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Review Article

A Review on one rare complication "Avascular Necrosis of Maxilla" after Le Fort I Orthognathic Surgery

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***ABSTRACT*:**
The sequelae of insufficient vascularity following maxillary orthognathic surgery can vary from loss of tooth vitality, to periodontal defects, to tooth loss to loss of major maxillary dentoalveolar segments. The literature is replete with reports of the successful surgical treatment of maxillary and mandibular skeletal deformities. It is unusual, however, to see a report of the case that failed. Understanding the blood supply of the maxilla and how possible patient related, anesthetic and operative factors affect it, is important in understanding how the vascularity of the maxilla can become compromised in a surgical procedure. Avascular necrosis of the maxilla is a rare complication of orthognathic surgery with few cases reported in the literature. There are identifiable risk factors that can influence the blood supply of the maxilla. Careful preoperative assessment is required to exclude patient factors that have the potential to affect tissue vascularity. This in conjunction with sound anesthetic and surgical technique should all minimize the risk of avascular necrosis. Even so it is still possible for this rare complication to occur.\
Key words: Avascular Necrosis, Orthognathic Surgery, osteonecrosis.

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BACKGROUND:
Orthognathic maxillary surgery is a safe, predictable and stable procedure. The incidence of perioperative complications is low and the number of life-threatening complications with this surgery is even lower. However, avascular necrosis of the maxilla after Le Fort I osteotomy is a rare complication that has been reported to occur in <1% of cases.[1] During down fracture of the maxilla, the blood supply is bythe ascending pharyngeal artery, the ascending palatine branchof the facial artery and the rich mucosal alveolar network overlyingthe maxilla.[2] Table 1 outlines the factors that mayresult in impaired blood supply to the maxillary segments andincrease the risk of ischemia. When the descending palatine artery is sacrificed, the main blood supply of the maxilla will now be the soft tissue pedicle, which incorporates the anterior faucial pillar and the palatal mucosa (Fig. 1).Maxillofacial orthopedic surgery is a safe, predictable, and stable procedure.[3,4] The number of life-threatening complications associated with this surgery appears to be very small.[3,5,6] Other minor intraoperative and perioperative complications have been reported, but their incidence is considered low.[3-6] Among these complications, avascular necrosis of the maxilla after Le Fort I osteotomy has been reported by a few studies.[5,7,8] Usually, these complications are related to the degree of vascular compromise[9] and occur in fewer than 1% of

cases.[10] Rupture of the descending palatine artery (DPA) during surgery, postoperative vascular thrombosis, perforation of palatal mucosa when splitting the maxilla into segments, or partial stripping of palatal soft tissues to increase maxillary expansion may impair blood supply to the maxillary segments.[8]Sequalae of compromised vasculature include loss of tooth vitality, development of periodontal defects, tooth loss, or loss of major segments of alveolar bone or the entire maxilla. [5,8,11] The risk and the extent of complications seem to be enhanced in patients with anatomical irregularities, such as craniofacial dysplasia's, orofacial clefts, or vascular anomalies.[5] Accordingly, the risk of ischemic complications is enhanced in patients who present anatomical irregularities that require extensive dislocations or transversal segmentation of the maxilla.[5] The treatment of avascular necrosis of the maxilla is not easily attained.[12] Although no treatment protocol has been established, aseptic necrosis of the maxilla should be treated by maintenance of optimal hygiene,[13] antibiotic therapy to prevent secondary infection, [8,13] heparinization,[8,14] and hyperbaric oxygenation. [8,13,14] A recent report[15] described treatment of avascular necrosis of the maxilla related to a previously performed orthognathic surgery by hyperbaric oxygenation, bone grafting, and oral rehabilitation by an implant-supported fixed prosthesis, with a successful outcome. The aim of this report is to present a clinical case of avascular necrosis of the maxilla during the first postoperative days after a bimaxillary orthognathic surgery performed in a middle-aged woman, emphasizing treatment of this condition and correlating it with the current literature.

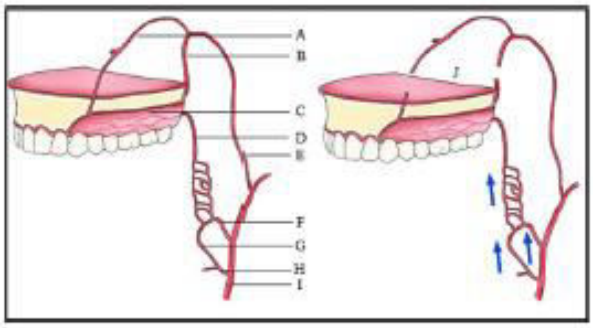

Figure 1: Blood supply of the maxilla. (A) Nasopalatine artery, (B) Descending palatine artery, (C) Greater palatine artery, (D) Lesser palatine artery, (E) Maxillary artery, (F) Ascending pharyngeal artery, (G) Ascending palatine artery, (H) Facial artery, (I) External carotid artery, (J) Le Fort I downfracture.

RARE COMPLICATION:

Blood flow to the maxilla is reduced by 50% in the first postoperative day after sacrifice of the descending palatine arteries.[16] However, there is excellent collateral blood supply particularly if only one artery is sacrificed, as in this case. Experimental studies have shown that loss of the descending palatine arteries results in a transient ischemic period that is compensated for by a vascular proliferation that allows tissue healing. The collateral microvasculature from other vessels including the ascending pharyngeal and facial arteries (Fig. 1), maintains the viability of the palatine pedicle. The pedicle can withstand stretching greater than 10mm of anterior repositioning of the maxilla. The patient had sickle cell trait. The haemoglobinopathy screen confirmed Hb A (Adult Hemoglobin) and Hb S (Sickle Hemoglobin). The Hb F (FetalHemoglobin) level was <0.5% and the Hb S level was 39.4%. These parameters are typical for sickle cell trait. However, for patients in Sickle Cell crisis or those expected to be exposed to severe hypoxia, Hb S level <30% is advised.[17] Craniofacial dysplasia's may represent areas of anatomical variation where the blood supply may be susceptible to disruption. Although the patient had a biopsy showing normal bone, the maxillary tuberosity was expanded and hyperplastic clinically. This in itself would not however have accounted for the degree of avascularity seen in this case. Literature suggests that segmental osteotomy is at a higher risk of ischemic complications.[18,19] Necrosis of the maxilla can be minimized in the following ways: – Divide into as few segments as possible and avoid small segments anteriorly. – Maintain the integrity of the palatal mucosa. – Perform sagittal segmentation in paramedian sites as the mucosa is thicker and the bone thinner than the midline. Although hypotensive anesthesia was not purposely utilized for the procedure, it is a technique commonly used to help maintain a bloodless surgical field.[20] A mean arterial blood pressure (MAP) 30% below a patient's usual MAP, with a minimum MAP of 50mmHg in American society of anesthesiologists Class I patients and a MAP not <80mmHg in the elderly, is suggested to be clinically acceptable.[21] It is recommended that with respect to hypotensive anesthesia: – It should be adjusted in relation to the patient's preoperative blood pressure rather than to a specific target pressure. – It should be limited to that level necessary to reduce bleeding in the surgical field. – It should be confined to that part of the surgical procedure deemed to benefit by it. – There is little need for blood transfusion perioperatively.[22] The anesthetic concerns include the management of perioperative hypoxia, acidosis, hypovolemia and hypothermia. Points to consider with this case are: (1) Hypoxia never occurred and is usually avoided as anaesthetized patients are always maintained with an FiO2 >0.35. (2) Acidosis may trigger a vaso-occlusive crisis and would affect all micro-circulation; it is easily prevented by proper perfusion and maintaining circulating blood volume. (3) Hypothermia was prevented by patient warming. (4) Hypovolemia could be contentious, especially with hypotension. With this patient, the mechanism of avascular necrosis could not clearly be explained. In the analysis of patient, surgical and anesthetic factors, there seemed to be issues that may have implied a greater risk.

DISCUSSION:

Aseptic necrosis of the maxilla is one of the possible consequences of ischemic problems occurring during Le

Fort I osteotomies, along with gingival recession.[5,8,10] Laningan et al[8] reported 36 cases of aseptic necrosis of the maxilla after Le Fort I osteotomies, usually related to multiple segmentation of the maxilla in conjunction with superior positioning and transverse expansion or palatal perforations. However, other studies[23,24,25] suggested that segmentation of the vascular pedicle by extensive anterior dislocation of the maxilla and transection of the descending vessels exhibit no relevant effect on revascularization or bone healing. Kramer et al[5] showed that the rate of intra- and perioperative complications of Le Fort I are infrequent and mostly associated with anatomical complications, including deviation of the nasal septum and nonunion of the osteotomy gap. Another possible cause of maxillary would be related to ischemic complications, reported to occur in approximately 1% of cases, including aseptic necrosis of the alveolar process and retraction of the gingival margin. Ischemic complications have occurred in a small number of patients without recognized damage to palatal vessels during surgery. All patients showing ischemic complications had anatomical irregularities, as also suggested by Drommer,[26] transversal segmentation, or extensive anterior dislocation of the maxilla of 9 mm or more. Also, Bays et al[27] reported a 0.7% incidence of aseptic necrosis of the maxilla after Le Fort I osteotomy with routine bilateral ligation of the DPA in 149 patients. However, Acebal-Bianco et al[6] and Panula et al[4] found no loss of maxillary bone segments resulting from vascularization problems in large-sample studies. The main cause of avascular necrosis of the maxilla is DPA ligation during surgery.[8,10,24,28,29] The main advantages of ligating DPAs during surgery are decreased risk of postoperative bleeding, easier maxillary mobilization, and shortening of surgical time.[9,30] Hemorrhage has been considered as a major complication in the first 48 postoperative hours, requiring blood transfusion in 1% to 1.1% of the cases.[3,5] However, blood transfusion should be avoided because of associated morbidity.[3,31] Excessive blood loss occurring during surgery or several hours later is mainly related to maxillary osteotomies. Controlled hypotensive general anesthesia and post positioning the patient in a slight anti-Trendelenburg position, along with use of a local anesthetic containing a vasoconstrictor,may help to reduce blood loss.[3] However, when using hypotensive anesthesia, laceration of the vessels may be masked, and even intraoperative inspection of vessel integrity does not guarantee that nobleeding will develop afterward.[30] Preserving DPA vessels during surgery is justified by the hypothetical benefit of maintaining blood flow and decreasing the risk of ischemic complications.[30]The known risk is postoperative bleeding caused by unrecognized laceration of the DPA.[11,13,30] A reasonable approach, therefore, is to routinely preserve the integrity of these vessels when feasible, and to ligate them when enhanced accessibility or visualization is required, such as superior or posterior repositioningof the posterior maxilla, allowing visualization and access to the tuberosity-pterygoid plate junction, and facilitating repositioning of the maxilla.[10] In clinical practice, bone contiguous to vessels is routinely removedby careful and meticulous use of a sharp osteotome and Kerrison forceps.[11] Vertical movements, especially setbacks, could perhaps lead to injuries of the DPA with subsequent thrombosis, even if no ruptureis observed. DPAs can be damaged intraoperatively during initial osteotomy cuts, maxillary down fracture, when achieving transverse modifications or duringthe intrusion or advancement procedure.[8] Advantages of identifying and protecting the DPA include protecting maxillary blood supply, particularly in multiple segmented osteotomies, removing mechanicalbony obstruction especially in impaction and setback movements, and preserving sensorial functions of the palatine nerves.[31] The pyramidal osseous release technique around DPAs with a rotary drill described by Johnson andArnett[32] prevents leaving bony contacts posterior to the artery, which could lead to immediate post fixation anterior open bite, as the condyle sets back in the fossa. Preservation of the DPA is also imperative when maxillary segmentation is necessary,[32] and doesnot significantly lengthen surgical time.[9]A modification of this technique was proposed by O'Regan and Bharadwaj[28] by the use of a spatula, which provides better visualization and lower risk of inadvertent injuryof the vessel. In the present clinical case, although the patient had developed unexpected intense bleeding during surgery, no ligation or electrocauterization of the DPA was performed,as hemostasis was promptly achieved by compression with gauze, and minor anterior displacement of the maxilla was anticipated by facial analysis software. The rationale for preserving DPAs during Le Fort I osteotomy is to optimize maxillary integrity by maintaining blood nutrition to the anatomical area, decreasing the risk of ischemic necrosis.[8] Another possible reason for aseptic necrosis of the maxilla is palatal perforation, which compromises the already tenuous blood supply to the anterior maxilla, and leads to avascular necrosis.[8]When the maxilla isto be expanded more than 3 to 5 mm, there is a risk of avulsing portions of the attached palatal pedicle during forceful manipulations,[13] which was not observed in the present clinical case. In addition, avascular necrosis of the maxilla can be related to impaired blood supply to marginal gingiva. After Le Fort I down fracture, blood supply of the maxillary and palatal regions is established primarily through major and secondary palatine branches of DPA, soft tissue branches of posterior superior alveolar artery, palatal branch of the ascending palatine artery and palatal branch of the facial artery.6 An 84% to 95% reduction in blood flow to the osteotomizedsegment in a group of animal subjects that had the vessels bilaterally severed suggests that this pedicle should be maintained during surgery.[29] A significant decrease in anterior maxilla gingival blood flow (GBF) occurs during the intraoperative course of Le Fort I osteotomy,[33] which could explain

buccal and lingual gingival recession observed in this case. According to Dodson et al,[9] ligation of the DPA was not associated with a change in anterior maxillary GBF during Le Fort I osteotomy. The trans osseous and soft-tissue collateral blood circulation and the freely anastomosed plexus of the gingiva, vestibule, palate, nose, maxillary sinus, and periodontium provided the necessary blood supply after sectioning of the descending palatine vessels.[28,29] Although bone segmentation, stretching of the vascular pedicle, flap design, and bilateral sectioning of the DPA have been implicated in impairing the hemodynamics of the maxillary pedicle,6 Bell et al[11] showed that vascular alterations are only transient and are compatible with clinical success without resultant ischemic complications. Stretching the vascular pedicle by a 7- to 10-mm anterior displacement of the maxillary segment did not interfere with eventual osseous healing and revascularization.[34] It is not known to what degree, and for how long, blood flow can be impaired so as not to disturb continuous blood supply to the tissues, as well as the range of individual variability. Aseptic necrosis, with loss of tooth vitality, may occur much more commonly than is clinically apparent after maxillary surgery because obvious clinical signs and symptoms frequently do not accompany this situation.[8] Toothvitality tests commonly used in dentistry, involving either electrical or thermal stimulation, are unreliable after orthognathic surgery because sensory fibers from the trigeminal nerve are severed at surgery. Gingival bleeding is reduced in patients who smoke, even in the presence of moderate to severe periodontal disease, accompanied by reduction of other clinical signs of inflammation.[35] These findings can be related to the diminished existence of large blood vessels in the buccal gingiva of patients who smoke compared with those who do not smoke, along with proliferation of small blood vessels, without significant alterations in vascular density.[36] Patients who smoke can show increased periodontal bone loss even in the absence of dental plaque,[37] suggesting that smoking is an important risk factor for periodontal disease.[38,39] Cessation of smoking habits results in stabilization of the periodontal condition after long-term follow-up, [40,41] although the periodontal conditions of former smokers have been found to be worse than those of nonsmokers.[42] Recent studies[43] have suggested that risk factors for aseptic osteonecrosis can be induced by intravascular thrombosis, for which risk factors are smoking andexcessive consumption of alcoholic beverages, with no relation to blood dyscrasias. These findings were also reported by Wolfe and Taylor-Butler,[44] who described avascular necrosis of the shoulders in a patient reporting long-term use of corticosteroids, smoking, and alcohol intake. Although the use of bisphosphonates has been extensively implicated in osteonecrosis of the jaws,[45] its incidence appears to be greater in obese patients and patients who smoke.[46] Smoking is significantly associated with osteonecrosis of the jaws and has been linked to effects in all organs of the human body. Specifically, in the oral cavity, carcinogens present in cigarettes, cigars, and pipe tobacco delay wound healing and are associated with a worsening of periodontal conditions of smokers.[47-49] Nicotine may cause vasoconstriction in bone, leading to ischemic states that underlie the pathological mechanisms of osteonecrosis.[50,51,52] Both periodontal disease and oral osteonecrosis seem to result from pathogenic mechanisms influenced by the interaction between environmental genotoxic risk factors and genetics, conferring individual susceptibility. According to Baldi et al,[53] osteonecrosiscan occur in patients exposed to high doses of DNA-damaging agents, such as chemotherapy and radiotherapy for cancer treatment, and bisphosphonates for the treatment of osteoporosis. Oxidativedamage caused by smoking plays a pathogenic role in periodontal disease, as established by the detection of mitochondrial DNA damage in the gingival tissue of patients with periodontitis. Endogenous risk factorsin dental diseases include polymorphisms for many metabolic enzymes, metalloproteases, cytokines, prothrombin, and DNA repair activities. Considering that, both osteonecrosis of the jaws and periodontal diseasecould be related to risk factors associated with environmental mutagenesis. Given that, in the present case, the patient was not obese and did not report use of bisphosphonates, corticosteroids, or excessive alcohol consumption, the only risk factor associated with both periodontal disease and avascular necrosis of maxilla was smoking. Correction of avascular necrosis of the maxilla is not easy to attain.[12] The risk and the extent of complications seem to be enhanced in patients with anatomical irregularities, such as craniofacial dysplasia, orofacial clefts, or vascular anomalies. In addition, the risk of ischemic complications is enhanced in patients requiring extensive dislocations or transversal segmentation of the maxilla,[5,29] which was not performed or diagnosed in the present case. Treatment of aseptic necrosis initially involves the establishment of optimal hygiene measures of thearea, accomplished by frequent irrigation with saline solution.6 Ideally, the patient should be treated with hyperbaric oxygen, and antibiotics should be considered to prevent secondary infection.[8,13] Surgical debridementis required to remove necrotic bone fragments, allowing earlier wound healing.[8] Hyperbaricoxygen may hasten the delineation of the necrotic segments and allow a definitive debridement to be done at an earlier time.[8,13] However, hyperbaric oxygenation does not reverse the development of aseptic necrosis once it has started, although it may limit the extent of such necrosis[8] No protocol for hyperbaric oxygenation has been proposed in literature. Recently, Singh et al[15] reported the treatment of a patient who had experienced avascular necrosis of the maxilla after an orthognathic surgery performed 8 years previously. The patient complained of maxillary pain, pronounced facial asymmetry, malocclusion, and difficulty in eating and was diagnosed with sinusitis, mobile maxillary teeth, and transverse alveolar collapse of maxilla,

mandibular anterior posterior excess, and facial asymmetry. Treatment was performed by 30 sessions of hyperbaric oxygenation, followed by extraction of condemned teeth, debridement of necrotic bone and maxillary right sinus, and reconstruction of the alveolar ridge with an iliac crest graft. Three months later, Osseo integrated implants were inserted, and the patient was rehabilitated with an implant-supported fixed prosthesis with adequate esthetic outcomes. Hyperbaric oxygenation is capable of normalizing the vascular bed within 10 days of treatment, probably because of synthesis of new vascular elements, and the same size of the vascular bed could be seen after 30 days. Blood flow, on the other side, was reduced after 10 days, probably because of new synthesis of blood vessels.[14] Heparinization is also reported as a treatment option.[8,14] Perfusion to ischemic regions may be improved by reducing blood viscosity, which is a primary factor in blood flow. Heparin administered subcutaneously reduced morphological tissue damage to the teeth and bone, but this treatment was complicated by significant hemorrhage from the surgical site.6 However, this treatment was not performed in the present case, thereby avoiding excessive bleeding from the wound at the postoperative period. Careful assessment of the circumstances involved when small or large bone segments are lost usually indicates that basic biological principles have been violated, such as inadequate soft tissue flap design or impairment of blood supply to maxillary segments. Excessively long and traumatic surgery, inappropriate selection of interdental sites for osteotomy, strangulation of the circulation by imprudent use of palatal splints, and excessive stretching of the palatal mucosalpedicle are other causes of compromised wound healing.[11] Basic biological principles were strictly followed during and after orthognathic surgery in the present case, with no ligation of descending palatine vessels to warrant proper bone supply to the osteotomized maxilla, and no hemorrhage from the surgical site at immediate postoperative follow-up. These findings suggest that aseptic necrosis of the maxilla was probably related to smoking rather than to anatomical irregularities or iatrogenic. This condition was completely resolved with hyperbaric oxygenation therapy along with antibiotic therapy and optimal hygiene care of the wound area, requiring no removal of necrotic bone. In an attempt to minimize the possibility of aseptic necrosis we suggest the following, even though we are cognizant of the fact that this complication can still occur even with the best surgical techniques due to inherent biological variability:

1. Preserve the descending palatine arteries whenever possible. In cases where a significant intrusion of the maxilla is performed, especially if the maxilla is also returned, an attempt should be made to limit the kinking of the vessels by gently teasing them out of the surrounding bone superiorly. This procedure is only useful provided the vessel is not encased in a thick bony buttress, because otherwise the trauma of the dissection could lead to increased damage to the vessel, resulting in hemorrhage or thrombosis.
2. Divide the maxilla into as few segments as possible and try to avoid small segments anteriorly. Good preoperative orthodontics should allow the number of sections to be minimized. In some cases, it may be better to compromise and accept a lack of go d contact in the second molar region rather than perform a four-piece maxillary osteotomy to get these teeth into occlusion. In three-piece maxillary osteotomies it may be possible to avoid extending the palatal bone cut across the midline. If, however, the maxillary anterior teeth have to be rotated into the splint to avoid torqueing them, this could place increased tension on the palatal mucosa and compromise blood supply more than making the osteotomy cut. If premolar teeth are extracted at the time of a Le Fort I osteotomy, and the anterior maxillary segment is repositioned posteriorly, this must be done very carefully to avoid damaging the palatal vascular pedicle because the palatal mucosa will already be buckled to some extent. Consideration could be given to preoperatively repositioning the anterior teeth orthodontically, or to performing a Wanderer anterior maxillary osteotomy, thereby predicting this segment to the labial mucosa, combined with bilateral posterior maxillary osteotomies, instead of a conventional multisegmental Le Fort I osteotomy.
3. Avoid compression of the palatal mucosa or gingivae by a palatal splint, or compression of the labial gingivae and alveolar bone by skelor infraorbital rim areas. The necessity for palatal splints and skeletal fixation has fortunately decreased with the use of rigid internal fixation.
4. If significant transverse expansion is necessary, consideration should be given to initial surgically assisted orthodontic palatal expansion. A Le Fort I osteotomy might be necessary at some time in the future for final correction in the vertical, anterior/posterior, or transverse dimensions. In cases where marked transverse problems exist, and two-jaw surgery is to be performed, consideration could also be given to narrowing the mandibular arch via a symphyseal osteotomy rather than doing all the transverse changes in the maxilla.
5. Consider a horseshoe-shaped palatal cut. rather than a midpalatal split if a large amount of transverse expansion is required.
6. Have good preoperative orthodontic separation of teeth in areas of osteotomy cuts to avoid damage to the interdental bone and roots.
7. Disimpaction forceps are probably best avoided whenever possible because their use may slightly injure palatal mucosa and could compromise blood supply. The maxilla should be able to be downfractured in most instances with only hand pressure.
8. If-unexpected occurrences happen at the time of surgery, it may be prudent to be conservative and either stop the procedure, or plan to accomplish less than was originally intended. If, for example, after completion of a Le Fort I

down fracture the maxilla is noted to be pale white, suggestive of arterial ischemia, or dark purple from venous congestion, the procedure should be temporarily stopped, the maxilla repositioned in its normal location, and the hypotensive anesthesia reversed. After a time, if the color does not improve it may not be prudent to proceed with significant anteroposterior or vertical changes, or transverse modifications. If a maxilla in a cleft palate patient is noted to develop poor color after an intended major anterior advancement, it may be advisable to rely on an intermediate splint where the maxilla has been advanced to a lesser extent. There is undoubtably a time limit wherein one can afford to wait to see if the situation improves, beyond which the situation is likely to deteriorate to irreversible aseptic necrosis. It would probably be imprudent to delay more than 8 to 12 hours before returning a patient to surgery to move the maxilla back to its original, or an intermediate position, to see if its color improves. Improvements in vascularity are more likely to occur when the surgical changes have primarily been anteroposterior, rather than vertical or transverse, since the latter modifications are less easily reversed. These situations demand judgment calls when a decision has to be made in the operating room or in the early postoperative period, and when a conservative approach may result in a less-than-optimal final result or necessitate additional surgery at a later time. It may, however, in certain instances avert a disastrous result. Consideration should be given to having duplicate models available, especially for cleft palate patients, in case an intermediate splint has to be fabricated in the early postoperative period to aid in repositioning the maxilla to a more suitable location.

9. Other suggestions made by surgeons responding to the questionnaire included performing the surgery under minimal hypotensive anesthesia, avoiding electrocautery for maxillary buccal vestibular incisions, and having patients avoid smoking postoperatively. Treatment of aseptic necrosis initially involves good hygiene in the area, with frequent irrigation with saline. Ideally the patient should be treated with hyperbaric oxygen, and antibiotics should be considered to prevent secondary infection. A surgical debridement will be required to try to speed up the resolution of the necrotic process.

Hyperbaric oxygen may hasten the delineation of the necrotic segments and allow a definitive debridement to be done at an earlier time. It will not, however, prevent or reverse the development of aseptic necrosis once it has started, but may limit its ultimate extent. Later reconstruction may require closure of the defects with soft tissue flaps, bone grafts from the iliac crest, or hydroxyapatite. The resulting defects can then be corrected by fixed or removable prosthetic appliances. Osseo integrated implants may be a useful adjunct.[13]Till date, little experimental work has been done to investigate the prevention of avascular necrosis in the jaws. Nilsson et al[54] examined the effects of heparin, dextran 40, dextran 70, and hyperbaric oxygen to reduce tissue damage to teeth and bone after mandibular osteotomies in rats. Tissue damage was recorded morphologically, whereas blood flow was determined by isotope techniques. Hyperbaric oxygen had a beneficial effect on the amount of tissue damage that occurred as compared with nontreated animals. It was hoped that perfusion to ischemic mandibular regions via collateral circulation could be improved by reducing blood viscosity, which is a primary factor in blood flow. Although in other experimental studies intravenous dextran had been reported to improve the microcirculation.[55] T"dextran 40 or dextran 70 had no preventive effect on tissue damage in this investigation. Heparin administered subcutaneously reduced morphologically determined tissue damage to teeth and bone, but this treatment was complicated by significant hemorrhage from the operative field. Jaques[56] has found in recent experiments that when heparin is given orally the endothelial cell concentration is from 100 to 1,000 times the plasma concentration. It is therefore possible that if heparin is given orally postoperatively it could improve the microcirculation without leading to significant bleeding complications. The possible usefulness in preventing or limiting the extent of aseptic necrosis with the use of agents such as heparin or dextran sulphate requires further investigation.

CONCLUSION:

It is hoped that the publication of this review will accentuate the importance of a sound background in anatomy, physiology, and basic surgicalprinciples which one must have before attempting any operation on a patient.At least, one should be expected to have a thorough understanding of a procedurebefore attempting it.It is incumbent upon our profession to prepare our trainees to be aware ofthe recent advances in oral surgery and to be capable of performing these proceduresor to recognize what they are not competent to handle.

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