

Implant Prosthodontics in Medically Challenged Patients: The University of Toronto Experience

• Emad S. Elsubeihi, BDS, MS, Dip Prosthodontics •
• George A. Zarb, BChD, DDS, MS, MS, FRCD(C) •

A b s t r a c t

A series of prospective studies started in the mid-1980s at the University of Toronto have provided evidence of the efficacy and effectiveness of implants in the treatment of the fully and partially edentulous patients. These studies have focused primarily on treatment outcomes at the surgical and prosthodontic levels, with an overall failure rate of 7.7% over a 20-year period. Because a considerable proportion of these failures (4.2%) occurred before insertion of the prosthesis, and because osseointegration is essentially a wound-healing process, factors that interfere with healing, including systemic conditions, may contribute to implant failure. This paper reviews studies on the impact of selected systemic conditions, including osteoporosis, cardiovascular diseases, diabetes mellitus, and hypothyroidism, as well as smoking behaviour, on the success or "survival" of oral implants in patients treated in the Implant Prosthodontic Unit at the University of Toronto.

MeSH Key Words: dental implants; osseointegration/physiology; risk factors

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A series of prospective studies undertaken since the mid-1980s in the Implant Prosthodontic Unit (IPU) at the University of Toronto have provided evidence of the efficacy and effectiveness of the osseointegration technique in the treatment of completely and partially edentulous patients. Data from this patient population have revealed impressive prosthodontic treatment outcomes: of the 1852 implants placed in 464 patients between 1979 and 1999, 143 (7.7%) failed, 78 (4.2%) of them failing to osseointegrate before insertion of the prosthesis.¹ Furthermore, studies have shown that failures tended to be concentrated in a few individuals, an occurrence described in the literature as the "cluster phenomenon".² Because osseointegration is essentially a wound-healing process, these observations suggest that factors that interfere with healing may contribute to implant failure. Hence, we contend that conditions shown to adversely affect wound healing may decrease the potential for successful osseointegration.

In this article we review IPU studies on the possible impact of certain systemic conditions, including osteoporosis,

cardiovascular diseases, diabetes mellitus, and hypothyroidism, as well as smoking behaviour, on the success or "survival" of dental implants in 464 consecutively treated patients. The protocols for patient selection and treatment in these studies have been previously published.³⁻⁷

Osteoporosis

The term osteoporosis has been used loosely in the dental literature, often to imply postmenopausal osteoporosis. Both human and animal studies indicate that bone loss associated with postmenopausal osteoporosis results from an increase in bone turnover, in which the rates of both bone resorption and bone formation are increased. However, because the former exceeds the latter, the net result is bone loss. Evidence at the gene expression,⁸ cellular⁹ and tissue^{10,11} levels indicate that the rate of bone formation increases in postmenopausal osteoporosis. Furthermore, experimental evidence has shown that estrogen depletion leads to a significant loss of bone mass in the edentulous mandible but not in the dentate mandible.¹² Other experimental studies showed that reduced

Table 1 Effect of age (a marker of osteoporosis) on implant failure rate^a

	Age group; total no. of patients in group (and implant failure rate, % ^b)	
	< 50 years	≥ 50 years
Women	48 (18.8%)	45 (22.2%)
Men	18 (11.1%)	18 (22.2%)

^aTable courtesy of Dao and others.¹⁹

^bFailure rate is based on the number of patients in each age group with implants, in contrast to Tables 3 to 5, where the failure rate is based on the total number of implants; consequently, the failure rates here appear higher than the failure rates in Tables 3 to 5. The failure rates were not significantly different between men and women 50 years of age or older. Likewise, no significant difference was detected between women less than 50 years of age and those 50 or older.

masticatory function resulted in a reduction of mandibular bone mass associated with reduced cortical thickness¹³ and reduction in mineral apposition rates¹⁴ consistent with findings from osteoporosis associated with immobilization (unloading).¹⁵ It is not known, however, if mandibular bone loss observed in the edentulous mandible in association with estrogen deficiency¹² results from increased bone resorption alone or the combined effect of increased bone resorption and reduction in bone formation rate.

Interestingly, biochemical analysis of bone derived from the human osteoporotic femoral head showed evidence of overhydroxylation of lysine and a consequent reduction in the stabilizing cross-links of the collagenous framework, which has been suggested to contribute to increased fragility of bone.¹⁶ Indeed, mechanical testing of healing femoral fractures in rats indicated that ovariectomy impairs fracture healing for up to 4 weeks after fracture. Healing returned to normal 6 weeks after fracture.¹⁷ Furthermore, when 17- β -estradiol was administered during the period of fracture repair, there was a dose-dependent increase in the peak force required to re-break the fracture.¹⁸ The impact of such findings on the bone-implant interface is not known.

In investigating the effect of osteoporosis on the success of dental implants, Dao and others¹⁹ examined data from 93 women and 36 men treated in the IPU. The authors used the Smith and Zarb²⁰ criteria of success and compared implant failure between women and men 50 years of age or older and between women less than 50 years of age and those 50 years of age or older. The study design was based on the assumption that because osteoporosis is more prevalent in women at least 50 years of age (i.e., subjects at risk for osteoporosis), the frequency of failure would be greater in this patient group than in other groups. Patients were followed for 2 to 11 years, and failure rates were analyzed according to the number of patients with implants (not the total number of implants) (Table 1). Dao and others¹⁹ concluded that patients at risk for osteoporosis did not appear to be at greater risk for implant failure. This

Table 2 Effect of cardiovascular diseases (CVD) on implant failure rate^{a,b}

	No. of patients	No. (and %) of patients with failed implants
Patients with CVDs of interest ^c	39	5 (13)
Control groups Healthy patients	98	12 (12)
Patients with systemic disease including CVDs not of interest ^d	109	15 (14)

^aTable courtesy of Khadivi and others.²³

^bFailure rate is based on the number of patients in each age group with implants, in contrast to Tables 3 to 5, where the failure rate is based on the total number of implants; consequently, the failure rates here appear higher than the failure rates in Tables 3 to 5.

^cCVDs of interest included hypertension, atherosclerosis, vascular stenosis, coronary artery disease and congestive heart failure.

^dCVDs not of interest include dysrhythmia and heart murmur.

observation led us to persist in our clinical judgement that a clinical diagnosis of osteoporosis should not preclude a prescription for implant prosthodontics. Subsequent studies^{21,22} have supported this premise. The specific role of osteoporotic bone in the interfacial osteogenesis upon which successful osseointegration depends is incompletely understood.

Cardiovascular Diseases

Certain cardiovascular diseases, including hypertension, arteriosclerosis, vascular stenosis, coronary artery disease, and congestive heart failure, can compromise blood flow and reduce oxygen tension and nutrient supply to tissues. They might therefore be expected to compromise the outcome of osseointegration.

In a retrospective study of 246 consecutively treated patients (153 women and 93 men) Khadivi and others²³ investigated the impact of these diseases on the outcome of osseointegration at stage II surgery using the Smith and Zarb²⁰ criteria of success. The prevalence of cardiovascular diseases in the examined population was 23.9%. There was no significant difference in the rate of implant failure between patients with cardiovascular diseases (13%) and the control population (12%) (Table 2). The authors concluded that patients with controlled cardiovascular diseases are not at higher risk of osseointegration failure than patients without such conditions.

Diabetes Mellitus

Diabetes mellitus is a common metabolic disorder affecting 4% of the Canadian population.²⁴ Diabetic patients have a wide range of defects that delay the healing process and that increase their susceptibility to infection.^{25,26} Furthermore, the prevalence of osteopenia among patients with diabetes tends to be greater than among the general population; this

Table 3 Effect of medically controlled diabetes mellitus on implant failure rate^{a,b}

	Total no. of implants	No. (and %) of failed implants
Diabetic group	59	4 (7)
Control group	111	7 (6)

^aTable courtesy of Accursi.²⁷^bImplant failure rate is based on the total number of implants. Failure was assessed according to the criteria of Zarb and Albrektsson.²⁸

difference may be related to hyperglycemia in the former group. It has been reported that long-term bone loss is more severe among patients with type 1 diabetes than among those with type 2 diabetes and that bone mineral density in patients with type 1 diabetes is at least 10% lower than among sex- and age-matched healthy people.²⁷ Studies performed with untreated type 1 diabetic animal models showed evidence of lower numbers of osteoblasts, less osteoid surface, and lower plasma osteocalcin levels, consistent with decreased rates of bone formation.^{28,29}

Accursi³⁰ examined the impact of diabetes on the success of dental implants in patients who were followed for 1 to 17 years. In that study each of 15 diabetic patients (representing 3.9% of the patient population at the IPU) was matched to 2 control subjects by age, sex, location of implants, type of prosthetic restoration, opposing dentition, and duration of edentulism. A total of 59 implants in the diabetic group were compared with 111 implants in the control group according to the Zarb and Albrektsson³¹ criteria of success. The diabetic patients were no more likely to experience implant failure than the nondiabetic patients (Table 3). In assessing changes in crestal bone levels around the implants, the researchers found that diabetic patients had greater loss of crestal bone during the first year of implant loading (mean \pm standard deviation 0.25 \pm 0.07 mm) than nondiabetic controls (0.06 \pm 0.03 mm). However, this difference disappeared in subsequent years, when loss of crestal bone among diabetic patients matched that for nondiabetic patients. Accursi³⁰ also reported interesting soft-tissue and neurological complications in the diabetic group. He found that soft-tissue complications were similar in number in the diabetic and control groups; in both groups, these complications were mainly of a minor nature (including redness, bleeding and minor swelling) and resolved with improvement in oral hygiene. Conversely, the incidence of paresthesia among diabetic patients was higher than among nondiabetic controls, and the diabetic patients reported less postoperative pain. These findings suggested that controlled diabetic patients are not at higher risk of implant failure.

Table 4 Effect of medically controlled hypothyroidism on implant failure rate^{a,b}

	Total no. of implants	No. (and %) of failed implants
Hypothyroid group	82	3 (4)
Control group	81	2 (2)

^aTable courtesy of Attard.³³^bImplant failure rate is based on the total number of implants. Failure was assessed according to the criteria of Zarb and Albrektsson.²⁸ There was no statistically significant difference in implant failure rate between the hypothyroid and control groups.

Hypothyroidism

Hypothyroidism decreases recruitment, maturation and activity of bone cells, leading to decreased bone resorption and formation.³² Thyroid hormone exerts a direct effect on bone to increase production of both insulin-like growth factor-I (IGF-I) and IGF binding protein II.³³ IGF-I increases the number of osteoblasts, enhances osteoblast differentiation and increases bone remodelling, but the levels of circulating IGF-I are decreased in hypothyroidism.³⁴ Experimental evidence³⁵ has suggested that hypothyroidism may inhibit fracture healing and impair the mechanical properties of fracture callus, which indicates that thyroid hormone is a critical factor in fracture healing.

Attard³⁶ investigated the survival of dental implants in hypothyroid patients receiving thyroid hormone replacement therapy. A total of 27 patients with hypothyroidism were matched with a control group by age, sex, location of implants, type of prosthesis and opposing dentition. The results for 82 implants in medically hypothyroid patients were compared with those for 81 implants in the control group (Table 4); the implants had been in place for 1 to 20 years. There was no statistical difference in the rate of implant failure between the 2 groups. However, analysis of marginal bone around the implants revealed that there was more loss of marginal bone in the first year of loading in the hypothyroid group. This bone loss seemed to slow down in subsequent years, approaching that of the control group. The results suggest that medically controlled hypothyroid patients are not at higher risk of implant failure than matched controls.

Smoking

Cigarette smoking impairs soft-tissue wound healing by affecting the circulatory and immune systems and by impairing normal cellular function. Furthermore, it appears that cigarette smoking during adulthood is associated with decreased hip³⁷ and vertebral³⁸ bone density later in life among both women and men. In a study of 41 pairs of twins, those who smoked more heavily had bone mineral density values 5% and 10% lower at the femoral neck and lumbar spine, respectively, for each 20 pack-year difference.³⁹ The exact mechanism by which smoking exerts its

Table 5 Effect of smoking on implant failure rate (in relation to number of implants)^a

	Total no. of implants	No. (and %) of failed implants	
		Early failure	Late failure
Stage I surgery			
Smokers	494	6 (1.2) ^b	10 (2.0)
Nonsmokers	1045	32 (3.1)	35 (3.3)
Smoking history			
Positive	860	38 (4.4)	32 (3.7) ^b
Negative	679	20 (2.9)	13 (1.9)

^aTable courtesy of Habsha.¹

^bP < 0.05.

negative effect on bone is not yet fully understood. Bone loss occurs if there is an imbalance between the amount of bone resorbed and the amount of bone formed. The evidence available examining whether one or both of these mechanisms contribute to the bone loss associated with smoking is limited. Hopper and Seeman³⁹ demonstrated that lower bone density at the lumbar spine in smokers was associated with higher serum calcium and urine pyridinoline levels, which would be consistent with increased bone resorption. Furthermore, it was suggested that increased bone resorption associated with smoking is, in part, due to decreased production and accelerated degradation of estrogen, which leads to early menopause and higher rate of bone loss. However, histomorphometric investigations⁴⁰ suggested that a reduction in bone formation is responsible for the deficit in bone volume seen in smokers. In vitro studies using rat bone marrow cell cultures showed that aryl hydrocarbons, environmental contaminants occurring at high levels in cigarette smoke, inhibit osteodifferentiation and osteogenesis.⁴¹ Furthermore, in vivo animal studies have shown that nicotine impairs bone healing.⁴²

Habsha¹ studied the survival of dental implants in relation to smoking history in 464 consecutively treated patients who had had their implants for 1 to 20 years. Initially, patients were grouped on the basis of whether they were smokers or nonsmokers. Smokers were defined as those who smoked at the time of implant placement (stage I surgery). Nonsmokers were defined as those who had never smoked or who had quit smoking before implant placement. Smokers had a higher rate of early implant failure than nonsmokers (Table 5). Patients were then grouped on the basis of their smoking history, where smoking history takes into account the quantity of cigarettes consumed and the number of years during which they were consumed. Two groups were compared: a control group of patients with negative smoking history, which included individuals who had never smoked or who had smoked no more than 25 cigarette-years until stage II surgery (where a

Table 6 Effect of smoking on implant failure rate (in relation to number of patients)^a

	No. of patients	No. (and %) of patients with failed implants	
		Early failure	Late failure
Stage I surgery			
Smokers	104	18 (17.3)	8 (7.7)
Nonsmokers	285	24 (8.4)	21 (7.4)
Smoking history			
Positive	192	27 (14.1)	19 (9.9)
Negative	197	15 (7.6)	10 (5.1)

^aTable courtesy of Habsha.¹

cigarette-year is the product of the number of cigarettes smoked per day and the number of years of smoking) and patients with a positive smoking history, which encompassed those with a smoking history of more than 25 cigarette-years. Although there were no significant differences in the rate of early implant failure between the groups, those with a positive smoking history had a significant prevalence of late implant failure (Table 5). Relative risks were calculated, and it was concluded that among patients who smoked during the initial healing phase the incidence of implant failure was 1.69 times greater than among those who did not smoke. Furthermore, patients with a significant smoking history (more than 25 years) had 1.91 times the risk of late implant failure than those who did not smoke.

In an analysis of implant failure according to the number of patients, it was found that a higher proportion of smokers sustained implant failure than nonsmokers (Table 6). This indicates that the failures were not clustered within individuals, as had previously been suggested.²

Conclusions and Future Research Directions

Data from patients treated in the IPU at the University of Toronto suggest that patients at risk for osteoporosis and those with cardiovascular diseases, controlled diabetes and hypothyroidism are not at greater risk of implant failure. However, patients who smoke are at greater risk. Although these results appear reassuring, it should be emphasized that the strength of these studies was limited by their retrospective design and small sample sizes. The effects of systemic conditions on bone changes in the jaws are not fully understood. Clearly, animal models are convenient research tools to investigate these changes and the impact of systemic diseases on the healing behaviour of osseointegrated implants. Although several attempts to address these concerns have been reported, important considerations such as appropriateness of the models have been overlooked. Furthermore, as more evidence is presented to

support the notion of heterogeneity of the skeleton, it is important that healing of implants be investigated in relevant sites, specifically the mandible and maxilla. The subsequent loaded and time-dependent integrity of the osseointegrated response must also be investigated to avoid inadequate interpretations of the pathogenesis of implant failure. A lack of scientific rigour in the reporting of the long-term outcomes of implant prosthodontic treatment has led to simplistic and possibly incorrect comparisons of implant failure to processes resembling periodontal disease. The fundamental difference between the developmental nature of a periodontal ligament and the healing response elicited in the osseointegration protocol is all too frequently overlooked, and much clinical confusion has resulted.⁴³ A basic understanding of the nature of the interfacial healing response of osseointegration, particularly in the context of relative or adverse occlusal overloading, is more likely to affect the dental profession's collective understanding of why osseointegration sometimes fails.

At this stage of our understanding of the nature of the osseointegration response, it appears prudent to conclude that implant failure is most likely multifactorial. The studies reviewed here attempted to control for confounding factors such as age, sex, time since implantation, implant location, bone quality, opposing dentition, medical conditions and smoking habits. Even when such variables are controlled for, studies may document the presence or absence of associations but do not prove causality. Proving causality usually requires a randomized intervention study, the design and performance of which are usually formidable. Experimental studies, which are easier to design and which allow for better control of confounding factors, as well as larger multicentre clinical studies, are clearly needed to further elucidate the causes and mechanisms of implant failure. ♦

Dr. Elsubeihi is a PhD student at the faculty of dentistry, University of Toronto, Toronto, Ontario.

Dr. Zarb is a professor and is head, prosthodontics, department of clinical sciences, faculty of dentistry, University of Toronto.

Correspondence to: Dr. Emad S. Elsubeihi, Department of Clinical Sciences, Faculty of Dentistry, University of Toronto, 124 Edward St., Toronto, ON M5G 1G6. E-mail: emad.elsubeihi@utoronto.ca.

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