Immediate or Early Placement of Implants Following Tooth Extraction: Review of Biologic Basis, Clinical Procedures, and Outcomes

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Purpose: *The aim of this article was to review the current literature with regard to survival and success rates, along with the clinical procedures and outcomes associated with immediate and delayed implant placement.* ***Materials and Methods:*** *A MEDLINE search was conducted of studies published between 1990 and June 2003. Randomized and nonrandomized clinical trials, cohort studies, case-control studies, and case reports with a minimum of 10 cases were included. Studies reporting on success and survival rates were required to have follow-up periods of at least 12 months.* ***Results:*** *Thirty-one articles were identified. Most were short-term reports and were not randomized with respect to timing of placement and augmentation methods used. All studies reported implant survival data; there were no reports on clinical success. Peri-implant defects had a high potential for healing by regeneration of bone, irrespective of healing protocol and bone augmentation method. Sites with horizontal defects (HD) of 2 mm or less healed by spontaneous bone fill when implants with rough surfaces were used. In the presence of HDs larger than 2 mm, or when socket walls were damaged, concomitant augmentation procedures with barrier membranes and bone grafts were required. Delayed implant placement allowed for resolution of local infection and an increase in the area and volume of soft tissue for flap adaptation. However, these advantages were diminished by simultaneous buccolingual ridge resorption and increased requirements for tissue augmentation.* ***Discussion:*** *Immediate and delayed immediate implants appear to be predictable treatment modalities, with survival rates comparable to implants in healed ridges. Relatively few long-term studies were found. Successful clinical outcomes in terms of bone fill of the peri-implant defect were well established. However, there was a paucity of data on long-term success as measured by peri-implant tissue health, prosthesis stability, and esthetic outcomes.* ***Conclusions:*** *Short-term survival rates and clinical outcomes of immediate and delayed implants were similar and were comparable to those of implants placed in healed alveolar ridges.* INT J ORAL MAXILLOFAC IMPLANTS 2004;19(SUPPL):12–25

Key words: *bone regeneration, dental implants, delayed implants, extraction socket, immediate implants, implant survival, literature review*

Since the first report of the placement of a dental implant into a fresh extraction socket,[1] there has been increasing interest in this technique for implant treatment (for reviews see Schwartz-Arad and Chaushu[2] and Mayfield[3]). The advantages of immediate implant placement have been reported to include reductions in the number of surgical interventions and in the treatment time required.[4,5] It has also been suggested that ideal orientation of the implant,[6,7] preservation of the bone at the extraction site,[8–10] and optimal soft tissue esthetics[6] may be achieved.

However, it has been reported that immediate implant placement may be adversely affected by the presence of infection[11–13] and lack of soft tissue closure and flap dehiscence over the extraction site,[14] particularly when barrier membranes have been used for guided bone regeneration.[15–20] Treatment outcomes for both submerged and nonsubmerged placements may be affected by lack of tissue volume[21] and thin tissue biotypes. In addition,

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Table 1 Human Histologic Studies Presenting Data on Osseous Regeneration in Extraction Sockets

Study	Duration	No. of patients	Sites	First appearance of osteoid	Initial calcification	Substantial bone fill of socket
Amler et al (1960)[32]	50 days	Not stated	Varied	7 days	18 days	38 days (⅔ fill)
Boyne (1966)[33]	19 days	12	Maxillary teeth	10 days	—	—
Amler (1969)[34]	50 days	Not stated	Varied	7 days	20 days	40 days (⅔ fill)
Evian et al (1982)[35]	16 weeks	10	Varied	—	4 to 6 weeks	10 weeks (complete fill)

incongruity between the shape of the implant body and that of the socket wall may lead to gaps between the bone and the implant. At the present time, there is a lack of consensus on the need for immediate implants and the optimal regenerative techniques to be used with them.[2,3] The clinician must therefore decide whether augmentation procedures are necessary and, if so, the most efficacious technique to use. To overcome the problems of immediate implantation, alternative techniques have been described, calling for implant placement at various intervals following initiation of wound healing subsequent to tooth extraction.[14,22–27]

This article will examine the biologic basis, as well as the indications and clinical outcomes, of immediate and delayed implant placement. It will not deal with techniques for delayed implant placement following soft and hard tissue augmentation at the time of tooth extraction (for a review on this topic, see Adriaens[28]). An understanding of extraction wound healing and subsequent bone resorption, regeneration, and remodeling of the healing socket is necessary to provide a basis for reviewing the outcomes of implants placed early after tooth extraction.

LITERATURE REVIEW AND SEARCH RESULTS

A MEDLINE search was conducted to identify clinical articles published between 1990 and June 2003. The search terms used were "immediate" and "implants," "implants" and "extraction sockets," "delayed-immediate" and "implants," "delayed" and "implants," "delayed implants" and "extraction," "delayed placement" and "implants," and "early placement" and "implants." In addition, the bibliographies of 2 review articles were checked for appropriate studies.[2,3] The reference lists of identified studies were then searched for additional citations. Randomized clinical trials and nonrandomized cohort studies, case control studies, and case series with a minimum of 10 cases were included. In addition, studies reporting on success and survival rates needed to have follow-up periods of at least 12 months.

A total of 31 studies that met the criteria for this review were identified. Of these studies, 18 provided data on survival rates of immediate and delayed implants. Nineteen studies provided clinical, radiographic, and re-entry data on healing around immediate and delayed implants.

HEALING OF EXTRACTION SOCKETS

Histologic Events

The events that occur in a healing extraction socket have been identified by examination of animal histologic material[29–31] and human biopsies (Table 1).[32–35] Five stages of healing have been described.[34] In the first stage, an initial clot forms as a coagulum of red and white blood cells derived from the circulation. In the second stage, granulation tissue replaces the clot over a 4- to 5-day period. Cords of endothelial cells are associated with budding capillaries. In the third stage, connective tissue gradually replaces granulation tissue over 14 to 16 days. The connective tissue is characterized by the presence of spindle-shaped fibroblasts, collagen fibers, and a metachromatic ground substance. In the fourth stage, calcification of osteoid is apparent, commencing at the base and periphery of the socket. Early osteoid is seen at the base and periphery of the socket by 7 to 10 days. Bone trabeculae almost completely fill the socket by 6 weeks. In the fifth stage, complete epithelial closure of the socket is achieved

Table 2 Studies Measuring Mean Apicocoronal and Buccolingual Change in Ridge Dimensions Following Tooth Extraction

Study	Healing time (mo)	Vertical change (mean ± SD)	Buccolingual change (mean ± SD)
Lekovic et al (1997)[39]	6	–0.86 ± 0.14 mm	—
Lekovic et al (1998)[40]	6	–1.50 ± 0.26 mm	—
Camargo et al (2000)[42]	6	1.00 ± 2.25 mm	3.06 ± 2.41 mm
Iasella et al (2003)[43]	4 to 6	–0.90 ± 1.60 mm	–2.63 ± 2.29 mm
Schropp et al (2003)[41]	12	–0.7 mm (–1.4/–0.2)*	–5.9 mm (–7.7/–4.7)*

*Indicates 25th/75th percentiles.

after 24 to 35 days. Substantial bone fill occurs between 5 and 10 weeks.[32–34] By 16 weeks, bone fill is complete, with little evidence of osteogenic activity at this time.[35]

Maximum osteoblastic activity, seen as a proliferation of cellular and connective tissue elements, with osteoblasts laying down osteoid around immature islands of bone, occurs between 4 and 6 weeks after extraction. After 8 weeks, the osteogenic process appears to slow down.[35]

External Dimensional Changes at Extraction Sockets

Morphologic changes in healing extraction sockets have been described by cephalometric measurements,[36,37] study cast measurements,[38–41] subtraction radiography,[41] and direct measurements of the ridge following surgical re-entry procedures.[39,40,42,43]

Measurements from diagnostic casts allow assessment of the gross morphologic changes that take place during healing and reflect changes in both the bone and overlying mucosa.[41] Approximately 5 to 7 mm of horizontal or buccolingual ridge reduction, representing about 50% of the initial ridge width, occurs over a 6- to 12-month period. Most of this change takes place during 4 months of healing.[38,44] A corresponding apicocoronal or vertical height reduction of 2.0 to 4.5 mm accompanies the horizontal change.[43,45] Greater apicocoronal changes take place at multiple adjacent extraction sites than at single extraction sites.[41,44,45]

The dimensional changes of the bone in healing sockets have been reported via intraoperative measurements (Table 2[39–43]). Loss of between 3.1 and 5.9 mm of buccolingual ridge width was observed in studies with observation periods of 4 to 12 months.[39–43] Schropp and coworkers measured dimensional changes in 46 healing sockets in 46 patients.[41] The extraction sites were confined to the premolars and molars in both jaws. All but 2 patients agreed to not wear a prosthetic replacement during the healing phase. It was found that a reduction in buccolingual width of approximately 50% (from 12.0 to 5.9 mm) took place over a 12-month period, with two thirds of this change occurring in the first 3 months after extraction. These changes were slightly greater in molar sites than in premolar sites, and in the mandible compared with the maxilla. At 3 months after tooth extraction, a reduction in apicocoronal ridge height of 0.8 mm was noted on the buccal aspect.

Apicocoronal crestal bone height reductions of 0.7 to 1.5 mm have been reported after 4 to 6 months.[39,40,42,43] In contrast, a gain in ridge height of 0.4 mm after 12 months was observed in one study.[41]

A variety of factors may influence the dimensional changes of the bone following tooth extraction, and it is clear that current knowledge is limited in many areas. Systemic factors may include the patient's general health and habits (eg, smoking). Local factors include the reasons for extraction, the number and proximity of teeth to be extracted, the condition of the socket before and after tooth extraction, the influence of tissue biotype on healing, local differences between sites in the mouth and the dental arches, and the type of interim prosthesis used.

Internal Dimensional Changes Within Extraction Sockets

Healing events within the socket reduce the dimensions of the socket over time. Vertical socket height reduction of 3 to 4 mm, or approximately 50% of the initial socket height, has been reported after 6 months of healing.[39,40] Horizontal socket width reduction of 4 to 5 mm, or approximately two thirds of the original socket width, has been shown to have occurred by 6 months of healing.[39,40,42]

A radiographic analysis using subtraction radiography over a 12-month period confirmed that bone formation within the socket occurred simultaneously with loss of alveolar crest height.[41] Most of this bone gain and loss occurred in the first 3 months following tooth extraction. In the same study, linear measurements of radiographs showed that crestal bone levels at the tooth surfaces adjacent to the extraction sites remained relatively unchanged over the 12-month observation period (mean 0.1 mm loss). In contrast, mesial and distal bone height levels in the extraction sockets were reduced by 0.3 mm. The level of bone that regenerated in the extraction sockets did not reach the level of the bone at the adjacent teeth.

Dimensional Changes in Damaged Extraction Sockets

The rate and pattern of bone resorption may be altered if pathologic or traumatic processes have damaged one or more of the bony walls of the socket. It is likely in these circumstances that fibrous tissue may occupy a part of the socket, thereby preventing normal healing and osseous regeneration from taking place.[28] There are insufficient data on the differences in rates and patterns of the healing of intact versus damaged extraction sockets.

Dimensional Changes of the Mucosa

It is generally believed that the form of the mucosa closely follows the changes in the underlying bone. An apical shift in the coronal bone may be followed by a similar shift in the position of the mucosa. However, in a study comparing healing of undisturbed sockets with healing of sockets grafted with freeze-dried bone allograft and a collagen membrane,[43] the authors reported that the thickness of the mucosa at the buccal aspect of the ridge crest increased by 0.4 mm after 4 months in the control group. The grafted group showed a loss of tissue thickness of 0.1 mm. The differences between test and control groups were significant.

Although complete epithelialization of the socket is established by the fifth week of healing, organization and maturation of the collagen in the underlying lamina propria takes longer to occur. Matrix synthesis begins at 7 days and peaks at 3 weeks; this is followed by a continuous process of maturation until complete tensile strength is restored several months later.[46] Lack of tensile strength in the mucosa of healing extraction sockets may result in wound dehiscence. Dehiscence rates of 5% to 24% have been reported at delayed implant sites treated with both resorbable and nonresorbable membranes, despite the presence of adequate tissue volume to achieve primary closure.[22,47]

CLASSIFICATION OF TIMING OF IMPLANT PLACEMENT AFTER TOOTH EXTRACTION

Several classifications have been proposed for the timing of implant placement following tooth extraction. In the classification of Wilson and Weber, the terms *immediate*, *recent*, *delayed*, and *mature* are used to describe the timing of implant placement in relation to soft tissue healing and the predictability of guided bone regeneration procedures.[14] However, no guidelines for the time interval associated with these terms were provided. In the recent classification of Mayfield, the terms *immediate*, *delayed*, and *late* are used to describe time intervals of 0 weeks, 6 to 10 weeks, and 6 months or more after extraction, respectively.[3] The interval between 10 weeks and 6 months was not addressed.

Most of the studies reviewed described immediate implant placement as part of the same surgical procedure and immediately following tooth extraction. The exceptions were Schropp and associates,[27] who defined immediate implantation as implants placed between 3 and 15 days (mean 10 days) following tooth extraction, and Gomez-Roman and coworkers,[48] who defined it as occurring between 0 and 7 days afterward. The majority of studies that described delayed implant placement used a delay period of 4 to 8 weeks after extraction. In a report published by Hämmerle and Lang, placement was delayed for 8 to 14 weeks.[25] In an additional 3 reports, implant placement was considered delayed when it occurred between 6 weeks and 6 months after extraction[47,49] and between 1 week and 9 months.[48] This variation indicates a lack of uniformity in the interpretation of the terms *immediate*, *delayed*, and *late*.

Thus, it is necessary to introduce clearer definitions of implant placement that are based on the morphologic, dimensional, and histologic changes following tooth extraction and on common practice derived from clinical experience.

CLINICAL OUTCOMES

Several studies have reported on clinical, radiographic, and bone defect changes that take place following placement of immediate and delayed implants (Tables 3 and 4). Ten studies reported on healing of immediate implants only,[17,18,51–53,57–61] and 3 studies dealt only with delayed implants.[25,54,55] Several articles compared immediate with delayed placement,[22,27,62] or immediate with late placement.[50,56] A total of 6 papers provided comparative data on immediate, delayed, and late placement.[10,24,26,47–49] The majority of the comparative reports were not randomized with respect to placement and augmentation techniques.

Healing of Immediate and Delayed Implant Sites

Following observation periods of between 1 and 4.5 years, no significant differences were reported to occur in radiographic crestal bone levels or in probing of pockets at immediate, delayed, or late implantation sites.[10,22,48,50,56]

The majority of studies reported that peri-implant defects associated with immediate implants healed with significant bone fill, irrespective of the placement protocol (submerged versus nonsubmerged) and augmentation method used.[51–53,57–61] However, significantly better bone fill (5.7 mm versus 3.2 mm) and less crestal bone resorption were reported at immediate implant sites treated with demineralized freeze-dried bone combined with nonresorbable barrier membranes, versus sites treated with a nonresorbable barrier membrane alone.[17] An exception to these positive findings above was reported in a study of immediate implants in 15 patients.[18] Substantial bone regeneration was observed histologically in only 3 of 15 tissue samples taken at the time of membrane removal. The results were compromised by wound dehiscences that resulted in early exposure of nonresorbable membranes in 10 of 15 patients. In other studies, premature exposure of nonresorbable membranes was reported to be associated with reduced volumes of regenerated bone in the peri-implant defects.[17,47,60] However, lower incidences of premature membrane exposure were observed using collagen membranes.[47,58]

Peri-implant defects encountered at the time of delayed placement have been reported to heal with significant reduction in defect dimensions. In the absence of augmentation techniques, defect height (DH) reduction was greater at sites with no horizontal defects (ie, the peri-implant space) compared to sites where horizontal defects were present (3.4 mm versus 1.1 mm).[54] Highly successful outcomes for defect area (DA) reduction (86% to 97% reduction) were reported in dehiscence defects treated with collagen barrier membranes and anorganic bovine bone mineral.[25,55]

Comparisons between immediate and delayed implantation sites showed a trend toward higher percentages of DH and DA reduction at delayed sites (range between studies for DH, 86% to 97%; for DA, 86% to 97%) compared with immediate sites (DH 77% to 95%; DA 77% to 95%). The exception was in the study of Schropp and coworkers, in which DH reductions were comparatively modest (48% immediate; 34% delayed).[27] In most cases, differences between groups for DH and DA reductions were not statistically significant.[24,47,49] However, Nemcovsky and colleagues found significantly better DH and DA reduction at delayed sites compared with immediate sites.[62]

Localized pathologic processes may lead to damage of one or more walls of the extraction socket, with the formation of dehiscence defects.[24,26,27,50,51] Sockets with dehiscence defects may lack the potential for complete bone regeneration, and the risk of long-term complications may be increased with immediate implants placed at these sites.[14] However, several reports have shown that bone regeneration may be achieved in dehisced sites adjacent to immediate implants using a variety of augmentation techniques, including a nonresorbable expanded polytetrafluoroethylene (e-PTFE) membrane and demineralized freeze-dried bone allograft,[51] a resorbable collagen membrane and anorganic bovine bone,[24] and autogenous bone alone.[27,52] In a comparative study, significantly greater defect height reduction was achieved in dehisced sites with delayed compared to immediate implant placement (88.8% versus 77.4%).[26] Interestingly, early placement (immediate and the earlier delayed) showed consistently better reduction of dehiscence defects than did late implantation in healed alveolar ridges.[24,26,47,49] Defect morphologies with early implantation present with 2 or 3 intact bony walls, whereas defects with late implantation tend to present as 1-wall or no-wall defects.[24] A report that 70% of 3-wall defects associated with immediate or early delayed implants healed without augmentation confirms the high potential for bone regeneration at these sites.[27] The location of the implant in relation to the socket appears to be a critical determinant of the outcome of regenerative treatment at dehisced sites. Thus, implants should be placed well within the confines of the socket to ensure a maximum number of bone walls and to take advantage of the healing potential of the socket.

Survival Rates

Eighteen studies were identified that fulfilled the selection criteria for this review (Table 5[10,11,13,22,24,48,50–54,56,61,63–67]). Only 4 studies involved nonsubmerged healing following immediate placement.[48,53,56,63] The majority of studies used a submerged healing protocol that required a second surgical procedure for abutment connection. Because the study of Polizzi and associates was a 5-year follow-up of a 3-year report of Grunder and coworkers, only the former study was included in the

Table 3 Clinical Studies Presenting Clinical and Radiographic Data of Healing at Immediate and Delayed Implant Sites and Comparing Immediate with Delayed or Late Placement

Study	Implant system/ surface	Randomization	Augmentation method;* healing protocol	No. of patients/implants			Observation period (mean, mo)	Mean radiographic crestal bone loss (mm)			Mean probing pockets (mm)		
				Immediate	Delayed (delay period)	Late		Immediate	Delayed	Late	Immediate	Delayed	Late
Yukna (1991)[50]	Calcitek/ HA	No	5; SUB	28†/14	—	28†/14	6	0.9	—	0.8	3.8 (REC 0.2)	—	3.2 (REC 0.3)
Mensdorff-Pouilly et al (1994)[22]	IMZ/HA, Brånemark/ MF	No	Various: 0, 1, 5, 1+3; SUB	31/93	36/97 (6 to 8 wk)	—	12.4	0.5	0.3	—	2.5	1.9	—
Brägger et al (1996)[56]	ITI/TPS	No	T = 1 only, C1 = ND; C2 = HS; NSUB	GroupT: 15/20; group C1: 6/8	—	GroupC2: 20/20	12	—	—	—	T: 2.54; C1: 2.47	—	C2: 2.70
Watzek et al (1995)[10]	IMZ/HA, Brånemark/ MF	No	Various: 0, 1+3, or 4; SUB	20†/97	20†/26 (6-8 wks)	20†/11 (edentulous sites)	27.1	1.0	0.8	0.5	1.8	2.9	1.5
Gomez-Roman et al (1997)[48]	Frialit/HA, Frialit TPS	No	1% sites required; NSUB	376†/86 (within 1 wk of extraction)	376†/164 (1 wk to 9 mo after extraction)	376†/446 (> 9 mo after extraction)	54	1	1	1	3	3	3

When not reported, values were derived from data contained in the original article.
*0 = no augmentation; 1 = e-PTFE membrane; 2 = collagen membrane; 3 = autogenous bone; 4 = anorganic bovine bone; 5 = hydroxyapatite, 6 = demineralized, freeze-dried bone.
†Indicates total number of patients for all groups.
SUB = submerged; NSUB = nonsubmerged; ND = no defects present; HS = healed sites; REC = recession; HA = hydroxyapatite-coated; MF = machine finished; TPS = titanium plasma-sprayed.

Table 4 Clinical Studies Presenting Surgical Re-entry Data on Peri-implant Bone Defect Changes Following Healing at Immediate and Delayed Implant Sites and Comparing Delayed Implant Placement with Immediate and/or Late Implant Placement

Study	Implant system/ surface	Random	Augmentation method*	No. of patients/implants: Immediate	Delayed (delay period)	Late (delay period)	Observation period (mean, mo)	Re-entry bone-defect changes
Gelb (1993)[51]	Brånemark/ MF	No	Various: 1, 1+6, 6; SUB	35/50	–	–	4 (mandible); 6 (maxilla)	Immediate: 100% thread coverage for all techniques, except in 1 case of a no-wall defect treated with treatment 6 only = 76% coverage
Becker et al (1994)[52]	Brånemark/ MF	No	3 only; SUB	30/54			Not stated	Immediate: VH 5.5 mm reduced to 0.5 mm‡; HD 3.7 mm reduced to 0.2 mm‡ (slightly less gain when buccal dehiscence present)
Gher et al (1994)[17]	ITI/TPS and Calcitek/HA	Yes	Test: 1+6, control: 1 only; SUB	Test: 20/22; control: 16/21	–	–	6 –1.53 mm	Immediate: Crestal bone loss from most coronal bone crest: test = , control = –1.59 mm; crestal bone loss from most apical part of crest: test = +1.39 mm, control = –0.11 mm (*P* < .05 between groups); Bonefill: test = 5.68 mm, control = 3.18 mm (*P* < .05 between groups)
Lang et al (1994)[53]	ITI/TPS	No	1 only; NSUB	16/21	–	–	5 to 7	Immediate: 20 of 21 sites with complete bone regeneration
Augthun et al (1995)[18]	Brånemark/ MF	No	1; SUB	15/20	–	–	6	Immediate: Histology: Evidence of bone formation in only 3 of 15 tissue samples
Zitzmann et al (1996)[49]	Brånemark/ MF	No	2+4; SUB	50†/25	50†/21 (6 wk to 6 mo)	50†/23 (> 6 mo)	4 to 6	Immediate: Defect area reduction of 89%‡ Delayed: Defect area reduction of 86%‡ Late: Defect area reduction of 76%‡
Zitzmann et al (1997)[47]	Brånemark/ MF	Yes	1+4, 2+4; SUB	25†/27	25†/17 (6 wk to 6 mo)	25†/40 (> 6 mo)	4 to 6	Immediate: Defect area reduction with e-PTFE membrane = 85%‡; with collagen membrane = 95%‡ Delayed: Defect area reduction with e-PTFE membrane = 94%‡; with collagen membrane = 97%‡ Late: Defect area reduction with e-PTFE membrane = 80%‡; with collagen membrane = 90%‡
Nir-Hadar et al (1998)[54]	Not stated	No	0; SUB	–	14/21 (6 to 8 wk)	–	3 to 6	Delayed: VH of defect 2.5 mm reduced to 0.4 mm with no HD present‡; VH of defect 3.9 mm reduced to 0.5 mm with HD present‡; HD 1.6 mm reduced to 0 mm‡
Hämmerle et al (1998)[57]	ITI/TPS	No	1; NSUB	10/11	–	–	5	Immediate: VH reduction from 4.7 mm at baseline to 2.1 mm at re-entry‡; 94% defect fill‡
Zitzmann et al (1999)[24]	Brånemark/ MF	No	2+4; SUB	75†/31	75†/23 (6 wk to 6 mo)	75†/48 (> 6 mo)	4 to 6	Immediate: Defect area reduction of 92% Delayed: Defect area reduction of 92% Late: Defect area reduction of 80%

Table 4 Clinical Studies Presenting Surgical Re-entry Data on Peri-implant Bone Defect Changes Following Healing at Immediate and Delayed Implant Sites and Comparing Delayed Implant Placement with Immediate and/or Late Implant Placement (continued)

Study	Implant system/ surface	Random	Augmentation method*	No. of patients/implants: Immediate	Delayed (delay period)	Late (delay period)	Observation period (mean, mo)	Re-entry bone-defect changes
Nemcovsky and Artzi (1999)[58]	Calcitek and SteriOss, MF and HA	No	1+4 vs 4 only; SUB	29/33	–	–	6 or 9	Immediate: VH reduction from 1.9 mm to 0.3 mm with no membrane‡; VH reduction from 4.6 mm to 0.7 mm with membrane‡
Nemcovsky et al (2000)[55]	Calcitek,/TPS and Calcitek/MTX	No	2+4; SUB	–	21/28 (5 to 7 wk)	–	6 to 8	Delayed: Defect area reduction of 97%
Van Steenberghe et al (2000)[60]	Brånemark/MF	No	4 at defects with HD > 3 mm; SUB	15/21	–	–	6	Immediate: Defect fill: Complete in 10 sites, partial in 9 sites, bone loss in 2 sites
Rosenquist and Ahmed (2000)[59]	Brånemark/MF	No	Homologous bone membrane; SUB	25/34	–	–	6	Immediate: VH reduction from 8.5 mm to 0.3 mm‡
Hämmerle and Lang (2001)[25]	ITI/TPS	No	2+4; NSUB	–	10/10 (8 to 14 wk)	–	6 to 7	Delayed: Defect area reduction of 86%‡
Goldstein et al (2002)[61]	Brånemark/MF and 3i /MF	No	6 ± barrier membranes; SUB	38/47 (27 sites with peri-implant defects)	–	–	Mean 6.5	Immediate: 100% defect fill
Nemcovsky et al (2002)[62]	Sulzer and SteriOss, MTX, TPS, and HA	No	2+4; SUB	19/23	24/31 (4 to 6 wk)	–	6 to 8	Immediate: Defect height reduction of 77.4%, defect area reduction of 90.2% ($P < .05$ between groups) Delayed: Defect height reduction of 91.2%, defect area reduction of 97.2% ($P < .05$ between groups)
Nemcovsky and Artzi (2002)[26]	Sulzer Calcitek/ MTX, TPS, and HA	No	All sites with buccal dehiscence defects, 2+4; SUB	19/23	25/39 (4 to 6 wk)	22/40 (HS)	6 to 8	Immediate: Defect height reduction of 77.4%, defect area reduction of 90.2% Delayed: Defect height reduction of 88.8%, defect area reduction of 95.6% Late: Defect height reduction 72.5% ($P < .05$ between the 3 groups); defect area reduction of 87.6% ($P < .05$ between the 3 groups)
Schropp et al (2003)[27]	3i /Osseotite/AE	Yes	3 (in dehiscence defects); SUB	23/23 (mean 10 d)	23/23 (mean 14.1 wk)	–	6	Immediate: Defect reduction: HW = 48%,‡ HD = 59%,‡ VH = 48%‡ Delayed: Defect reduction: HW = 39%,‡ HD = 77%,‡ VH = 34%‡

When not reported, values were derived from data contained in the original article.
*0 = no augmentation; 1 = e-PTFE membrane; 2 = collagen membrane; 3 = autogenous bone; 4 = anorganic bovine bone; 5 = hydroxyapatite; 6 = demineralized freeze-dried bone allografts.
†Indicates total number of patients for all groups.
‡Indicates significant change ($P < .05$) from baseline within the treatment group.
AE = acid-etched; MF = machine-finished; TPS = titanium plasma-sprayed; HA = hydroxyapatite coated; MTX = microtextured; HW = horizontal defect width (mesiodistal); HD = horizontal defect depth (buccolingual); VH = vertical defect height (apicocoronal); SUB = submerged; HS = healed sites; NSUB = nonsubmerged.

Table 5 Clinical Studies with Follow-up Periods of 1 Year or More Reporting Survival Rates of Immediate and Delayed Implants and Comparing Delayed with Immediate Implants

Study	Type of study	Implant system/ surface	SUB/ NSUB	Immediate		Delayed		Delayed placement time	Follow-up period (mean)	Survival rate (%)	
				No. of patients	No. of implants	No. of patients	No. of implants			Immediate (CSR)	Delayed (CSR)
Ashman (1990)[64]	Pros	Steri-Oss/MF	SUB	16	16	–	–	–	6 to 24 mo (NG)	94.1	–
Yukna (1991)[50]*	Pros	Calcitek/HA	SUB	14	14	–	–	–	8 to 24 mo (16 mo)	100	–
Gelb (1993)[51]	Retro	Brånemark/MF	SUB	35	50	–	–	–	8 to 44 mo (17 mo)	98.0	–
Becker et al (1994)[52]	Pros	Brånemark/MF	SUB	49	49	–	–	–	1 y	93.9	–
Mensdorff-Pouilly et al (1994)[22]	Retro	57 IMZ/HA, 40 Brånemark/ MF	SUB	31	93	36	97	6 to 8 wk	1 to 4 y (12.4 mo)	92.5 (80 mo)	94.9 (60 mo)
Lang et al (1994)[53]	Pros	ITI/TPS	NSUB	16	21	–	–	–	21 to 42 mo (30.3 mo)	100	–
Watzek et al (1995)[10]	Retro	20 IMZ/HA, 5 Brånemark/ MF	SUB	20†	97	20†	26	6 to 8 wk	4 to 83 mo (27.1 mo)	99.0	92.3
De Wijs et al (1995)[65]	Pros	IMZ/HA	SUB	–	–	81	173	3 mo or later	3 to 64 mo (33.5 mo)	–	96.1 (3y)
Rosenquist and Grenthe (1996)[11]	Pros	Brånemark/ MF	SUB	51	109	–	–	–	1 to 67 mo (30.5 mo)	93.6	–
Brägger et al (1996)[56]	Pros	ITI/TPS	NSUB	21	28	–	–	–	1 y	100	–
Gomez-Roman et al (1997)[48]*	Pros	Frialit-2/HA and TPS	NSUB	376†	86	376†	164	1 wk to 9 mo	1 to 5 y (4.5 y)	97.1 (4.5 y)	99.4 (4.5 y)
Cosci and Cosci (1997)[66]	Retro	Integral and Onmiloc/HA	SUB	353	423	–	–	–	1 to 7 y (NG)	99.5	–
Nir-Hadar et al (1998)[54]	Pros	Brånemark/ MF	SUB	–	–	14	21	4 to 8 wk	1 y	–	95.2
Zitzmann et al (1999)[24]	Retro	Brånemark/ MF	SUB	75†	31	75†	33	6 wk to 6 mo	1 y	96.8	93.9
Polizzi et al (2000)[13]	Pros	Brånemark/ MF	SUB	143†	217	143†	47	3 to 5 wk	5 y	92.4 maxilla, 94.7 mandible (5 y)‡	92.4 maxilla, 94.7 mandible (5y)‡
Schwartz-Arad et al (2000)[67]	Pros	NS; 47 MF, 9 HA	SUB	43	56	–	–	4 to 60 mo	15 mo	89.3	–
Gomez-Roman et al (2001)[63]	Pros	Frialit-2/ GB, AE	NSUB	104	124	–	–	–	5 to 6 y	97.0 (5.6 y)	–
Goldstein et al (2002)[61]	Pros	Brånemark/ MF and 3i/ MF	SUB	38	47	–	–	–	1 to 5 y (39.4 mo)	100	–

When not reported, calculations for survival rates were derived from data contained in the original paper.
*Includes data on late implant placement.
†Indicates total number of patients for all groups.
‡Indicates survival rates for immediate and delayed placement were combined.
Surfaces: MF = machined; TPS = titanium plasma-spray coated; HA = hydroxyapatite-coated; GB = grit-blasted; AE = acid-etched
Healing protocol: SUB = submerged healing; NSUB = nonsubmerged healing
CSR = cumulative survival rate derived from life-table analysis.
Pros = prospective; Retro = retrospective; NG = not given; NS = not stated

review.[12,13] Eleven studies reported on the survival rates of immediately placed implants, with mean observation periods ranging from 1 to 5.6 years.[11,51–53,56,61,63–67] One study reported on survival rates of delayed implants following an observation period of 12 months.[54]

Six reports were identified as comparative studies. One study compared immediate with late implant placement after a mean of 16 months.[50] The remaining 5 studies compared survival rates between immediate and delayed implants[10,13,22,24] and between immediate, delayed, and late placement[47] with observation periods of 1 to 5 years. No statistical differences in survival rates for immediate, delayed, and late placement techniques were reported in the comparative data. Most reports were of short duration, with only 4 studies presenting cumulative survival data on mean follow-up periods of 3 to 5 years.[13,48,61,63]

When grouped according to implant surface characteristics, there were 3 studies of hydroxyapatite-coated implants (610 implants; survival rates of 96.1% to 100%), 8 studies of machined-surface implants (620 implants; survival rates of 93.6% to 100%), 2 studies of titanium plasma spray-coated implants (130 implants; survival rate of 100%), 1 study of grit-blasted/acid-etched implants (124 implants; survival rate of 97.0%), and 4 studies of mixed surfaces (496 implants; survival rates of 89.3% to 99.4%). In general, the trend suggested that immediate and delayed implants had similar short-term survival rates and that these survival rates were comparable to rates for conventional placement in healed ridges.

There were no reports of the long-term clinical success of immediate or delayed implants. To make a comprehensive assessment of the clinical success of immediate and delayed implants, additional parameters are required that describe the health of the peri-implant tissues, function of the prosthetic reconstruction, and esthetic results. Therefore, the long-term success of immediate and delayed implants as measured by these parameters remains undefined.

Management of Local Pathology

A number of studies have demonstrated that the survival rate of implants placed following extraction of teeth with root fractures, perforations, and combined endodontic-periodontal problems is similar to that of implants placed in healed ridges.[68–71] However, implants placed in sites where teeth have been affected by chronic periodontitis have been associated with slightly elevated failure rates.[11–13] There is currently a lack of definitive evidence regarding the effect of local pathology on the success and survival of immediate implants.

Systemic Antibiotics

In most of the studies reviewed, broad-spectrum systemic antibiotics were used in conjunction with immediate and delayed implant placement.[11,20,27,50–53,72] However, the effect of systemic antibiotics on treatment outcome is unknown; thus, controlled studies are needed.

Bone Integration of Immediate and Delayed Implants

The basic prerequisites for successful bone healing in immediate and delayed implant sites are the same as for implants placed in healed alveolar ridges. In addition, a space often exists between the surface of the implant and the socket walls that needs to be filled with bone to achieve an optimal outcome. This bone healing is dependent on stabilization of the initially formed coagulum in this space. Animal experimental studies have shown that both the distance from the bone to the implant and the surface characteristics of the implant are critical factors for stabilization of the coagulum.[73–76] Clot stabilization and bone formation may be adversely affected by lack of intact bony walls. In such situations, techniques utilizing barrier membranes and/or membrane-supporting materials have been shown to be effective in regenerating bone and allowing osseointegration to occur.[17,75]

In the intact socket, a critical component of the peri-implant defect is the size of the horizontal defect (HD), which is the longest distance in a perpendicular direction from the implant surface to the socket wall.[20] It has been demonstrated that for implants with a HD of 2 mm or less, spontaneous bone healing and osseointegration take place if the implant has a rough surface.[20,77–79]

In 2001, a well-designed study examined 96 experimental titanium plasma-sprayed mini-implants in 48 patients.[78] Half of the implants were placed into extraction sockets with HDs of 2 mm or less; the other half were placed into mature bone and served as controls. No membranes or grafts were used, and primary soft tissue closure was done. Examination of the test implants following surgical re-entry at 6 months showed complete bone fill of the previous defects. Subsequent histologic examination showed no statistically significant differences between test and control sites in the percentage of bone-to-implant contact and initial level of bone-to-implant contact between test and control sites.

HDs in excess of 2 mm have been shown to not heal predictably with bone.[20] However, it may be possible to achieve predictable bone fill in such situations by using collagen barrier membranes and implants with a sandblasted and acid-etched surface.[79] A combination of a barrier membrane and a bone graft has been shown to enhance the percentage of bone-to-implant contact in large HDs in an animal model.[74]

CLINICAL INDICATIONS

Esthetics

Although esthetics are frequently cited as a reason for immediate implant placement,[6] data are lacking on esthetic outcomes following immediate implant placement.[80,81] However, adjunctive techniques to mobilize flaps[55,58,82,83] and to augment soft tissue volume[72,84–86] for wound closure at immediate implant sites may be beneficial in achieving acceptable esthetic results. Novel techniques, including nonsubmerged immediate implant placement[53,56,63] and flapless procedures,[87,88] need further evaluation with respect to esthetic outcomes.

When implant placement is delayed for a period of time after tooth extraction, soft tissue healing may provide opportunities to maximize tissue volume to achieve proper flap adaptation and acceptable soft tissue esthetics. However, this advantage is offset by resorption of bone and loss of ridge dimensions. In one report, a delay of 3 months or more after tooth extraction in the anterior maxilla resulted in such an advanced stage of resorption that only narrow-diameter implants could be used.[65] Thus, timing of implant placement following tooth removal may be important to take advantage of soft tissue healing but without risk of losing bone volume through resorption. The data to support enhanced soft tissue esthetic outcomes with delayed implant placement are lacking.

Augmentation Procedures

Several reports have shown that bone augmentation techniques may not be required where the distance between the implant body and bony wall is less than 2 mm.[78,89–92] If barrier membranes are used, wound dehiscence may lead to early exposure of nonresorbable membranes and reduced quality and volume of bone regeneration in the peri-implant defects.[17,18,47,60] Lower incidences of premature membrane exposure have been reported in studies using collagen membranes.[47,58]

Delaying implant placement for several weeks after tooth extraction allows time for bone regeneration to occur at the base and periphery of the socket,[32,33] thereby reducing the dimensions of the socket and avoiding the need for augmentation procedures.[54] However, the concomitant resorption of buccal bone may increase the need for augmentation buccolingually. An interesting observation was a lower incidence of wound dehiscence and membrane exposure with delayed implant placement, irrespective of the type of membrane used.[26,47]

CONCLUSIONS

There have been a number of reports on the subject of immediate and delayed implants with observation periods of 12 months or more. However, longitudinal studies with mean follow-up periods between 3 and 5 years were limited to 4 reports. Most reports were nonrandomized with respect to timing of the placement and augmentation methods used. Despite these limitations, short-term survival rates of immediate and delayed implants appear to be similar. Furthermore, survival rates for immediate and delayed implants appear comparable to those of implants placed conventionally in healed alveolar ridges. Studies of healing of immediate nonsubmerged implant sites are limited. Further examination of this protocol for placement is required.

As an alternative to immediate implant placement, delayed placement has several advantages. These include resolution of infection at the site and an increase in the area and volume of soft tissue for flap adaptation. However, these advantages are diminished by concomitant ridge resorption in the buccolingual dimension. Thus, 4 to 8 weeks appears to be the optimal period to defer implant placement to allow adequate soft tissue healing to take place without undue loss of bone volume.

Peri-implant defects associated with immediate and delayed implants have a high potential for bone regeneration. At sites with HDs of 2 mm or less, spontaneous bone regeneration and osseointegration may be expected when implants with a rough surface are used. At sites with HDs greater than 2 mm, or where one or more walls of the socket are missing, concomitant augmentation procedures with combinations of barrier membranes and bone grafts are required. No conclusions can be drawn from the available data regarding the optimal bone augmentation technique in these situations. However, if membranes are used, resorbable membranes appear to be effective and are associated with lower rates of wound dehiscence and membrane exposure than nonresorbable materials.

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