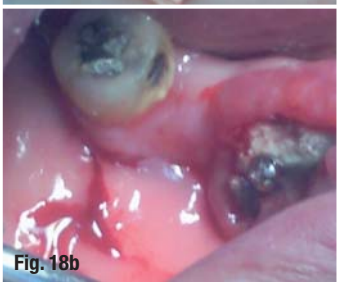
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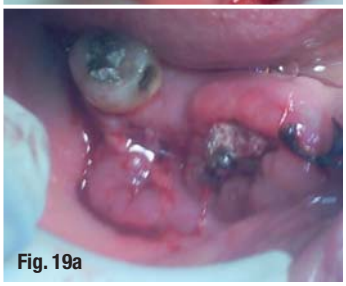


Fig. 19a

gained resorbed after eight months, especially in the grafted bone outside the contours.

3) Overextension of the bone graft is not a prophylactic measure against resorption: the greater the overextension of the grafted area, the greater the resorption.
4) The location of the grafted area appears to have an influence on the intensity of resorption. Maximum resorption of the grafted bone four months after surgery was found in the anterior region of the mandible, followed by the posterior region. This phenomenon can be explained by muscle activity.
5) The type of flap and use of the tunnel technique for grafting procedures appear to reduce the amount of bone resorption. This can be explained by the influence of periosteal integrity on osteoclast activity.

_Nerve injury

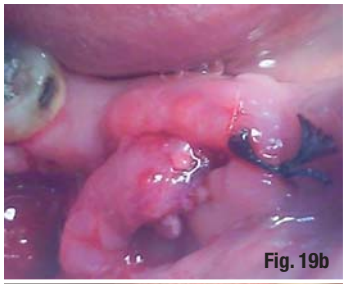
Trauma to the inferior alveolar nerve during implant placement may lead to loss of or altered sensation (paraesthesia) or to painful symptoms (dysaesthesia). Numerous unpleasant sensations may be described by the patient, including numbness, a crawling feeling, constant or periodic sharp pain, itching, tingling, hypersensitivity and burning, throbbing, pins and needles, prickling, and warmth or cold. In implant dentistry, these sensations often affect the lower lip, chin and the lower anterior gingiva. Rarely, the tongue may also have an altered sensation. Patients with tongue symptoms suffer a loss of taste. According to Girard et al., the inferior alveolar nerve may have the potential for recovery up to two years after injury.[20] However, Sunderland estimates that 75–90 % of distal nerve atrophies are irreparable after one year of altered feeling.[21]

Classification of nerve injuries

1) Neurapraxia: neurapraxia is a mild injury caused by compression injury to the nerve or retraction of the nerve. Examples of compression injuries include
   - pressure from saline or blood from the implant site while the implant is being screwed into position;
   - post-operative bleeding within the bone or around the mental foramen;
   - an implant inserted into the mandibular canal;
   - a piece of bone that invaded the canal during site preparation or implant insertion; and
   - a tie-back suture on the facial or lingual flap.

In neurapraxia, there is no axonal degeneration distal to the point of the nerve injury, but there is a temporary conduction block during nerve recovery. Spontaneous recovery of the altered sensations most often occurs weeks after this type of injury. When the patient presents with symptoms of a nerve injury within two days of surgery, an oral dose of a corticosteroid (e.g. Decadron 8 mg) decreases inflammation and swelling in the region. If the nerve trunk is compressed or retracted during surgery beyond the usual protocol, the intravenous form of a corticosteroid (e.g. 1–2 mm of Decadron 4 mg/ml) may be applied (not injected) to the injured area for 1–2 minutes. This direct application will decrease the risk of Nissl body disintegration, which the causes the paresthesia.[22]

2) Axonotmesis: axonotmesis is a nerve injury with loss of axonal continuity but with the general structure of the nerve remaining intact (the endoneurium is preserved). These injuries are more significant and may result in dysaesthesia or less-than-normal nerve recovery. Examples of axonotmesis injuries include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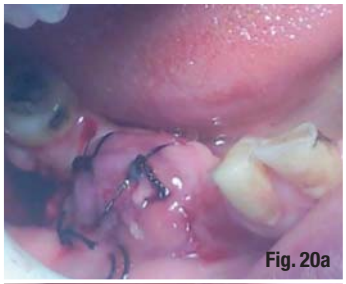
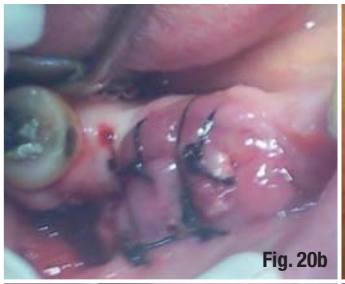


Fig. 20a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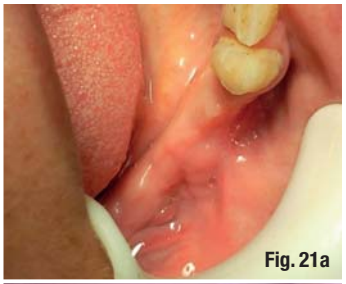
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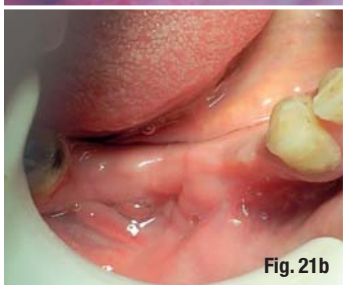
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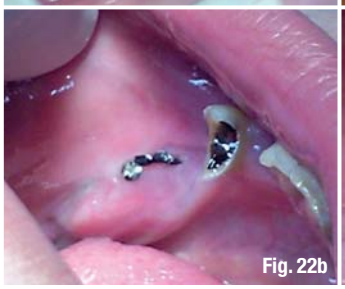
Fig. 22a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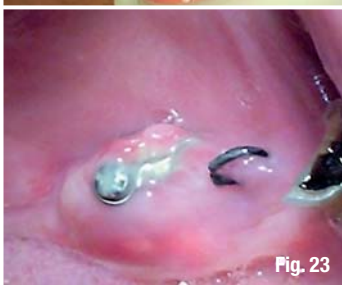


Fig. 23

– a nerve stretch injury from reflection of a soft-tissue flap;
– an implant drill proceeding through the top of the neurovascular canal; and
– an implant violating the canal.

If a post-operative radiograph shows that an implant may have slightly violated the canal space, it is prudent to unscrew the implant, introduce Decadron 4 mg/ml into the osteotomy site and place a shorter implant after 2–3 minutes. In addition, a corticosteroid is given orally for three to five days (the usual dose is 8–12 mg in the morning of the first day, 4–6 mg in the morning of the second day and 2–4 mg in the morning of the third day).[23]

3) Neurotmesis: neurotmesis is the complete severance of the nerve trunk. When this occurs, all axons distal to the injury undergo Wallerian degeneration. Anaesthesia of the soft tissue innervated by the affected nerve is a consequence of this condition. When a discontinuity or gap is present between the nerve ends, scar tissue forms between the structures and axonal sprouts from the proximal aspect of the nerve are prevented from penetrating the endoneurial tubules. Neurotmesis is suspected when anaesthesia is present or has been present for more than three months.

_Complications during and after second-stage implant surgery

Exposure of the graft

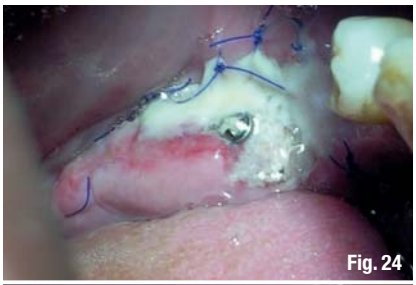
Even several months after grafting, significant amounts of non-vital bone can be found.[24] Vascularisation of the transplanted bone is poorer than in the residual crest, and neither a humoral immune response nor secondary wound healing is guaranteed. This leads to the necessity of careful soft-tissue management in second-stage surgery. Several techniques are reported to achieve adequate peri-implant soft tissue.[25] If parts of the transplanted bone are exposed, soft-tissue closure has to be surgically performed after debridement.[5]

Mobility of the implant

If an implant fails in the augmented site, granulation tissue has to be removed carefully. Radiographic evaluation and implant placement can be performed six to eight weeks later.[5]

Flap necrosis

Traumatic surgery, infection or insufficient vascularisation may lead to flap necrosis. Secondary healing can take place if the underlying bone is vital and well vascularised. If the underlying bone is still premature, complications can arise.[5]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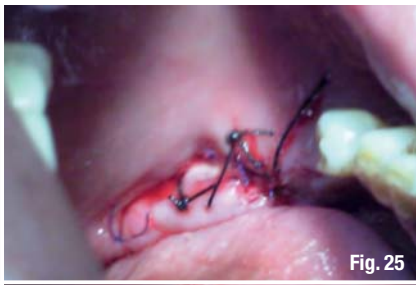
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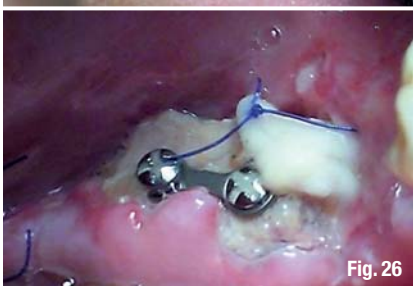
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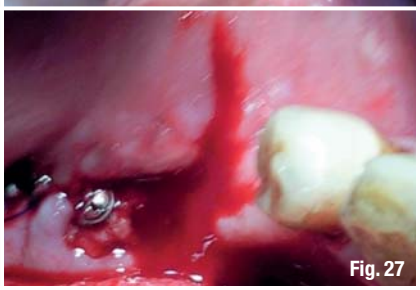
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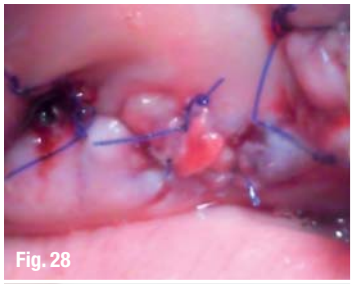
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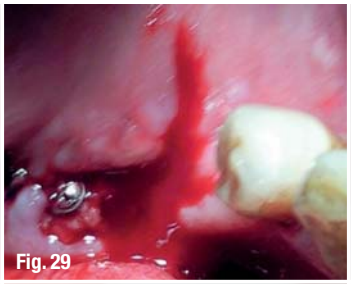
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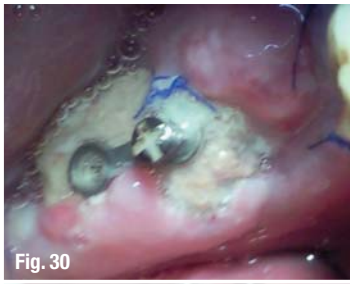
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Fig. 31a


Fig. 31b

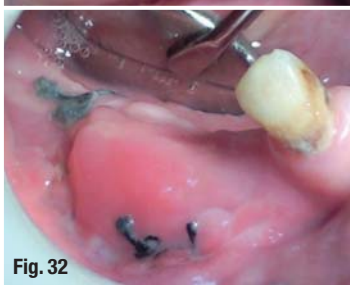
Fig. 32

_Late complications after prosthetic restoration

Bone loss

In an experiment on dogs, Berglund and Lindhe demonstrated that the thickness of peri-implant soft tissue influences the amount of bone resorption that takes place after second-stage surgery to establish biologic width.[26] In this process, a biological interface between the bone, soft tissue and implant is established, which is composed of the barrier epithelium (2 mm) and the connective tissue attachment (1–1.5 mm). These phenomena result in bone loss of up to 2 mm on a radiograph. A higher rate of bone loss may be of concern because it can be the result of mechanical overload or chronic inflammation of the peri-implant soft tissue.[5]

Loss of attached gingiva

A loss of fixed gingiva is frequently observed around implant restorations. Bengazi et al. and Grunder reported an average loss of 0.5 mm of fixed gingiva in the initial years after prosthetic restoration.[27,28] In the mandible, vestibuloplasty and connective tissue grafts are suitable techniques to shape aesthetic and functional peri-implant soft tissue.[5]

_Treatment outcome of clinical complications

A traumatic ulcer affecting the overlying mucosa of a maxillary molar (Figs. 1a&b) was treated with a maxillary partial denture to maintain centric occlusion (Fig. 2), protecting the overlying mucosa and NanoBone block graft. After the treatment, a complete healing of the traumatic ulcer was observed (Fig. 3). During this treatment, the use of a chlorhexidine solution several times a day was useful in reducing bacterial infiltration. An infection of the NanoBone graft (Fig. 4a) and Fisiograft (Fig. 4b) from the suture was treated by removing the suture (Figs. 5a & b), prescribing an antibiotic and a mouthwash, which in total lead to a complete healing (Figs. 6a & b).

In the early healing stage, the screws have to remain in place for proper stabilisation of the graft. In four cases, a fixation screw had loosened and become exposed in the late stage of graft healing (Figs. 7a & b). This screw was removed to prevent infection of the graft (Figs. 8 a&b). In three cases, the soft-tissue perforation healed after several days (Fig. 9). In cases in which the area around the mini-plate and the second cover screw became inflamed (Figa. 10 & 11), we removed the remaining cover screw and mini-plate (Fig. 12), sutured the wound (Fig. 13), and the soft tissue healed after several days (Fig. 14).

In a case in which the mesial part of the NanoBone graft had become exposed (Fig. 15), a mesial mucosal pedicle graft was performed to cover the exposed bone graft (Figs. 16a & b). Afterwards, the wound was sutured (Figs. 17a & b). For the same case, the distal part of the graft had become exposed (Figs. 18a & b) and a distal mucosal pedicle graft was performed to cover the exposed graft (Figs. 19a & b), but the graft size markedly decreased. Then, a wound suture was performed (Figs. 20a & b).

A decrease in augmentation size was noticed (Figs. 21a & b). Once the cover screws (Figs. 22a & b) and mini-plate had been exposed but not loosened (Fig. 23) with partial exposure of the graft (Fig. 24), resuturing of the dehiscent area was performed (Fig. 25) after reducing the volume of the NanoBone

graft (Fig. 26) and performing buccal relieving incisions (Fig. 27) with resuturing afterwards (Fig. 28). However, this led to a larger exposed surface of the NanoBone graft (Fig. 29) owing to subsequent flap necrosis. The NanoBone graft had to be removed completely owing to infection and the result was a total graft failure.

An infected NanoBone graft (Fig. 30) was treated by incision and pus drainage (Fig. 31 a&b). The NanoBone graft healed but decreased in size (Fig. 32).

_Conclusion

Careful patient follow-up after ridge augmentation using a NanoBone block is very important for the success of the augmentation procedure. Great attention to detail and a meticulous technique may prevent the progression of complications to failure of the graft. Treatment of a bone block graft exposed in the early stage is very difficult and has a poor prognosis. To date, there is no predictable method for treating this type of complication. Resuturing the dehiscent area at an early stage can lead to an even larger exposed surface of the graft owing to subsequent flap necrosis. Therefore, it is better to wait until the soft tissue matures. During this time, the use of a chlorhexidine solution several times a day can be useful in reducing bacterial infiltration. After a period of at least four weeks, surgical closure can be performed after reducing the volume of the block graft. However, the probability of saving all or parts of the graft is very low.[5]

Editorial note: This is the second part of a two-part article, the first of which was published in implants 1/14 ("Ridge augmentation for an atrophied posterior mandible using NanoBone block"). A complete list of references is available from the publisher.

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Dr Omar Soliman
PhD Candidate Perioimplant Dentistry

Tel.: +20 1009634358, +20 1201005457
Omar.Soliman77@yahoo.com

Prof. Dr Dr Mohamed Nassar
Professor of Perioimplant Dentistry
Faculty of Dentistry, Tanta University, Egypt

Tel.: +20 1121522221
prof_mnassar@yahoo.com

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