Microleakage at the Abutment-Implant Interface of Osseointegrated Implants: A Comparative Study

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Microleakage can occur at the abutment-implant (A-I) interface in osseointegrated implants and may cause malodor and inflammation of peri-implant tissues. The degree of microleakage at the A-I interface of 5 implant systems was comparatively assessed at varying closing torques. Using colored tracing probes driven by a 2-atm pressure system, the interface microleakage of Brånemark, Sulzer Calcitek, 3i, ITI, and Steri-Oss implants was determined spectrophotometrically. Microleakage through the A-I interface occurred in all systems, with variability between systems, samples, and closing torques. As closing torque increased from 10 Ncm to 20 Ncm to manufacturers' recommended closing torques, microleakage decreased significantly ($P < .005$) for all systems. Analysis of variance showed significant interaction between closing torques and the time course of microleakage, and between systems and the time course of microleakage ($P < .001$). The results indicate that fluids and small molecules are capable of passing through the interface of all the A-I assemblies studied. Presumably in an in situ situation, fluids containing bacterial byproducts and nutrients required for bacterial growth may pass through the interface gap, contributing in part to clinically observed malodor and peri-implantitis.
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Key words: closing torque, implant, microleakage, preload, screw loosening

Peri-implant pathology has been described as "peri-implant mucositis" with reversible inflammatory soft tissue reactions, and peri-implantitis has been described as inflammatory reactions with loss of supporting bone in the tissue surrounding a functioning implant.[1] Poor oral hygiene around implants has shown a direct relationship between bacterial plaque accumulation and peri-implant mucositis in humans.[2] Progressive peri-implantitis and attachment loss have been documented in humans over a 6-month period with a significant difference in marginal plaque between stable and affected implants.[3] Implants with peri-implantitis in humans have been found to harbor a complex microflora of conventional periodontal pathogens.[4–6] Bacterial plaque around subgingival ligatures causes peri-implant mucositis and peri-implantitis in beagle and labrador dogs and cynomolgus monkeys.[7–11]

Clinical studies have demonstrated the presence of viable bacteria in the internal part of functioning Brånemark (Nobel Biocare, Göteborg, Sweden) implants.[12,13] In vitro studies have shown fluid and bacterial leakage into abutment-implant (A-I) assemblies of Brånemark implants with "tightly secured" standard abutments.[14,15] Thus, it is probable that the entire inner Brånemark implant space is contaminated and may be the source of clinically observed malodor, peri-implant mucositis, and peri-implantitis.[12–15]

In dogs, a zone of inflammatory cell infiltrate was consistently present in the connective tissue adjacent to the A-I interface in Brånemark implants.[16,17] This has been termed the *abutment*

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Table 1 Characteristics of Implants and Abutments Tested

Implant system	Implant type	Diameter (mm)	Abutment interface	Abutment type	Recommended closing torque (Ncm)
Spline (Sulzer Calcitek)	Cylinder	4.0	Spline	Fixed abutment	28
CeraOne (Nobel Biocare)	Screw	3.75	External hex	CeraOne abutment	32
Steri-Oss	Cylinder	3.8	External hex	H1 straight abutment	35
3i	Screw	4.0	External hex	Abutment post	20
ITI	Screw	4.0	Morse taper	6-degree fixed partial prosthesis abutment	35

infiltrated connective tissue (abutment ICT) and is distinct from the marginal plaque associated infiltrate.[7] It has been suggested that the interface inflammatory infiltrate is related to microorganisms in the inner implant spaces.[17]

It is postulated that in all commercial screw-retained A-I assemblies, a potential microscopic space exists at the A-I interface, along the abutment screw threads, and at the base of the screw chamber. This inner implant space may facilitate microleakage of fluids and macromolecules originating from crevicular fluid and/or saliva. The purpose of this study was to comparatively assess the degree of fluid microleakage at the A-I interface of 5 commercially available implant systems at varying closing torques.

Materials and Methods

Systems Studied. Five implant systems were selected for the study: Spline (Sulzer Calcitek, Carlsbad, CA), ITI (Straumann, Waldenburg, Switzerland), CeraOne (Nobel Biocare), Steri-Oss (Steri-Oss, Yorba Linda, CA), and 3i (Implant Innovations, West Palm Beach, FL). Commercially packaged implants and abutments were used. At least 3 A-I assemblies were studied for each system. Abutments were for cement-retained superstructures, with each abutment engaging the appropriate antirotational interface geometry, except ITI, which uses a Morse taper interface. The systems and components used are listed in Table 1.

Preparation of Implants. Each implant was sectioned at the apical aspect, and a channel was prepared from the apical cut to the base of the screw chamber within the implant. Tungsten and diamond burs were used with copious irrigation, and care was taken not to heat the implant, not to introduce metal microparticles into the screw threads, and not to damage the screw chamber base.

Prior to taking each set of consecutive measurements, the individual implants, abutments, and coping screws were separately cleaned with compressed steam for 5 minutes.

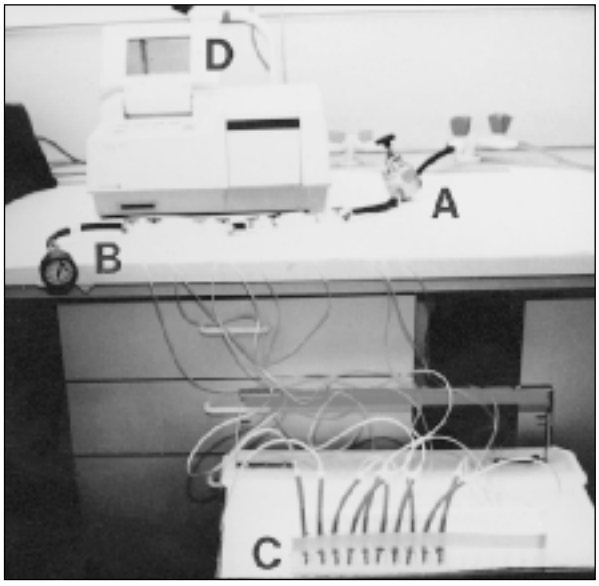

Fig 1 Experimental model: 2 atm are applied through a manifold regulated by a pressure valve (A) and a pressure gauge (B) into the sectioned bases of A-I complexes suspended in distilled water vials (C). Leakage of gentian violet dye through the interface is measured using a spectrophotometer (D).

Closing Torque Procedures. Closing torques of 10 Ncm, 20 Ncm, and manufacturers' recommended values were used. Closures of 10 Ncm and 20 Ncm represent normal (habitual) and maximal possible manual closing torques, respectively.[18] Manufacturers' recommended closing torques are shown in Table 1. Each implant was clamped into a Tonichi Torque Gauge (Pneumatic torque screwdriver, torque gauge model, TG Tonichi, Tokyo, Japan). Appropriate manual torque drivers and wrenches were used to close the abutment screws at corresponding closing torques. Torque was applied for 5 seconds for each closure.

Experimental Model. The experimental model is shown in Fig 1. The system is designed to intro-

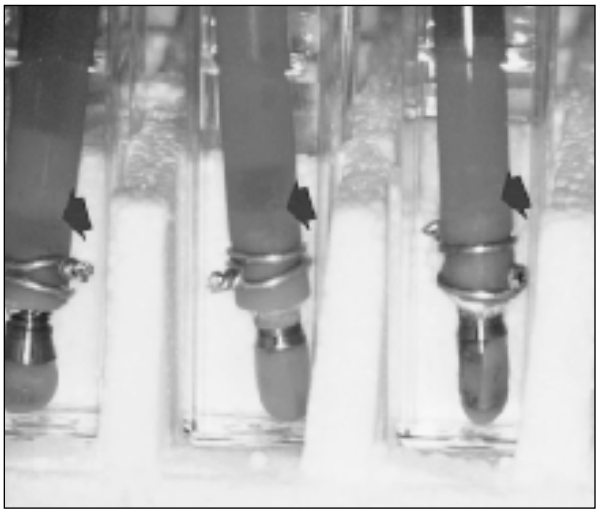

Fig 2a Implants in primary silicone tubes suspended in distilled water with occlusal and tube implant junctions sealed. Arrows indicate site of tracing dye introduced by negative pressure.

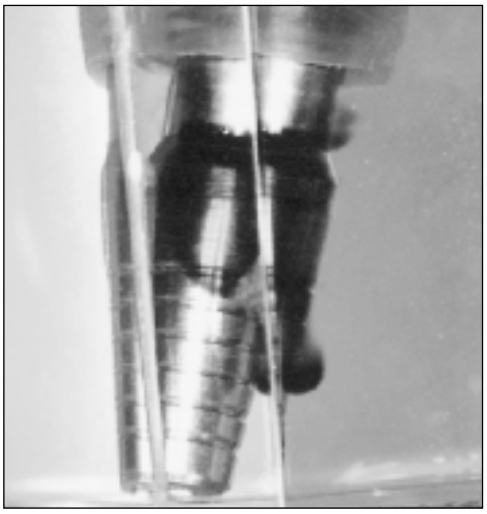

Fig 2b Dye leakage via the interface between implant and abutment.

duce controlled air pressure of 2 atm to a series of silicone tubes, which contain a low molecular weight tracing dye, that lead into the base of the sectioned implants. Constant pressure was achieved with a pressure regulator and a pressure gauge. Pressure was used to avoid air entrapment, which would impede passive fluid passage through the interface gap, where leakage may occur. This also facilitated comparison between systems.

After setting up the experimental model, components were cleaned as described above and each A-I assembly was closed at the appropriate torque. Implant bases were wrapped with 3 layers of plumber's isolation tape (PTFE) and inserted into a primary silicone tube, and the tube was sealed with soft wire ligatures. The implant-silicone margins and occlusal access holes were sealed with sticky wax (Fig 2a). Assemblies were connected to the pressure delivery system, suspended in distilled water, examined for air leakage for 1 minute, and resealed when necessary.

Particle-free gentian violet (molecular weight 408) was diluted with distilled water, and 500 µL were introduced by negative pressure (to remove trapped air) into each primary silicone tube (Fig 2a, *arrows*). Each tube was connected to the air pressure delivery system. Groups of 9 A-I assemblies were tested and recorded simultaneously.

Sequence of Measurement. Abutment-implant assemblies were suspended in 4 mL of distilled water in transparent primary vials (Elkay Ultra-Vu disposable cuvettes, Elkay, Shrewsbury, MA). A-I assemblies remained for 30 minutes without air pressure as passive initial controls. Using a micropipette (Glison Medical Electronics, Villiers-le-Bel, France), 0.6 mL was transferred from each primary vial to secondary microvials (Elkay Ultra-Vu disposable cuvettes) and the absorbance at 465 nm was measured (UV 1202 UV-VIS spectrophotometer, Shimadzu, Tokyo, Japan). These readings constituted initial passive controls and served as the baseline measurement for each vial and A-I assembly. The contents of the microvial were returned to the original primary vial and the air pressure system was activated. Pilot experiments showed total leakage after 140 minutes in some A-I assemblies at 10 or 20 Ncm; therefore, readings were taken at intervals of 5, 20, and 80 minutes from the time of initial pressure activation. Prior to each measurement, the contents of the primary vial were mixed to ensure homogeneous dye distribution and 0.6 mL was transferred to the original secondary microvial for spectrophotometric readings. Microvial contents were returned to the original primary vial after each reading and the same microvial was used for each A-I assembly. All values were calculated with the initial passive control reading as the baseline for each A-I assembly.

An example of dye leakage through the A-I cervical interface is shown in Fig 2b. A schematic diagram of sections through the 5 implant systems is shown in Fig 3. Abutment-implant interfaces through which fluid passage was measured are illustrated. To further ensure that the measured tracing dye leaked only from the A-I interface, all samples were retested at the end of each experi-

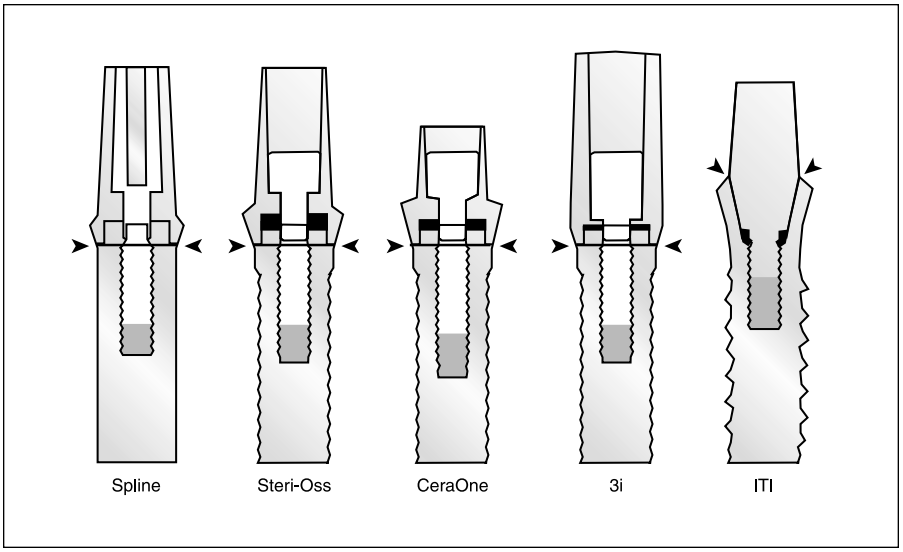

Fig 3 Schematic diagram representing sections studied through the 5 implants. Arrows indicate abutment-implant interface through which fluid passage was measured.

Table 2 Fluid Microleakage through A-I Interface of 5 Systems

Closing torque	Test (min)	Mean amount of microleakage through A-I interface*				
		Spline	CeraOne	3i	ITI	Steri-Oss
10 Ncm	5	0.065 ± 0.062	0.036 ± 0.021	0.04 ± 0.042	0.069 ± 0.076	1.034 ± 1.41
10 Ncm	20	0.084 ± 0.068	0.067 ± 0.012	0.10 ± 0.052	0.21 ± 0.18	1.022 ± 1.406
10 Ncm	80	0.113 ± 0.058	0.122 ± 0.034	0.102 ± 0.048	0.45 ± 0.331	1.021 ± 1.407
20 Ncm	5	0.016 ± 0.01	0.021 ± 0.019	0.01 ± 0.05	0.03 ± 0.0267	0.541 ± 1.212
20 Ncm	20	0.034 ± 0.025	0.042 ± 0.019	0.016 ± 0.003	0.071 ± 0.028	0.633 ± 1.222
20 Ncm	80	0.064 ± 0.045	0.07 ± 0.027	0.024 ± 0.015	0.15 ± 0.055	0.734 ± 1.282
Recommended	5	0.013 ± 0.005	0.019 ± 0.009	0.009 ± 0.005	0.029 ± 0.01	0.014 ± 0.013
Recommended	20	0.018 ± 0.004	0.02 ± 0.008	0.015 ± 0.007	0.067 ± 0.02	0.021 ± 0.013
Recommended	80	0.019 ± 0.005	0.024 ± 0.004	0.017 ± 0.004	0.067 ± 0.056	0.037 ± 0.029

*Dye leakage values are expressed as absorbance at 465 nm. For assemblies of Spline, CeraOne, 3i, and ITI implants, n = 3; for Steri-Oss implant assemblies, n = 7; ± denotes standard deviation.

ment by sealing the A-I interface with sticky wax and allowing the active pressure system to function for 15 minutes. No further dye leakage was observed in any of the A-I assemblies.

Data were analyzed using one-way analysis of variance (ANOVA) and ANOVA with repeated measures and square-root transformation.

Results

Mean active dye microleakage levels at 2 atm of the 5 A-I systems at 10 Ncm, 20 Ncm, and recommended closing torques are shown in Table 2. A gradual increase in active microleakage over time was observed for all samples. Since the 3 initial samples of Steri-Oss showed considerably higher mean and SD leakage values at 10 Ncm and 20 Ncm compared with the other 4 systems, the sample size was increased to 7 for this group only. The 7 assemblies also showed notably higher mean leakage values, with SD at least 10 times greater than that of CeraOne, Spline, 3i, and ITI assemblies. Statistical analysis was therefore performed separately for the latter 4 systems at 10 Ncm, 20 Ncm, and recommended closing torques (Table 3). ANOVA showed significant interaction between closing torque and the time course of dye leakage ($P < .0001$) and between systems and the time course of leakage ($P = .0004$). Microleakage decreased significantly ($P = .0013$) as the closing torque increased from 10 Ncm to 20 Ncm to manufacturers' recommended closing torque. Significant differences were observed among the systems tested ($P = .012$).

Dye microleakage values in all 5 systems at the manufacturers' recommended closing torque are shown in Fig 4. One-way ANOVA followed by pairwise *t* test indicate that the differences are significant ($P = .005$) at 20 minutes only. The microleakage of the ITI assemblies appeared to be

Table 3 ANOVA with Repeated Measures and Square-Root Transformation of the Data Presented in Table 2 for the 5 A-I Assemblies

Source	Sum of squares	Degree of freedom	Mean square	Freedom	Tail probability
Mean	5.03914	1	5.03914	365.94	0.0000
Systems	0.29189	3	0.09730	7.07	0.0123
Error	0.11016	3	0.01377		
Closing torques	0.48064	2	0.24032	10.31	0.0013
Systems and closing torques	0.03231	6	0.00538	0.23	0.9603
Error	0.37295	16	0.02331		
Time	0.29368	2	0.14684	88.95	0.0000
Time and systems	0.08020	6	0.01337	8.10	0.0004
Error	0.02641	16	0.00165		
Closing torques and time	0.08775	4	0.02194	9.38	0.0000
Closing torques, time, and systems	0.04910	12	0.00409	1.75	0.1020
Error	0.07486	32	0.00234		

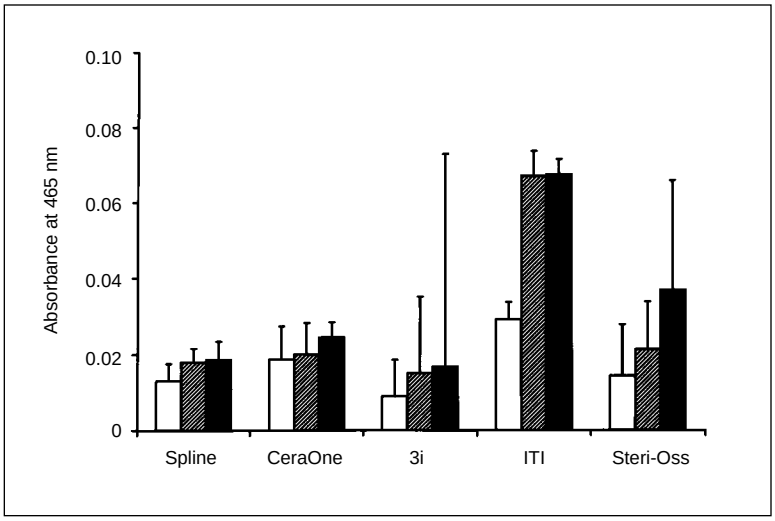

Fig 4 Mean active microleakage of 5 systems at recommended closing torques. Absorbance at 465 nm. White bar = 5 minutes; shaded bar = 20 minutes; black bar = 80 minutes; vertical line = standard deviation.

higher than that of the other 4 systems. At 5 and 80 minutes the differences in microleakage between systems were not significant.

Discussion

Active microleakage at the A-I interface was seen to be a recordable phenomenon in each assembly of the 5 systems tested in this study. The extent of leakage varied between systems, samples, and closing torques, with significantly less leakage occurring at recommended closing torques for all systems. At recommended closing torques, active microleakage was higher in the tapered mating interface as compared with flat peripheral mating rim connections regardless of the antirotational design.

Sample variability was seen in all systems, but most markedly at 10 Ncm and 20 Ncm in the Steri-Oss samples. Of these, 3 samples behaved similarly to the Spline, CeraOne, and 3i groups; 2 showed intermediate leakage; and 2 exhibited total leakage at 10 Ncm and 20 Ncm. Leakage values of all samples diminished to the minimal range at the recommended closing torque of 35 Ncm. It is postulated that this difference could be the result of the presence of initial surface asperities that were reduced at the higher closing torque. This phenomenon has been termed embedment relaxation.[19,20]

Most manufacturers now supply mechanical torque wrenches that ensure that the optimal recommended torque is reached. In a previous study,[18] 9 dentists were asked to perform manual abutment closure in 5 systems. When asked to apply normal habitual closing force with manual drivers, operators generated values of 7 to 15 Ncm, or 28 to 55% of the manufacturers' recommended values. When applying maximum possible manual closing torque, values of 9 to 20 Ncm

were recorded (31 to 75% of the manufacturers' recommended torque). Closing torques used in the present study were selected based on the above. Closure at 10 Ncm and 20 Ncm represented normal habitual and maximum possible manual closing torque, respectively.

Active pressure was used in this study to drive dye through the A-I interface to avoid errors caused by air entrapment. Leakage studies in endodontics have demonstrated that entrapped air may prevent a true demonstration of fluid transport.[21] Active pressure in conjunction with marker dyes consistently overcame this problem. This improved the demonstration of leakage, allowed comparison between systems, and was shown to correlate with passive fluid diffusion.[22]

The results of the present study suggest that the interface microgap can allow passage of fluids regardless of the implant system. Functional rocking effects and screw loosening are likely to increase such leakage,[23,24] which can be reduced by ensuring optimal component fit, minimal abutment micromovement, minimal prosthetic misfit, optimal prosthetic design and occlusion, and sustained preload.

The clinical phenomenon of bleeding and malodor characteristic of anaerobic bacteria on the removal of abutments or healing screws may be the result of, in part, the effects of microleakage. The results of this study suggest that fluids containing small molecules in the range of disaccharides and short peptides may diffuse through the interface. Presumably in an in situ situation, diffused fluids could also contain bacterial byproducts or nutrients required for bacterial growth. This scenario is thought to be related in part to the peri-implantitis and malodor observed clinically around implant abutments and to the abutment-infiltrated connective tissue reaction described in the animal experimental model.[16,17] The specific long-term effects of microleakage and its role in peri-implantitis need further evaluation. It has been suggested that this causes the initial bone loss in smooth titanium screw-shaped implants with the bone crest, 1 to 1.5 mm apical of the interface margin in the dog model.[12,16] This remains to be verified and distinguished from the suggested phenomenon of bone resorption adjacent to a smooth implant neck and compared with supracrestal interface tissue reactions.[17,25,26]

The question of whether bacteria penetrate the A-I interface in the systems used in this study is currently under investigation. Quirynen and van Steenberghe[12] demonstrated significant quantities of microorganisms in the apical portion of 18 standard Brånemark abutment screws that had been in function for 3 months. Bacterial presence was also described in Brånemark implants connected to standard abutments and EsthetiCone abutments in function from 1 to 8 years. Of the 28 implants studied, abutment screws were reported as "tightly secured" in 16 abutments (57%), "easily removed" in 5 (18%), and "loose" in 7 (25%). Original closing torques were not specified.[13] Traversky and Birek[14] demonstrated bidirectional fluid leakage and bacterial passage in vitro via the interface of "tightly secured" Brånemark implant abutment assemblies. Bacterial microleakage in vitro via the Brånemark standard abutment to the internal part of Brånemark implants with assemblies closed at 10 Ncm has also been shown by Quirynen et al.[15] In the present study, microleakage decreased significantly as abutment closing torque was increased from 10 Ncm to 20 Ncm to recommended values. It is reasonable to assume that the decrease in microleakage was the result of a tighter interface fit as closing torque increased. Therefore, bacterial interface penetration at high closing torques may not necessarily be assumed.

Microleakage is a recordable phenomenon at recommended closing torques in 5 commonly used implant systems. Its potential affects need to be addressed regarding health and pathology of the peri-implant milieu. Abutment closure at recommended closing torques could help to minimize the potential adverse effects of microleakage.

Conclusions

1. Leakage at the A-I interface is a recordable phenomenon, with variability between systems, samples, and closing torques.
2. Significant differences in microleakage were seen between 10 Ncm, 20 Ncm, and recommended closing torques for all systems, and microleakage decreased significantly as torque increased to recommended values.
3. Abutment closure at recommended closing torques may reduce potential adverse effects of microleakage.

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