The effects of hydrochloric acid on all-ceramic restorative materials: an *in-vitro* study

SADJ April 2014, Vol 69 no 3 p106 - p111

A Harryparsad[1], H Dullabh[2], L Sykes[3], D Herbst[4]

ABSTRACT

Objectives: The aim of this *in-vitro* study was to assess the long-term effects of hydrochloric acid on the surface roughness of three all-ceramic restorative materials CEREC VITABLOC® Mark II CAD, IPS Empress CAD® and IPS e.max CAD®.

Materials and Methods: Six cylindrical specimens (10mm diameter, 3mm height) of each material type were prepared, using the CEREC CAD/CAM machine. The unpolished samples were immersed in 15ml hydrochloric acid (pH 2) at 370C. Before immersion (baseline) and at periods of 7.5 hours, 45 hours and 91 hours, the specimens were removed from the acid and two randomised areas (10µm X 10µm) were selected and tested on each. The atomic force microscope (Bruker Dimension icon) was used to assess surface roughness and surface area at baseline and after each exposure time. The materials were compared over time with respect to surface roughness and surface area (baseline, 1 month, 6 months, 1 year) in a repeated measures analysis of variance (ANOVA).

Results: Sample groups differed significantly for roughness ($p<0.0001$) and surface area ($p<0.0001$). For both parameters a significant interaction also existed between material and time (surface roughness: $p=0.0085$; surface area: $p=0.0014$). CEREC VITABLOC® Mark II CAD and IPS Empress CAD® had substantially higher levels of roughness and surface area than IPS e.max CAD®, which was also affected to a lesser extent over time. Conclusion: The results showed that IPS e.max CAD was least affected by long-term exposure to hydrochloric acid.

1. **A Harryparsad:** *BDS, PDD, MChD (Prosthodontics).* Senior Lecturer/Specialist, Department of Prosthodontics, University of Pretoria, South Africa.
2. **H Dullabh:** *BChD, MSc(Dent), MDent(Prosthodontics).* Head of Department, Department of Prosthodontics, University of Pretoria, South Africa.
3. **L Sykes:** *BSc, BDS, MDent (Prosthodontics).* Head Clinical Unit, Department of Prosthodontics, University of Pretoria.
4. **D Herbst:** *BChD, MChD (Prosthodontics)* Consultant, Department of Prosthodontics, University of Pretoria.

Corresponding author
A Harryparsad:
Department of Prosthodontics, School of Dentistry, University of Pretoria,
Tel: +27 012 3192446. Fax: +27 012 323 0561.
E-mail: ashana.harryparsad@up.ac.za

ACRONYMS

GERD: Gastro-oesophageal reflux disease
AFM: Atomic Force Microscope

Clinical Significance: These results can aid the clinician in choosing the appropriate materials for patients who are bulimic or who suffer from gastro oesophageal reflux.

Keywords: ceramics, erosion, hydrochloric acid, surface roughness

INTRODUCTION

Tooth surface loss can be attributed to four main factors: erosion, abrasion, attrition and abfraction. Dental erosion was first reported in the 19th century, and can be defined as "the loss of dental hard tissue through either chemical etching or dissolution by acids of non-bacterial origin or chelation."[1] While certain countries have reported a decrease in the prevalence of caries, others are experiencing an increase in erosion, making this a subject of interest amongst the dental fraternity.[1] The loss may be attributed to extrinsic or intrinsic factors.[2] The former include excessive intake of fizzy drinks, fruit juices, sports drinks, acidic foods or medicaments, misuse of drugs, over-zealous oral hygiene practices and environmental influences. Enamel begins to show signs of demineralization at a pH of 5.5, with dentine dematerializing at a higher pH.[3] The intrinsic cause of loss of tooth material is exposure to acid derived from within the body, the main source being gastric acid that is regurgitated into the oral cavity. This scenario is reportedly becoming more frequent due to the increasing prevalence of bulimia nervosa and gastro-oesophageal reflux disease (GERD).[4]

GERD is defined as a condition that develops when the reflux contents cause symptoms and complications.[5] Obtaining accurate prevalence figures is difficult, as most results are based on the assumption that heartburn or regurgitation are the only indicators of the disease.[6] However, patients may present with clinical evidence of GERD and have no such complaints of heartburn or regurgitation.[7] GERD can affect all age groups, with reported prevalence rates of 1.8-7.2 % in children (3 to 9 years old), and 3-5% in adolescents (10 to 17 years old).[8] Whilst prevalence rates of 10% to 20% have been reported in the Western world, and a less than 5% occurrence in Asia, 6, no data appears to have been derived for Africa.

Bulimia nervosa is defined as a disorder associated with excessive concern about body weight and shape, binge eating, frequent self-purging and other inappropriate behaviour to prevent weight gain.[9] It is generally a disease of secrecy and prevalence data are difficult to obtain as self-reporting is inaccurate.[10] Rates of 1-1.5% in woman have been reported,[11] with lifetime rates of 1-3%.[9] Males and children may also be bulimic. An American study on 9 to 14 year old children found 7.1% of boys and 13.4% of girls had some form of eating disorder.[12] The binging and purging episodes are individual specific. On average, bulimics binge 1.4 to 1.6 times a day and purge on average three times a day, exposing dentition and restorations to repeated episodic acid attacks.[13,14] These purging sessions can last from two to fifteen minutes and their duration is dependent on the individual.[15]

In both GERD and bulimia, the gastric acids cause softening of the tooth structure and erosion of the dental tissues leading to irreversible loss of enamel and dentine. Prolonged acid exposure results in loss of tooth lustre, appearance of shallow concavities coronal to the CEJ, cusp tips becoming rounder or cupped, edges of restorations appearing raised above the level of the adjacent tooth structure, and in severe cases there may be loss of vertical crown height.[16] There is also a decrease in the micro-hardness of enamel, which makes the tooth structure more prone to other forms of tooth-surface loss.[16] It is for this reason that clinically erosion is often accompanied by other forms of tooth wear. Clinical manifestations of this type of dental erosion may only be evident after regular acid contact several times a week for a period of at least one to two years.[17]

Dental erosion is not only dependent on the frequency and duration of vomiting or regurgitation but also on the oral hygiene practices of the patients following exposure to the acids, the degree of mineralisation, fluoride content of the tooth structures, and the quantity and quality of secreted saliva.[18] In both GERD and bulimia, the erosive lesions are located on the palatal surfaces of the maxillary teeth (perimolysis).[19,20] It is usually the incisors that are the worst affected. In cases of long-standing regular vomiting of longer than five years duration, lesions can be located on both the palatal and buccal surfaces of the teeth.[17] This makes the teeth more prone to dental caries, and to fractures, leading to the need for extensive restorative work at an earlier age.[4]

A theory has been proposed to explain the erosive patterns seen in gastro-oesophageal reflux disease (GERD).[3] In the early stages of the disease the damage is more pronounced on the palatal surfaces of the maxillary teeth. This has been attributed to the force with which the regurgitation passes from the pharynx into the mouth, which results in a first contact with the palatal surfaces of the maxillary teeth. The tongue may also aid in maintaining acid contact with the tooth surfaces. As with bulimia, saliva plays an important role in neutralising and clearing the acidic content from the oral cavity, thereby reducing its contact with the tooth surface. However, more recent studies have shown that patients with GERD also have associated saliva complications.[21,22,23] A reduction in buffering capacity,[21,22] reduced unstimulated salivary flow[21,23], and reduced saliva pH[21] are some of the complications reported. It can therefore be extrapolated that a deficit in saliva function could contribute to the severity and rate of the erosion experienced in patients with GERD and bulimia.

Advances in technology and research on indirect tooth-coloured restorative materials have led to the development of many new types of ceramics and manufacturing procedures. The all-ceramic materials are gaining popularity because of their excellent aesthetic properties in translucency and good colour match to natural teeth, to their biocompatibility, and resistance to wear. In addition, patient concerns about the possible adverse effects of metal restorations on their health have increased the demand for metal free restorations.[24-28]

Considerable differences exist between ceramics due to their different chemical composition and microstructure.[13] McLaren and Cao[29] proposed four categories, namely: glass based systems (mostly silica); glass based systems with fillers (leucite or lithium); crystalline-based systems with glass fillers (alumina); and polycrystalline solids (alumina and zirconia). All systems have both intrinsic and extrinsic factors that contribute to their overall physical performance. The intrinsic factors include: crystallinity; crystal-size geometry; modulus of elasticity; phase transformation; and thermal expansion mismatch.[30] Extrinsic factors include: the oral environment; humidity; pH; cyclic loading and peak loads.[29] The durability of ceramics in the oral environment will be influenced by their composition and microstructure as well as the acidity, length of time of exposure and temperature of the chemical agents present.[13] Exposure of ceramics to erosive agents leads to degradation of the material through selective leaching of alkaline ions leading to reduced stability, decreased flexural strength, and potential for crack development and propogation.[31] Degradation of all-ceramic materials results in a rougher surface, which will promote plaque accumulation and cause increased wear of the opposing teeth. Change in colour of the material can also occur following damage by acid exposure.

CEREC VITABLOC® Mark II CAD is composed mainly of silicon dioxide (silica or quartz) with varying amounts of alumina.[29] It is reportedly the most abrasion-resistant dental ceramic due to the small particle size (average 4μm) and the sintering process.[32] IPS Empress CAD® consists of leucite-based glass ceramic ingots that are processed using CAD/CAM technology. This material has increased strength, and reduced crack propogation.[33] IPS e.max CAD® is a lithium disilicate glass ceramic material, which has been designed for the CAD/CAM technique and has high strength and edge stability. Etching of the material results in dissolution of the glassy phase.[34]

Degradation of dental ceramics can occur via mechanical or chemical attack. Chemical degradation can result in selective leaching of alkaline metal ions from the ceramic materials.[31] This degradation leads to change in surface topography, and can result in a rougher surface. This could have side effects such as: increased abrasion of the opposing tooth structure; increased plaque retention; and a release of possible harmful concentration of elements due to abrasion or dissolution.[31,35]

Surface profilometery has been the standard method for assessing surface roughness of dental materials.[36,37] However, the development of the Atomic Force Microscope (AFM) has led to much more accurate, and less time-consuming assessment. Thus this study aimed to assess the long-term effects of HCl on the surface roughness and surface area of three all-ceramic restorative materials (CEREC VITABLOC® Mark II CAD, IPS Empress CAD® and IPS e.max CAD®) using an AFM.

MATERIALS AND METHODS

Three commonly used ceramics were selected to determine the effects of HCl on the surface roughness of each. A total number of 18 discs were prepared, six for each material type. All samples were produced in one dental laboratory using the CEREC® inLAB MC XL CAD/CAM milling unit, to assure consistent specimen size. They were milled into discs of 10mm diameter and 3mm thick. All specimens from each material type were placed in three volumetric flasks (one for each sample type) with 15ml HCl (pH 2). The flasks were placed into an incubator (Scientific Series 9000) at 37°C for the allocated times as follows: 7.5 hours (representing one month of gastric acid exposure), 45 hours (representing six months of exposure) and 91 hours (representing one year of exposure). These times were calculated on the assumption that a bulimic patient purges three times a day[29,14] for an average of five minutes per purge.[15] Therefore, on average, teeth would be exposed to gastric acid for 15 minutes a day.

After each time frame, all specimens were rinsed with distilled water and air dried, then placed in a desiccator to remove any liquid remnants that might interfere with the AFM analysis. Two randomised areas (10µm X 10µm) were selected on each sample and analysed using the AFM microscope (Bruker Dimension icon) to obtain values for mean surface roughness (Ra) and mean surface area. The AFM functions via a laser beam (Figure 1), which scans the specified dimensions (10µm X 10µm) and transfers this information to the computerized software (NanoScope Analysis). This software automatically calculated the scanning results for mean surface roughness and mean surface area, and these figures were analysed statistically. With each new exposure time frame, a fresh batch of acid (pH 2) was used and tested to ensure the correct pH was maintained.

RESULTS

The three ceramics were compared with respect to surface roughness (ηm) and surface area (µm2) over time (baseline, 1 month, 6 months and 12 months) in a repeated measures analysis of variance (ANOVA) test.

The materials differ significantly (p<0.0001) from each other for surface roughness, and materials and time also interacted significantly (p=0.0085) (Table 1 & Figure 2). Thus the differences between materials needs to be interpreted with caution. The IPS e.max CAD® had significantly lower mean surface roughness values than CEREC VITABLOC® Mark II CAD and IPS Empress CAD®. These latter two materials also do not seem to differ with respect to mean surface roughness but do display an interaction over time, particularly at 1 and 12 months.

Figure 1: Sample placed on the mounting plate for evaluation.

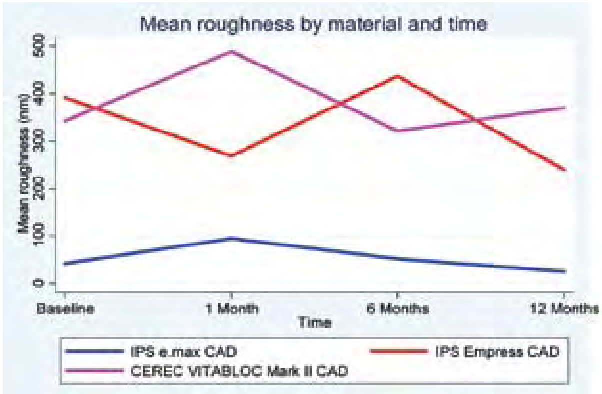

Figure 2: Mean surface roughness of material over time.

Figures 3 (A-D) represent a three-dimensional (3-D) graphic representation of the samples to visually assess the changes in surface roughness for each sample after each treatment time.

For the IPS e.max CAD® samples, mean surface roughness readings of 42.375ηm (9.967), 94.708ηm (57.938), 52.308ηm (37.319) and 25.133ηm (7.656) were obtained for baseline, 1 month, 6 month and 12 months respectively (Figure 4). It had an overall mean surface roughness of 53.631ηm (41.862) making it the best performing material tested (i.e. least amount of erosion).

For the IPS Empress CAD® samples, mean roughness readings of 391.667ηm (86.204), 268.500ηm (103.119), 437.167ηm (194.623) and 239.750ηm (124.166) were obtained at baseline, 1 month, 6 months and 12 months respectively. These results had a distinctive pattern (Figure 5), in that after the first exposure time they showed a reduction of roughness (except sample 6) followed by an increase in roughness and finally another reduction (except sample 5). Sample 5 differed in that it showed a pattern of increase

Table 1: Mean surface roughness (ηm) and standard deviation over time

Time	Materials			
	IPS e.max CAD®	IPS Empress CAD®	CEREC VITABLOC® Mark II CAD	Total
Baseline	42.375 (9.967)	391.667 (86.204)	342.333 (136.457)	258.792 (181.429)
1 month	94.708 (57.938)	268.500 (103.119)	488.333 (184.096)	283.847 (203.838)
6 months	52.308 (37.319)	437.167 (194.623)	320.917 (76.405)	270.131 (201.918)
12 months	25.133 (7.656)	239.750 (124.166)	369.817 (126.236)	211.567 (175.000)
Total	53.631 (41.862)	334.271 (150.311)	380.350 (143.242)	256.084 (188.877)

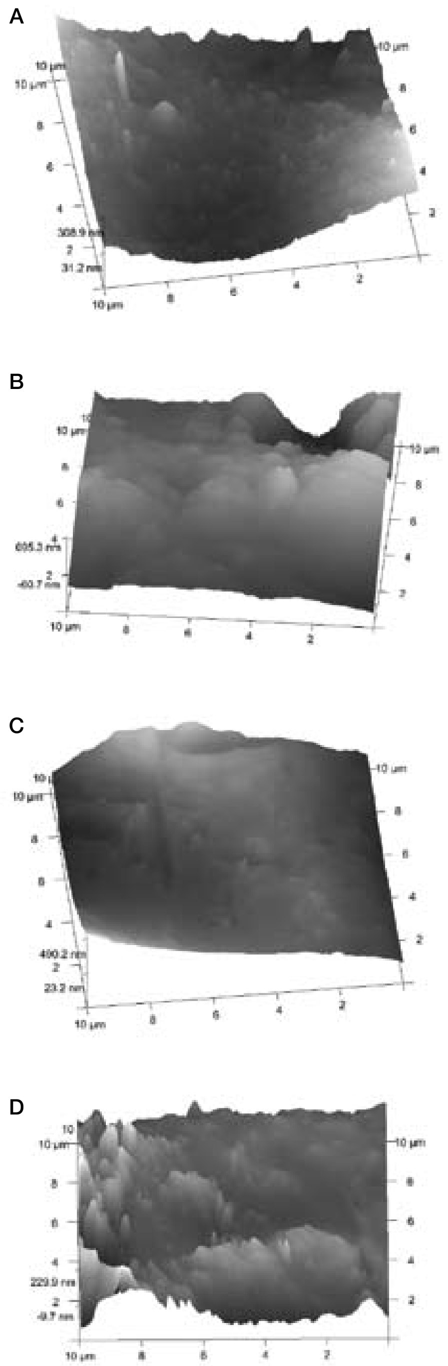

Figure 3 (A-D): 3-D graphic representation of surface morphology of the roughest IPS e.max CAD® sample at baseline **(A)**, after 7.5 hours **(B)**, after 45 hours **(C)** and after 91 hours **(D)**.

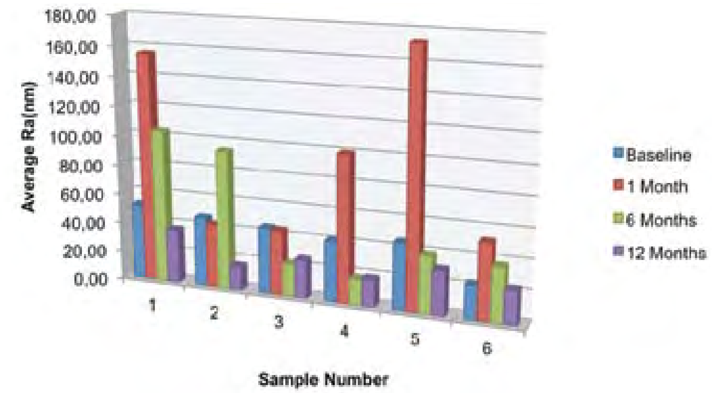

Figure 4: IPS e.max CAD® samples average surface roughness (ηm) over time tested

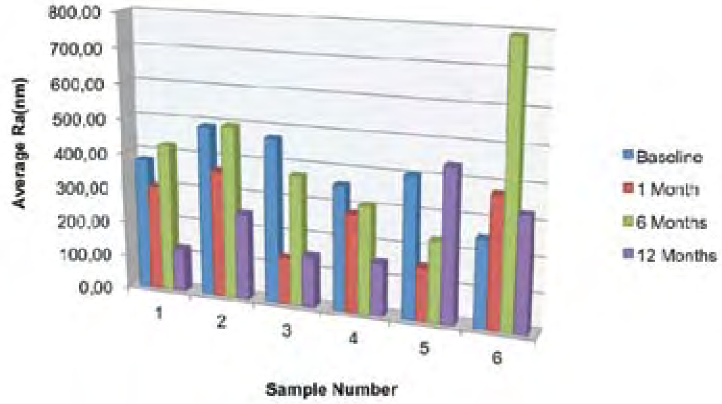

Figure 5: IPS Empress CAD® samples average surface roughness (ηm) over time tested

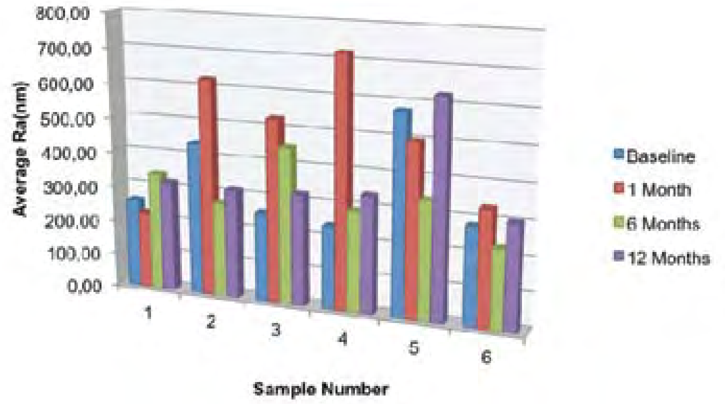

Figure 6: CEREC VITABLOC® Mark II CAD samples average surface roughness (ηm) over time tested

after the last exposure time frame. Sample 6, after the 6 months exposure time frame showed a very marked increase in surface roughness, which is inexplicable. A mean overall surface roughness recorded for IPS Empress CAD® was 334.271ηm (150.311).

The CEREC VITABLOC® Mark II CAD had a reverse pattern when compared to IPS Empress CAD®, with mean roughness readings of 342.333ηm (136.457), 488.333ηm (184.096), 320.917ηm (76.405) and 369.817ηm (126.236) at baseline, 1 month, 6 months and 12 months respectively (Figure 6). With some exceptions (samples 1 and 5) roughness tended to increase initially, and then decrease, followed by a further increase. A mean overall surface roughness of 380.350ηm (143.242) was recorded making it the worst of all the materials.

The ANOVA assessment for mean surface area (Table 2) showed similar outcomes as those of mean surface roughness. The materials differed significantly ($p<0.0001$) with respect to surface area and that the interaction of materials over time was also significant ($p=0.0014$). However, once again, the differences between the materials over time need to be interpreted with caution.

A comparison of the results for both surface area and surface roughness showed that there is a linear correlation between both entities. Therefore, if surface area increased, surface

Table 2: Mean surface area (µm²) and standard deviation over time

Time	Materials			
	IPS e.max CAD®	IPS Empress CAD®	CEREC VITABLOC® Mark II CAD	Total
Baseline	101.500 (0.775)	133.000 (19.537)	122.917 (0.096)	119.139 (17.873)
1 month	106.917 (3.368)	126.417 (13.959)	156.333 (22.833)	129.889 (25.521)
6 months	104.500 (3.674)	138.250 (12.825)	122.250 (8.448)	121.667 (16.569)
12 months	103.000 (1.949)	135.083 (12.407)	151.083 (16.129)	129.722 (23.369)
Total	103.979 (3.245)	133.188 (14.634)	138.146 (21.442)	125.104 (21.264)

roughness also increased. The results obtained show that IPS e.max CAD® is the least affected by acid exposure in respect of surface roughness and surface area.

DISCUSSION

Patient's aesthetic demands and expectations as well as new material choices and manufacturing techniques are major driving forces behind material selection when restoring teeth. As such many of the newly developed ceramics are being more frequently used, especially those manufactured using CAD CAM technology. Very few studies have been done to test the effects of simulated gastric acid on these ceramic restorative materials. Matsou *et al*. 35 exposed three different ceramic materials to a pH solution of 3.8 for 24 hours at 37°C. They also used AFM to analyze the samples, but found no statistical significant differences between the materials after acid exposure. However a major shortcoming of their experiment was the short exposure time, which was not a cumulative 24 hours, but rather a simulated vomiting exposure over 24 hours. The present study used cumulative exposure times and an acidic solution with a lower pH to mimic the oral conditions more closely.

Materials that contain glass particles are prone to acid dissolution of these particles that may result in their having a decrease flexural strength.[33,34] All the materials tested in this study contained glass particles of varying sizes, ranging from 4µm, for CEREC VITABLOC® Mark II CAD, 1-5 µm for IPS Empress CAD® and the smallest of 0.2-1.0µm for IPS e.max CAD®.[32,33,34] It can be hypothesized that the dissolution of these particles will result in surface defects correlating to the sizes of the particles lost. This study confirmed that assumption with both CEREC VITABLOC® Mark II CAD and IPS Empress CAD® having similar surface roughness results and IPS e.max CAD® showing the smallest changes in surface roughness.

A linear relationship was observed between surface area and surface roughness. It can be hypothesised that following the erosive pattern seen for IPS e.max CAD® a possible continuation of the decrease of the surface area will follow over a longer time period, resulting in a 'smoother' material. Therefore, one can expect IPS e.max CAD® to have the least amount of abrasiveness and plaque retentitive potential when compared with the other two tested materials.

On completion of the study, IPS Empress CAD® results also ended on a downward trend. However, the results obtained for this material were significantly higher than the IPS e.max CAD®. (Mean surface area of 135.083µm2 (12.407) and roughness of 239.750ɳm (124.166) for the IPS Empress CAD® compared with 103µm2 (1.949) and 25.133ɳm (7.656) respectively for the IPS e.max CAD®.

The poorest performing material was CEREC VITABLOC® Mark II CAD. On completion of the study the results obtained were 151.083µm² (16.129) and 369.817ɳm (126.236) for the mean surface area and mean surface roughness respectively. It can therefore be hypothesized that CEREC VITABLOC® Mark II CAD will have the most abrasive and plaque accumulating characteristics post-acid exposure of the three materials tested. Based on these results it would also be the least suitable material for restorations in patients with bulimia or GERD, while IPS e.max CAD® would be the best choice.

CONCLUSION

Within the limitations of this study, it was shown that IPS e.max CAD® had the least mean surface roughness after long-term exposure to acids. CEREC VITABLOC® Mark II CAD showed the worst overall results. Clinically, IPS e.max CAD® would be the superior choice of material for patients with bulimia and GERD.

The poor performance of CEREC VITABLOC® Mark II CAD could be attributed to the composition of the material and needs further investigation. Further studies are also needed to assess whether glazing and/or polishing has a retardant effect on the erosive potential of gastric acids on all-ceramic materials as well as *in-vivo* effects of these changes on surface roughness of the ceramic material on the opposing dentition and on plaque and bacteria accumulation.

Declaration - No conflict of interest declared.

References

1. Amaechi BT, Higham SM. Dental erosion: possible approaches to prevention and control. J Dent. 2005 Mar;33(3):243-52.
2. Imfeld T. Dental erosion. Definition, classification and links. Eur J Oral Sci. 1996 Apr;104(2(Pt 2)):151-5.
3. Ranjitkar S, Smales RJ, Kaidonis JA. Oral manifestation of gastroesophageal reflux disease. J Gastroenterol Hepatol. 2012 Jan;27(1):21-7.
4. Yu H, Wegehaupt FJ, Wiegand A, Roos M, Attin T, Buchalla W. Erosion and abrasion of tooth-colored restorative materials and human enamel. J Dent. 2009 Dec;37(12):913-22.
5. Vakil N, van Zanten SV, Kahrilas P, Dent J, Jones R, the Global Consensus Group. The Montreal definition and classification of gastroesophageal reflux disease: a global evidence-based consensus. Am J Gastroenterol. 2006 Aug; 101(8):1900-20.
6. Dent J, El-Seraq HB, Wallander MA, Johansson S. Epidemiology of gastro-oesophageal reflux disease: a systematic review. Gut. 2005 May;54(5):710-7.
7. Zagari RM, Fuccio L, Wallander MA, Johansson S, Fiocca R, Casanova S *et al*. Gastro-oesophageal reflux symptoms, oesophagitis and Barrett's oesophagus in the general population: the Loiano-Monghidoro study. Gut. 2008 Oct;57(10):1354-9.
8. Nelson SP, Chen EH, Syniar GM, Christoffel KK. Prevalence of symptoms of gastroesophageal reflux during childhood: a pedi-

atric practice-based survey. Pediatric Practice Research Group. Arch Pediatr Adolesc Med. 2000 Feb;154(2):150-4.
9. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 5th ed. (DSM-V). Washington, DC: American Psychiatric Association; 2000.
10. Wakeling A. Epidemiology of anorexia nervosa. Psychiatry Res. 1996 April 16;62(1):3-9.
11. Fairburn CG, Beglin SJ. Studies of the epidemiology of bulimia nervosa. Am J Psychiatry. 1990 April;147(4):401-8.
12. Treasure J, Claudino AM, Zucker N. Eating Disorders. Lancet. 2010 Feb 13;375(9714):583-93.
13. Kukiattrakoon B, Hengtrakool C, Kedjarune-Leggat U. Chemical durability and microhardness of dental ceramics immersed in acidic agents. Acta Odontol Scand. 2010 Jan;68(1):1-10.
14. Hetherington MM, Altemus M, Nelson ML, Bernat AS, Gold PW. Eating behavior in bulimia nervosa: multiple meal analyses. Am J Clin Nutr. 1994 Dec;60(6):864-73.
15. Pro-Eating Disorder Society 2012, viewed 09 February 2012, <http://proeatingdisordersociety.yuku.com/topic/6348#.TzQ-JOlFps4Y>.
16. Lussi A. Erosive Tooth Wear – A multifactorial condition of growing concern and increasing knowledge. In: Lussi A, editor. Dental Erosion from Diagnosis to Therapy. Basel (Switzerland): Karger AG; 2006.p. 1-8.
17. Scheutzel P. Etiology of dental erosion - intrinsic factors. Eur J Oral Sci. 1996 Apr;104(2):178-90.
18. Jarvinen VK, Rytömaa II, Heinonen OP. Risk factors in dental erosion. J Dent Res. 1991 Jun;70(6):942-7.
19. Mehler PS. Clinical practice. Bulimia nervosa. N Engl J Med. 2003 Aug;349(9):875-81.
20. Robb ND, Smith BG. Anorexia and bulimia nervosa (the eating disorders): conditions of interest to the dental practitioner. J Dent. 1996 Jan-Mar;24(1-2):7-16.
21. Mihailopol CF, Lăcătuşu S, Codreanu CM, Pancu G, Topoliceanu C, Ghiorghe CA. Correlations between dental erosion severity and salivary factor in patients with gastroesophageal reflux disease. Romanian J Oral Rehabil. 2001 Dec;3(4):63-6.
22. Moazzez R, Barlett D, Anggiansah A. Dental erosion, gastro-oesophageal reflux disease and saliva: how are they related? J Dent. 2004 Aug;32(6):489-94.
23. Campisi G, Lo Russo L, Di Liberto C, Di Nicola F, Butera D, Vigneri S *et al.* Saliva variations in gastro-oesophageal reflux disease. J Dent. 2008 Apr;36(4):268-71.
24. Blair FM, Wassell RW, Steele JG. Crowns and other extra-coronal restorations: preparations for full veneer crowns. Br Dent J. 2002 May;192 (10):567-71.
25. Shillingburg HT, Hobo S, Whitsett LD, Jacobi R, Brackett SE. Fundamentals of Fixed Prosthodontics. 3rd ed. New Delhi: M/s Passi Publications; 2002.
26. Rosenstiel SF, Land MF, Fujimoto J. Contemporary Fixed Prosthodontics. 4th ed. St. Louis: Mosby Elsevier; 2006.
27. Manicone PF, Rossi Lommetti P, Raffaelli L. An overview of zirconia ceramics: basic properties and clinical applications. J Dent. 2007 Nov;35(11):819 – 26.
28. Zahran M, El-Mowafy O, Tam L, Watson PA, Finer Y. Fracture strength and fatigue resistance of all-ceramic molar crowns manufactured with CAD/CAM technology. J Prosthodont. 2008 Jul;17(5):370–7.
29. McLaren EA, Cao PT. Ceramics in dentistry - part 1: classes of materials. Inside Dentistry. 2009 October;94-104.
30. Denry I, Holloway JA. Ceramics for dental applications: a review. Materials. 2010;3:351-68.
31. Anusavice KJ. Degradability of dental ceramics. Adv Dentl Res. 1992 Sept;6:82-9.
32. Vident. VITA VITABLOC® Mark II for CEREC® Material Science and Clinical Studies [Pamphlet]. Vident; 2009.
33. Ivoclar Vivadent. IPS Empress CAD® Scientific documentation [Pamphlet]. Ivoclar Vivadent AG; October 2011.
34. Ivoclar Vivadent. IPS e.max® CAD Scientific documentation [Phamphlet]. Ivoclar Vivadent AG; March 2011.
35. Matsou E, Vouroutzis N, Kontonasaki E, Paraskevopoulos KM, Koidis P. Investigation of the influence of gastric acid on the surface roughness of ceramic materials of metal-ceramic restorations. An *in vitro* study. Int J Prosthodont. 2011 Jan-Feb;24(1):26-29.
36. Van de Vyver P, de Wet FA. Effect of finishing systems on the surface roughness of an experimental low-shrinkage composite resin. SADJ. 2009;64(6):260-265.
37. Heurich E, Beyer M, Jandt KD, Reichert J, Schnabelrauch M *et al.* Quantification of dental erosion - a comparison of stylus profilometery and confocal laser scanning microscopy (CLSM). Dent Mater. 2010 Apr;26(4):326-36.