Review Article

Current results and trends in platform switching

Hadi Salimi1, Omid Savabi2, Farahnaz Nejatidananesh3

1Dental Implant Research Center, 2Torabinejad Dental Research Center, 3Dental Materials Research Center, Departments of Prosthodontics, School of Dentistry, Isfahan University of Medical Sciences, Isfahan, Iran

ABSTRACT

The platform switching (PLS) concept was introduced in the literature in 2005. The biological benefits and clinical effectiveness of the PLS technique have been established by several studies. In this article different aspects of PLS concept are discussed. Crestal bone loss, biologic width, and stress distribution in this concept are comprehensively reviewed. In this article the relative published articles from 1990 to 2011 have been evaluated by electronic search. Because of controversial results especially in immediate loading and animal studies, further modified research is needed to establish the mechanism and effect of the PLS technique. Essential changes in studies including using the control group for accurate interpretation of results and long-term observation, particularly through, randomized, prospective, multicenter trials with large numbers of participants, and implants are necessary.

Key Words: Alveolar bone loss, dental implants, dental implant-abutment design, platform switching

INTRODUCTION

In 1991, 3i Implant Innovations (BIOMET 3i Inc., FL) aimed to construct wide-diameter implants with the larger diameter restorative platforms than standard implants. But, for some time, corresponding prosthetic components were unavailable; hence, standard prosthetic abutments (4.1 mm diameter) were used instead of abutments that matched the 5 and 6 mm implant diameters. The consequence of this form of treatment was an unintentional “change of platform”, which became known as platform switching (PLS).[1] This concept was introduced in the literature by Lazzara and Porter, and Gardner.[1,2]

The biological benefits and clinical effectiveness of the PLS technique have been established by several studies.[3-7] In this article different aspects of platform switching concept will be discussed. Crestal bone loss, biologic width, and stress distribution in this concept will be comprehensively reviewed.

CRESTAL BONE LOSS AND PLS

In the studies on PLS, including data with a follow-up period of 4–169 months, the documented bone loss varies between 0.05 and 1.4 mm [Table 1].[2,3,5,7-24] Crestal bone loss is a major criterion for implant success, which includes the evaluation of crestal bone level changes over time.[25-27] This has been the initial diagnostic instrument used to depict periimplant states.[28,29] Albrektsson et al.,[25] Lang et al.,[30] and Roos et al.,[31] determined that a successful implant is defined in terms of marginal bone loss around an implant restoration, with no more than 1.5 mm during the first year and no more than 0.2 mm during each succeeding year. Bone resorption around the implant neck is frequently observed after loading by a reduction in bone dimension, both horizontally and vertically,[32] and appears to depend on both biological and mechanical factors, such as surgical trauma to the periosteum,[33] characteristics of the implant neck.
### Table 1: Investigations regarding crestal bone loss of platform switching (PLS) and nonplatform switched (NPLS) implants

<table>
<thead>
<tr>
<th>Authors</th>
<th>Type of study and studied groups (implant diameter/abutment diameter)</th>
<th>No. implants (control)</th>
<th>Follow-up (months)</th>
<th>Mean±standard deviation or range of crestal bone loss (mm)*</th>
<th>Survival rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calvo-Guirado et al.,[22] 2011</td>
<td>Clinical cases (PLS)</td>
<td>64</td>
<td>70</td>
<td>M: 0.08±0.42; D: 0.14±0.56</td>
<td>97.1</td>
</tr>
<tr>
<td>Enkling et al.,[23] 2011</td>
<td>Randomized clinical trial PLS (4/3.3); NPLS (4/4)</td>
<td>50(25)</td>
<td>12</td>
<td>Test: 0.53±0.35; Control: 0.58±0.55</td>
<td>100</td>
</tr>
<tr>
<td>Canullo et al.,[24] 2011</td>
<td>Randomized clinical trial PLS group 1 (4.3/3.8); NPLS group 3 (3.8/3.8)</td>
<td>22(5)</td>
<td>36</td>
<td>Group: 1 0.83±0.44; Group: 2 0.48±0.22; Group: 3 0.37±0.12; Control: 1.35±0.39</td>
<td>100</td>
</tr>
<tr>
<td>Yun et al.,[11] 2011</td>
<td>Clinical cases (PLS)</td>
<td>79</td>
<td>7.4</td>
<td>0.16±0.08</td>
<td>100</td>
</tr>
<tr>
<td>Cocchetto et al.,[25] 2010</td>
<td>Clinical cases (PLS)</td>
<td>15</td>
<td>18</td>
<td>0.05–1.63 (range)</td>
<td>100</td>
</tr>
<tr>
<td>Canullo et al.,[26] 2010</td>
<td>Randomized clinical trial PLS group 1 (4.3/3.8); NPLS group 3 (3.8/3.8)</td>
<td>60(17)</td>
<td>33</td>
<td></td>
<td>100</td>
</tr>
<tr>
<td>Bilhan et al.,[13] 2010</td>
<td>Clinical cases (PLS)</td>
<td>126</td>
<td>36</td>
<td>M: 0.89±0.16; D: 0.91±0.17</td>
<td>100</td>
</tr>
<tr>
<td>Wagenberg and Froum[10] 2010</td>
<td>Clinical cases (PLS)</td>
<td>106</td>
<td>132-169</td>
<td>M: 0–3.2 (range); D: 0–3.6 (range)</td>
<td>88.6</td>
</tr>
<tr>
<td>Prosper et al.,[16] 2010</td>
<td>Clinical cases (PLS)</td>
<td>120</td>
<td>60</td>
<td>Immediate loading group: 1.31±0.44; Delayed loading group: 1.01±0.59</td>
<td>96.67</td>
</tr>
<tr>
<td>Rodriguez-Ciurana et al.,[19] 2009</td>
<td>Clinical cases multicenter (PLS)</td>
<td>82</td>
<td>6-24</td>
<td>Vertical: 0.62; Horizontal: 0.60</td>
<td>100</td>
</tr>
<tr>
<td>Prosper et al.,[16] 2009</td>
<td>Randomized clinical trial 1 Submerged (4.5, NPLS); 2 Submerged (4.5/3.8); 3 Nonsubmerged (4.5, NPLS); 4 Submerged (3.8, NPLS); 5 Submerged (3.8/3.3); 6 Nonsubmerged (3.8, NPLS)</td>
<td>360(180)</td>
<td>12-24</td>
<td>Group 1: M: 0.25±0.12; D: 0.36±0.18; Control M: 1.13±0.33; Control D: 1.25±0.40</td>
<td>98.3</td>
</tr>
<tr>
<td>Canullo et al.,[27] 2009</td>
<td>Test and control group PLS (5.5/3.8); NPLS (5.5/5.5)</td>
<td>22(11)</td>
<td>24-27</td>
<td>Test M: 0.25±0.12; Test D: 0.36±0.18; Control M: 1.13±0.33; Control D: 1.25±0.40</td>
<td>100</td>
</tr>
<tr>
<td>Calvo-Guirado et al.,[16] 2009</td>
<td>Clinical cases (PLS)</td>
<td>61</td>
<td>12</td>
<td>M: 0.08±0.53; D: 0.09±0.65</td>
<td>96.7</td>
</tr>
<tr>
<td>Trammel et al,[18] 2009</td>
<td>Randomized clinical trial PLS group NPLS group</td>
<td>25</td>
<td>24</td>
<td>Test: 0.99±0.53; Control: 1.19±0.58</td>
<td>100</td>
</tr>
<tr>
<td>Cappiello et al.,[28] 2008</td>
<td>Test and control group PLS (4.8/4.1); NPLS (4.1/4.1)</td>
<td>131(56)</td>
<td>12</td>
<td>Test: 0.95±0.32; Control: 1.67±0.37</td>
<td>99.2</td>
</tr>
<tr>
<td>Calvo-Guirado et al.,[7] 2008</td>
<td>Clinical cases (PLS)</td>
<td>105</td>
<td>16</td>
<td>0.6±1.0</td>
<td>99.1</td>
</tr>
<tr>
<td>Calvo-Guirado et al.,[8] 2007</td>
<td>Clinical cases (PLS)</td>
<td>10</td>
<td>1,2,3,6</td>
<td>Central incisor M: 0.05; D: 0.07; Lateral incisor M: 0.07; D: 0.06</td>
<td>100</td>
</tr>
<tr>
<td>Hurzeler et al.,[9] 2007</td>
<td>Test and control group PLS (5/4.1); NPLS (4.1/4.1)</td>
<td>22(8)</td>
<td>12</td>
<td>Test: 0.12±0.40; Control: 0.29±0.34</td>
<td>100</td>
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(Continued)
Cappiello et al.,[20] in a clinical and radiographic prospective study showed that PLS decreased bone resorption to 0.95 mm compared to 1.67 mm in the control group. The aim of the study was to evaluate the biologic effect of PLS.

Prosper et al.,[15] in the first randomized prospective study of 360 implants, compared expanded platforms versus cylindrical implants involving abutments of the same size, placed in 60 partially edentulous patients. The results showed lower amount of bone loss in the group with reduced platforms, with the preservation of up to 98.3% versus 66.1% after 12 months, and 97.2% versus 53.3% after 2 years for two groups respectively.

Several controlled clinical trials have shown that implants with PLS had significantly less bone resorption compared with traditional matching implant–abutment connection.[5,17,20] Canullo et al.[13] in a randomized controlled trial study established a relationship between the extent of PLS and the amount of marginal bone loss for the first time.

Wagenberg and Froum[9] in a prospective study evaluated implant survival, and crestal bone levels around implants that used the PLS concept and followed for a minimum of 11 years. Seventy-one of the 94 implants (75.5%) showed no bone loss on the mesial aspect, and 67 implants (71.3%) showed no bone loss on the distal aspect. This is the longest follow-up to a prospective investigation of platform-switched implants and confirms the concept for preservation of crestal bone levels.

The beneficial effect of PLS on bone loss in immediate loading or placement studies remains controversial, because most studies do not have the control group.[3,4,14,16]

Crespi et al.,[41] revealed the influence of PLS on crestal bone level changes at 1, 3, 6, 12, and 24 months. Implants with PLS (n=30), and external hexagon (n=34), were positioned immediately after tooth extraction and were loaded immediately. Results showed no differences in bone level changes between PLS, and conventional external-hexagons implants after 24 months. Canullo et al.,[21] evaluated bone level response around single, immediately placed and provisionalized PLS implants. The mean follow-up period was 25 months and the average bone resorption level in the PLS group (0.3±0.16 mm) was smaller than that in the non-PLS group (1.10±0.35 mm), and this difference was statistically significant (P<0.005).

**BIOLOGIC WIDTH AND PLS**

The periimplant soft-tissue seal comprise of a junctional epithelium and connective tissue. This biologic soft-tissue coats the implant supporting bone in a 3 to 4 mm wide zone.[42,43]

Cochran et al.,[39] and Hermann et al.,[44] were reported preimplant histometric outcomes and confirmed the presence of biologic width. This is true for any implant patients, whether on one-stage or in two-stage placement protocols on two-piece implants.[42,44,45]

Tarnow et al.,[46,47] showed that not only this width progresses apically, but also a lateral component of the biologic width exists around implants. This lateral component varies from 1.04 mm when two adjoining implants are placed less than 3 mm apart to 0.45 mm when the implants are placed more than 3 mm apart.

Inhibition of bone resorption is an important factor in achieving good esthetic results in the maxillary

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**Table 1: (Continued)**

<table>
<thead>
<tr>
<th>Authors</th>
<th>Type of study and studied groups (implant diameter/abutment diameter)</th>
<th>No. implants (control)</th>
<th>Follow-up (months)</th>
<th>Mean±standard deviation or range of crestal bone loss (mm)*</th>
<th>Survival rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Canullo and Rasperini[14] 2007</td>
<td>Clinical cases (PLS)</td>
<td>10</td>
<td>22</td>
<td>0.78±0.36</td>
<td>100</td>
</tr>
<tr>
<td>Vela-Nebot et al.,[17] 2006</td>
<td>Test and control group</td>
<td>60(30)</td>
<td>6</td>
<td>Test M: 0.76±0.19</td>
<td></td>
</tr>
<tr>
<td></td>
<td>PLS (5/4.1)</td>
<td></td>
<td></td>
<td>Test D: 0.77±0.19</td>
<td></td>
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<tr>
<td></td>
<td>NPLS (5/5)</td>
<td></td>
<td></td>
<td>Control M: 2.53±0.23</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Control D: 2.56±0.33</td>
<td></td>
</tr>
<tr>
<td>Gardner[2] 2005</td>
<td>Clinical cases (PLS)</td>
<td>1</td>
<td>4</td>
<td>1.3-1.4</td>
<td>No data</td>
</tr>
</tbody>
</table>

*M: Mesial, D: Distal
aesthetic zone and providing sufficient bone to support the implants. Vertical bone resorption, which often extends 1–2 mm below the implant–abutment interface, may lead to a possible biomechanical disadvantage by increasing stress values at the bone–implant interface.\(^{[44,45]}\) Horizontal bone loss may cause resorption of the buccal plate, loss of the interproximal bone peak, and loss of support for the adjacent interimplant papilla.\(^{[47]}\)

Results from Lazzara and Porter\(^{[1]}\) showed the history and importance of the microgap and the reconstruction of a biologic width around dental implants.

The saucerization procedure leads to wound healing by means of lack of bone apposition, and fibrous scar tissue formation. This zone of connective tissue is infiltrated by chronic inflammatory cells and is always present around the implant–abutment junction (IAJ) of two-piece implant systems.\(^{[20]}\) Ericsson et al.\(^{[38,48]}\) have identified an inflammatory connective tissue (ICT) zone infiltrate in the junctional epithelium of the periimplant mucosa. This inflammatory zone developed vertically for about 0.5–0.75 mm coronal and 0.5–0.75 mm apical to the implant–abutment junction (IAJ). An approximately 1 mm wide layer of healthy connective tissue separates the ICT from bone.\(^{[49]}\) This tissue provides protection and reinforcement of the crestal bone, by prevention the passage of microorganisms.\(^{[43]}\)

In an experimental study in dog, the thickness of this mucosal seal was approximately 3 mm and intentional reduction of this protective layer to 2 mm or less lead to greater crestal bone loss.\(^{[42]}\)

Luongo et al.\(^{[49]}\) reported that at the IAJ, it was possible to clearly distinguish a zone of ICT infiltrate in which the presence of plasma cells and lymphocytes was detectible. A noteworthy finding was that this inflammatory infiltrate extended vertically for 0.35 mm coronal and the IAJ along the healing abutment, while in the horizontal direction, it did not exceed the length of the implant with PLS. Rodriguez-Ciurana et al.\(^{[19]}\) indicated that the biologic width around the platform-switched implants are located more coronally than the biologic width around the nonplatform switched implants.

In animal experiments Becker et al.\(^{[50,51]}\) could not differentiate the PLS and control groups statistically but concluded that PLS could prevent the apical down-growth of the barrier epithelium in 28 days.

The PLS technique causes the margin of the IAJ inward, toward the central axis of the implant. The inward movement of the IAJ is believed to shift the inflammatory cell infiltrate to the central axis of the implant and away from the adjacent crestal bone, which is thought of limiting crestal bone resorption. This may result in a reduced inflammatory effect within the surrounding soft tissue and crestal bone. Consequently, (I) a biologic width dimension forms without an increase in loss of crestal bone around dental implants; (II) the relative impact of bacterial leakage at the microgap on bone remodeling around dental implants decreases; and (III) soft-tissue levels that helps to avoid esthetic deformities, phonetic problems, and food impaction preserves.

**STRESS–STRAIN RESPONSE AND PLS**

Several studies have described methods (photoelastic analysis, strain gauge placement, finite-element analysis (FEA)) for evaluating the biomechanical advantages of the PLS.\(^{[52–59]}\) Maeda et al.\(^{[56]}\) in a FEA concluded that the PLS configuration shifted the stress concentration away from the peri-implant marginal bone but increases the stress in the abutment or the abutment screw. Canullo et al.\(^{[60]}\) performed a 3D finite element analysis on three different implant abutment configurations: A 3.8 mm implant with a matching diameter abutment (Standard Control Design, SCD), a 5.5 mm implant with matching diameter abutment (Wider Control Design, WCD), and a 5.5 mm implant with a 3.8 mm abutment (Experimental Design, ED). Their results showed that the ED configuration minimized the stresses at the implant/abutment interface region. This reduction was 160% compared to SCD and 33% compared to WCD. Pessoa et al.\(^{[61]}\) in a computed tomography-based three-dimensional FEA demonstrated that the platform-switched designs can be considered a valid treatment option, equivalent to the conventional matching diameter abutment–implant configurations.

Pellizzer et al.\(^{[59]}\) in a photoelastic analysis found no significant difference between wide-diameter and PLS implants with respect to the magnitude of stress but stress concentration decreased in the cervical region of the platform-switching implant.

Sabet et al.\(^{[57]}\) using strain gages evaluated the effect of PLS on strain developed around implants supporting mandibular overdenture. The results showed that the increasing amount of strain developed because decreasing the abutment size does not favor the use of PLS in implant-supported mandibular overdentures.
Stress concentrations on PLS implants are located at the center of the implant–abutment joint (at the level of the implant screw) will be helpful in reducing crestal bone resorption.

**REVENUE OF PLS**

Concerns exist about loss of implant papilla and exposing the metal collar at the implant shoulder in the esthetic zone.[62-67] As a consequence, the PLS technique has been developed to either preserve or regenerate the interimplant soft tissue and impede an unsightly metal display.

In a situation in which limitation of the residual bone height, poor-quality bone, and narrow edentulous sectors exist and cannot be resolved, an alternative approach, which may possibly be used to overcome these problems, is the PLS technique.

Differences in interpretation of function of the PLS technique have been noted in several studies.[11,17,44,56,68] However, clinical advantages of the PLS technique may be the cause of a reduction in the crestal bone loss and maintaining the counterpart soft tissue around the implant. Therefore, PLS can preserve soft and hard tissues and may provide better biological, mechanical, and esthetic outcomes.

**CONCLUSION**

Because of controversial results especially in immediate loading and animal studies, further modified research is needed to establish the mechanism and effect of the PLS technique. Therefore, essential changes in studies including using the control group for accurate interpretation of results and long-term observation, particularly through, randomized, prospective, multicenter trials with large numbers of participants and implants are necessary.

The four common clinical conditions requiring a selective PLS technique are as follows:

1. Where anatomic structures such as the sinus cavity, the nasal floor, the incisive canal, and the alveolar nerve limit the residual bone height.
2. Where implants must be placed less than 3 mm apart (between 1.5 and 3 mm) in narrow edentulous sectors.
3. Where using short implants and in atrophic areas.
4. When achieving good esthetic results in the anterior maxilla is more important.

**REFERENCES**


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