A multivariable hospital-based retrospective analysis of factors affecting non-surgical periodontal treatment response in the East London population

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ABSTRACT

Periodontitis is a chronic inflammatory disease, initiated by dental plaque bacteria and characterised by destruction of the tooth supporting tissues including alveolar bone. Historically, disease progression in periodontitis was thought to be correlated with the amount of plaque biofilm (Frunker and Gardner, 1956, Grant D et al., 1968, Russell, 1964, Waerhaug, 1956). However, our current understanding of the disease suggests that a loss in equilibrium between the commensal and pathogenic microorganisms (dysbiosis) and the reduction of the protective host response drives the disease.

Since periodontitis is initiated by bacterial subgingival biofilms, mechanical removal of subgingival products and plaque retentive factors (e.g., calculus) is required. Although, complete removal of deposits is frequently not achieved (Waerhaug, 1978, Eaton et al., 1985, Caffesse et al., 1986, Sherman et al., 1990, Wylam et al., 1993, Breininger et al., 1987, Rateitschak-Pluss et al., 1992), mechanical non-surgical periodontal treatment (NSPT) continues to be an integral part of periodontal therapy.

NSPT comprising of root surface debridement (RSD) alongside adequate oral hygiene to reduce bacterial load, not only improves gingival health and arrests disease progression (Heitz-Mayfield, 2005, Suvan, 2005, Cobb, 1996, Van der Weijden and Timmerman, 2002), but also reduces the risk of tooth loss (Badersten et al., 1985b, Badersten et al., 1985a, Badersten et al., 1984). The efficacy and effectiveness of NSPT is proven by many studies demonstrating reductions in periodontal probing depths (PPD), clinical attachment level (CAL) gain and bleeding on probing (BOP) (Van der Weijden and Timmerman, 2002, Trombelli et al., 2015, Suvan, 2005, Badersten et al., 1984, Suvan et al., 2020).

Clinical outcomes of NSPT can be influenced by several factors including age (Trombelli et al., 2010), gender (Mascarenhas et al., 2003), cigarette smoking (Papantonopoulos, 1999, Pahkla et al., 2006, Wan et al., 2009), diabetes (Christgau et al., 1998, Tervonen and Karjalainen, 1997), operator experience (Fleischer et al., 1989) and patient compliance (Leininger et al., 2010).

The Royal London Dental Hospital (RLH) in Tower Hamlets is one of the most socio-economically deprived and ethnically diverse boroughs in the UK. Recent data from a large sample of our local East London population has suggested that periodontitis is more severe in minority ethnic groups (Delgado-Angulo et al., 2016). Although, in general these groups are frequently under-represented in clinical outcome studies compared to Caucasian populations (Jiao et al., 2017). Therefore, there is a need to better understand the factors influencing non-surgical success in our local East London population.

The aim of this hospital-based retrospective study was therefore to evaluate factors influencing the effectiveness of NSPT in patients referred for periodontal treatment at Royal London Dental Hospital using data extracted from their clinical records. We also wanted to specifically assess if individuals from a South East Asian (SEA) ethnic background had poorer outcomes after NSPT. Univariate, bivariate, and multivariate analysis to measure the relative contributions of the different factors in the local population of East London. Mean PPD change was the primary outcome variable. In this study, from a sample size of 108 patients, no differences were found between ethnicities or between the SEA group and ‘others’ group in relation to NSPT outcome measured by mean PPD change. There were however differences between the SEA and other groups in terms of age of referral, levels of self-reported attendance, and levels of stress. Plaque scores at reassessment did not impact NSPT outcome but compliant patient had poorer outcomes. However univariate analysis showed that patients achieving at least a 10% improvement in plaque between baseline and reassessment showed better non-surgical treatment outcomes. There were also significant

differences in NSPT response when comparing patients reassessed at <120 days (n=80) compared to those reassessed at >120 days (n=28). Baseline Disease severity was the only significant predictor of good treatment response in the binary logistic regression model which explained 26.6% of the variability in the dependent outcome variable.

The multilevel analysis also showed that patients presenting with higher levels of disease (Highest 50% percentile) at baseline (Mean PPD) are 3.9 times more likely to have a good non-surgical treatment response (more pocket depth reduction) compared to those with lower levels of disease (Lowest 50% percentile). However all other variables were non-significant in this model, whilst many factors significant in the univariate analysis became non-significant when combined into the same binary logistic regression model.
There are several limitations to this study especially in terms of the number of patients we were able to recruit which may explain some of the outcomes which contradict previous studies.

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LIST OF ABBREVIATIONS

A.a	Aggregatibacter actinomycetemcomitans
ANOVA	Analysis of Variance
B	Baseline
BI	Bleeding Index
BOP	Bleeding on Probing
BSP	British Society of Periodontology and Implant Dentistry
CAL	Clinical Attachment Loss
CHX	Chlorhexidine Gluconate
DM	Diabetes Mellitus
DPTT	Diabetes and Periodontal Therapy Trial
EFP	European Federation of Periodontology
FI	Furcation involvement
fMLP	N-formyl-methyl-leucylphenyl- alanine
FMBS	Full Mouth Bleeding Score
FMD	Full Mouth Disinfection
FMPS	Full Mouth Plaque Score
FMS	Full Mouth Scaling
FPM	Frequency of Periodontal Maintenance
GCF	Gingival Crevicular Fluid
HbA1C	Glycosylated Haemoglobin
IL- 6-174 G/C	G/C promoter polymorphism at nt (-174) of the interleukin-6
ITI	International team of Implantology
mm	millimetres
MWF	Modified Widman Flap
NHANES	National Health and Nutrition Examination Survey
no.	Number of:
NS	Non-Surgical
NS1	Reassessment 1
NSPT	Non-Surgical Periodontal Treatment/Therapy
OH	Oral Hygiene
OHI	Oral Hygiene Instructions
OFD	Open Flap Debridement
PAL	Probing Attachment Level
P.g	Porphyromonas gingivalis
PMNs	Polymorphonuclear Leukocyte
PPD	Probing Pocket Depth
PRF	Plaque Retention Factor
PROM	Patient Related Outcome Measure
PS	Plaque score
RLDH	Royal London Dental Hospital
RPL	Root Planing
RSD	Root surface Debridement
SD	Standard Deviation
SE	Standard Error
SEA	South East Asian
SEP	Socioeconomic position
Sig	Significance level
SPSS	Statistical Package for the Social Sciences
SRP	Scaling & Root Planing

T.d	Treponema denticola
USA	United States of America
UK	United Kingdom

CHAPTER 1: INTRODUCTION

Periodontitis is a chronic inflammatory disease, initiated by dental plaque bacteria and characterised by destruction of the tooth supporting tissues including alveolar bone, which may result in tooth loss. Historically, disease progression in periodontitis was thought to be correlated with the amount of plaque biofilm (Frunker and Gardner, 1956, Grant D et al., 1968, Russell, 1964, Waerhaug, 1956). However, our current understanding of the disease suggests that a loss in equilibrium between the commensal and pathogenic microorganisms (dysbiosis) and the reduction of the protective host response drives the disease.

Traditional Scaling and Root Planing [SRP] was therefore advocated to be undertaken in three separate stages: debridement, scaling, and root planing (Kieser, 1994). Debridement was defined as instrumentation to disrupt or remove microbial biofilms, scaling defined as instrumentation for removal of mineralised deposits, i.e. calculus, and root planing defined as instrumentation to remove “contaminated” cementum and dentine in order to restore the biologic compatibility of periodontally diseased root surfaces (Kieser, 1994).

Since periodontitis is caused by bacteria residing in subgingival biofilms, the need to lower the microbial load by disruption or removal of the subgingival biofilm is clear. Although it does not itself induce inflammation, calculus has been demonstrated in cross-sectional and longitudinal epidemiological studies to be associated with periodontitis (Adriaens and Adriaens, 2004, Albandar et al., 1996, Albandar and Kingman, 1999, Albandar et al., 1998, Anerud et al., 1991, Christersson et al., 1992, Clerehugh and Lennon, 1986, Clerehugh et al., 1997, Clerehugh et al., 1995, Griffiths et al., 2001, Grossi et al., 1994, Julihn et al., 2008, Lovdal et al., 1958, Martınez-Canut et al., 1999, Ong, 1996, Roberts-Harry and Clerehugh, 2000, Sjodin and Matsson, 1994, Waerhaug, 1952). Furthermore, it has been demonstrated that periodontal healing following flap surgery at sites with residual calculus had more inflammation than sites without calculus (Fujikawa et al., 1988). Rather than being a cause of inflammation, it has instead been considered that calculus may form as a result of periodontal inflammation formed by the calcification of subgingival plaque in the presence of an increased high flow of gingival crevicular fluid [GCF] (Jepsen et al., 2011). This increase in GCF is strongly induced as part of the inflammatory process whilst also providing the minerals required for plaque mineralisation (Mandel and Gaffar, 1986).

It is however difficult to distinguish whether the effects on the periodontium are due to calculus or plaque that covers calculus (White, 1997), especially as early experimental animal studies showed that sterile calculus does not cause pronounced inflammation or abscess formation in connective tissues (Allen and Kerr, 1965). It was also shown that normal epithelial attachment can be formed on calculus if its surface has been treated with chlorhexidine (Listgarten and Ellegaard, 1973). Further studies have demonstrated that removal of subgingival plaque covering the subgingival calculus resulted in periodontal healing indicating that calculus may not be involved in the primary aetiology (Lang et al., 2008a, Mombelli et al., 1995, Nyman et al., 1986, Nyman et al., 1988). Although calculus does not necessarily itself induce inflammation, it can provide an ideal surface for microbial colonisation (Waerhaug, 1952) whilst subgingival calculus provides an ideal environment for bacterial adhesion (Lang et al., 2008a, Schroeder, 1969, Zander et al., 1960). In patients with moderate to severe periodontitis, living aerobic and anaerobic bacteria have been detected in supragingival calculus samples (Tan et al., 2004) whilst periodontal pathogens Aggregatibacter actinomycetemcomitans [A.a], Porphyromonas gingivalis [P.g] and Treponema denticola [T.d] have been detected within the lacunae of subgingival calculus (Calabrese et al., 2007).

Calculus may therefore have a secondary role of in the development and progression of periodontitis, by providing an ideal porous vehicle for bacterial plaque retention and growth

as well as a reservoir for toxic bacterial products and antigens. Once formed, the presence of calculus may prevent adequate oral hygiene procedures thereby allowing plaque accumulation. As a prominent plaque retentive factor, it must be removed for adequate periodontal therapy and prophylaxis (Lang et al., 2008a, Clerehugh et al., 1996). The rationale for calculus removal is to eliminate as much as possible any surface irregularities that may harbour pathogenic bacteria.

The original rationale for performing 'root planing' was based on the concept that bacterial endotoxins penetrate into the cementum (Aleo et al., 1974, Hatfield & Baumhammers, 1971). It was therefore deemed necessary to remove the underlying cementum as well as the biofilms and calculus. However, subsequent evidence demonstrated that endotoxins were only loosely attached to the surface and did not penetrate cementum (Hughes and Smales, 1986, Moore et al., 1986, Hughes et al., 1988, Cadosch et al., 2003). Hence, cementum removal was no longer deemed necessary. The aim of non-surgical periodontal treatment [NSPT] was therefore considered to require only the removal of plaque biofilms and calculus. However, several in vitro (Rateitschak-Pluss et al., 1992, Breininger et al., 1987)and in vivo studies (Waerhaug, 1978, Eaton et al., 1985, Caffesse et al., 1986, Sherman et al., 1990, Wylam et al., 1993) have shown that complete removal of hard and soft deposits is not feasible, even with the most meticulous scaling and root planing procedures [SRP].

Despite its limitations and with the adoption of other treatment techniques, e.g. laser therapy, NSPT continues to be an integral part of periodontal therapy as demonstrated by the marked reduction in clinical signs and symptoms of the disease following treatment (Heitz-Mayfield, 2005, Suvan, 2005, Cobb, 1996, Van der Weijden and Timmerman, 2002). Non-surgical periodontal treatment comprising of root surface debridement [RSD] alongside adequate oral hygiene to reduce bacterial load, not only improves gingival health and arrests disease progression (Heitz-Mayfield, 2005, Suvan, 2005, Cobb, 1996, Van der Weijden and Timmerman, 2002), but also reduces the risk of tooth loss (Badersten et al., 1985b, Badersten et al., 1985a, Badersten et al., 1984). The efficacy (as established in strictly defined research setting to minimise confounding factors) of non-surgical subgingival instrumentation as part of periodontal treatment is well-documented, demonstrated by reductions in probing pocket depths [PPD], Clinical Attachment Level [CAL] gains, and bleeding on probing [BOP] reductions (Van der Weijden and Timmerman, 2002, Trombelli et al., 2015, Suvan, 2005, Badersten et al., 1984, Suvan et al., 2020). However, there is a lack of data addressing effectiveness (established in a real world setting such as clinical practice with potential additional confounding factors) of non-surgical interventions.

A recent systematic review (Suvan et al., 2020) found that the weighted PPD reduction was 1.0 mm at 3/4 months and 1.4 mm at 6/8 months. The proportion of closed pockets was estimated to be 57% and 74% at the two time points. Initially shallow sites revealed a weighted mean PPD reduction of 1.5 mm at 3/4 months and 1.6 mm at 6/8 months. Whilst for initially deep sites, a weighted PPD reduction of 2.6 mm at 3/4 months and 2.6 mm at 6/8 months was observed. The weighted mean BOP reduction, based on relative reduction in patient-based scores, at 3/4 months was 56.7% whilst at 6/8 months was 62.7%. These results confirm that subgingival instrumentation is an efficacious treatment in reducing inflammation, PPD and number of diseased sites in patients affected by periodontitis. This effect was consistent, irrespective of the choice of instrument (sonic/ultrasonic vs. hand) or mode of delivery (full mouth vs. quadrant).

From this review, it can be inferred that well-performed NSPT may limit the need of other additional/alternative treatment approaches, which may entail higher costs and patient morbidity. However there is a lack randomised randomized clinical trials specifically addressing the benefit of subgingival instrumentation as the necessary study design would likely have ethical implications (Suvan et al., 2020). The only randomised controlled trial that could be considered, adopted a three-month delay in delivering the subgingival treatment in

the control group and demonstrated a significant benefit for NSPT in terms of percentage of pocket closure at three months (Kapellas et al., 2013). Other studies addressing efficacy of subgingival instrumentation were often not randomised and/or demonstrated a high risk of bias.

The European federation of Periodontology S3 guidelines for treatment of Stage I to III cases (Sanz et al., 2020), which was implemented by the British Society of Periodontology [BSP] (West et al., 2021) outlines the sequence of non-surgical periodontal treatment (Appendix 9/10). Step one of these guidelines for NSPT include completing Oral hygiene instruction [OHI], risk factor control and undertaking Professional Mechanical Plaque removal [PMPR] to remove supragingival and subgingival deposits. The emphasis in the latest guidance is on developing health behaviour strategies to help facilitate patient motivation in order to achieve a high-level of self-performed supragingival plaque control as well as management of lifestyle habits such as smoking to ensure patients are engaged and aware of their vital role in non-surgical therapy (Ramseier and Suvan, 2015). Whether oral hygiene and risk factor control is adequate, should be evaluated to assess patient engagement before proceeding with the next step. If the patient is engaging, they can proceed to the next stage, which in addition to reinforcement of OHI and risk factor control, involves subgingival instrumentation with hand, powered or a combination of both. If adjunctive antibiotics are determined to be necessary, they should be prescribed at this stage by a level two or three practitioner. This guidance therefore outlines how the main goal of treatment of periodontitis is the establishment of adequate infection control, that is reduction in the bacterial load below individual threshold levels of inflammation/ disease (Suvan et al., 2020). Supplemental to patient self-care, subgingival instrumentation serves the purpose of altering the subgingival ecological environment through disruption of the microbial biofilm and removal of hard deposits, thereby suppressing soft tissue inflammation (Heitz-Mayfield and Lang, 2013, Jepsen et al., 2011)

Clinical outcomes of NSPT can be influenced by several factors including age (Trombelli et al., 2010), gender (Mascarenhas et al., 2003), cigarette smoking (Papantonopoulos, 1999b, Pahkla et al., 2006, Wan et al., 2009, Papantonopoulos, 1999a), diabetes (Christgau et al., 1998, Tervonen and Karjalainen, 1997), operator experience (Fleischer et al., 1989) and patient compliance (Leininger et al., 2010). However, as we are just starting to understand the relative importance of each risk factor in periodontitis (Bryk and Raudenbush, 1992, Goldstein, 1995), a multivariable analysis approach will be best suited to measure the relative contributions of the different factors in the local population of East London.

The Royal London Dental Hospital (RLDH) in Tower Hamlets is one of the most socio-economically deprived and ethnically diverse boroughs in the UK, with 69% belonging to minority ethnic groups, 32% of which are Bangladeshi (Tower-Hamlets, 2018). Recent data from a large sample of our local East London population has suggested that periodontitis is more severe in minority ethnic groups (Delgado-Angulo et al., 2016). Although, in general these groups are frequently under-represented in clinical outcome studies compared to Caucasian populations (Jiao et al., 2017). The only previous treatment outcome study involving our local population showed smoking as a significant negative predicting factor in non-surgical clinical outcome of Generalised Aggressive Periodontitis but interestingly ethnicity was not reported (Hughes et al., 2006b). Therefore, there is a need to better understand the factors influencing non-surgical success in our local East London population.

1.1 Factors influencing outcomes of NSPT

Many studies have undertaken univariate analyses of factors that may influence the outcome of NSPT. In the current literature, several studies have also carried out such a multilevel analysis to assess the factors that influence the outcome of non-surgical periodontal treatment (Tomasi et al., 2007, Wan et al., 2009, Jiao et al., 2017, D'Aiuto et al., 2005). Tomasi *et al* (2007) treated forty-one patients with either full mouth piezoelectric debridement or four rounds of quadrant SRP with hand instruments, with the main outcome variable being the probability of ‘‘pocket closure’’ (i.e. probing pocket depth [PPD]<4 mm). The logistic model revealed that smoking habits, plaque at site level and tooth type were significant factors in determining the short-term clinical outcome of NSPT (Tomasi et al., 2007).

Wan *et al.* (2009), recruited forty male patients, twenty of whom were smokers and assessed the outcome of NSPT with the main outcome variable being PPD reduction. Multilevel multiple regression showed that three predictors: non-smokers (subject level), anterior teeth (tooth-level) and sites without plaque at baseline (site level), were significantly associated with a greater reduction in PPD in initially diseased sites over the twelve-month study period ($p<0.05$). Smokers also showed less favourable PPD reduction at deep sites after non-surgical periodontal therapy (Wan et al., 2009).

Jiao *et al.* (2017) analysed factors which may influence the outcome of NSPT in a large Chinese population (10,789 patients) based on PPD and bleeding index [BI] reductions. Multilevel analysis demonstrated that PPD and BI reductions were mainly influenced by baseline PPD, baseline attachment loss [AL], baseline mobility, tooth type and frequency of periodontal maintenance [FPM] (Jiao et al., 2017).

D’Aiuto *et al.* (2005) also evaluated the relative contribution of patient, tooth, and site associated variables that may influence the outcome of NSPT with a hierarchical multilevel analysis. Eighty percent (80%) of variability in PPD reductions was attributed to site level parameters, while 12% was at the tooth level and 8% at the patient level. Cigarette smoking and carriage of the rare allele of the IL- 6-174 G/C polymorphism were associated with less PPD reduction. Incisors and canines responded better to treatment than premolars and molars, meanwhile a dose-dependent effect of mobility was observed where higher baseline mobility resulted in significantly greater decreases in PPD. At the site level, greater reductions were observed at interdental sites (compared to facial/oral), and at deeper sites (>6 mm) (D'Aiuto et al., 2005).

Many other studies undertaking a multilevel analysis also organise the factors influencing the outcome of NSPT according to site, tooth, or patient level variables (Figure 1). Operator factors (operator experience, use of hand or machine instrumentation, quadrant or full mouth approaches) have not been part of these multilevel analyses but have been explored in many studies and systematic reviews.

1.2 Site level Factors

1.2.1 Baseline PPD

As discussed, a recent systematic review (Suvan et al., 2020), demonstrated that subgingival instrumentation is an efficacious treatment to reduce inflammation, PPD and the number of diseased sites in patients affected by periodontitis. Shallow sites (4-6 mm) were shown to achieve less mean reduction of PPD (1.5 mm) at 6/8 months compared to deeper sites (≥7 mm) (2.6 mm). A previous systematic review (Van der Weijden and Timmerman, 2002) corroborated these findings that sites with initially deeper pockets had greater PPD reduction than initially shallow pockets.

Previous studies have also examined the outcome of NSPT based on their baseline PPD. When treating pockets 4-7 mm deep in periodontitis patients, after thirteen months only thirteen sites (12.2%) out of initial 106 sites more than 6 mm remained as deep pockets (Badersten et al., 1981). However, in patients with PPDs up to 12 mm deep, 43 out of 305 sites (14.1%) with baseline PPD >7 mm remained twelve months after NPST (Badersten et al., 1984). Initially deeper sites also showed more gingival recession, more gain in probing attachment and deeper residual pocketing compared to sites with initially shallower pockets (Badersten et al., 1984). With regards to attachment gain/loss, sites with initially shallow PPD (<3.5 mm) demonstrated loss whereas sites with initial PPD >8 mm showed 0.9-2.3 mm average gain in attachment gain. Moderately deep sites (4-7 mm deep) had a mean total PPD reduction of 1.3-1.7 mm at four/five months, whereas for deep sites (>7 mm), the average reduction in PPD was 3.6-3.9 mm at twelve months. PPDs of sites initially 7-12.5 mm markedly reduced following a non-surgical approach, with a mean attachment gain of 4-6.5 mm being achieved. Deep pockets showed more gain in probing attachment compared to moderately deep sites (Badersten et al., 1981, Badersten et al., 1984).

Another study compared the long-term effectiveness of SRP alone to SRP followed by periodontal surgery. This split mouth study had seventeen subjects with moderate to advanced periodontitis. One half of each subject's dentition was randomly selected to be subjected to a modified Widman flap [MWF]. After treatment was completed, recall prophylaxis and oral hygiene reinforcement were administered for four years. Shallow sites (1–3 mm) subjected to either procedure tended to increase slightly in depth and exhibit a slight loss of clinical attachment when compared to baseline measurements. Moderately deep pockets (4–6 mm) treated by either procedure were reduced and demonstrated a sustained gain or maintenance of attachment level. As with the previous studies mentioned, deep pockets (initially ≥7 mm) exhibited the greatest reduction in PPD as well as the largest gain in attachment (Pihlstrom et al., 1981).

A meta-analysis (Heitz-Mayfield, 2005) compared the effectiveness of surgical therapy to non-surgical therapy after twelve months. Shallow pockets (1–3 mm) showed greater attachment loss following surgery compared to SRP, however this difference in attachment loss between surgery and NSPT was unclear. There was no significant difference in PPD reduction between procedures. In 4-6 mm pockets, SRP resulted in 0.4 mm more clinical attachment gain and 0.4 mm less PPD reduction than surgery. In deep pockets (> 6 mm), surgery resulted in 0.6 mm more PPD reduction and 0.2 mm more clinical attachment gain than non-surgical therapy.

Another split mouth clinical trial (Ramfjord et al., 1987) assessed the results following four different modalities of periodontal therapy (pocket elimination or reduction surgery, modified Widman flap surgery, subgingival curettage, and SRP) over five years. For ninety patients, the treatment methods were applied on a random basis to each quadrant. Patients were given PMPR and OHI every three months. PPD and attachment levels were scored once a year. For 1-3 mm probing depth, SRP and subgingival curettage led to significantly less attachment loss than pocket elimination and modified Widman flap surgery. For 4-6 mm pockets, SRP and curettage had better attachment results than pocket elimination surgery. For the 7-12 mm pockets, there was no statistically significant difference between procedures. This study therefore again highlighted the effectiveness of NSPT and how even for deep sites >7 mm no other method was statistically superior to SPR. This study follows a longer follow up of the same cohort (Hill et al., 1981) of patients assessed previously at two years when again there was no statistically differences in outcome between the four methods.

Another study comparing SRP with other surgical modalities to achieve PPD reduction (Isidor et al., 1984) found that root planing resulted in considerable reduction in PPD, however shallow pockets achieved more PPD reduction following modified Widman flap and reverse bevel flap surgery. Clinical gain of attachment was obtained following all modalities, but SRP resulted in slightly more gain of attachment than the surgical procedures.

The concepts of critical probing depths for different treatment modalities for periodontitis was assessed in an investigation carried out on fifteen individuals referred for treatment of moderately advanced periodontal disease (Lindhe et al., 1982a). After baseline examinations and OHI, patients were given periodontal treatment utilising a split mouth design where SRP in combination with MWF on half the dentition and only SPR on the contralateral side was completed. During the healing phase (first six months post treatment) the patients were recalled for PMPR every two weeks. During the maintenance phase (six to twenty-four months post treatment) the interval between recall appointments was extended to three months. Re-examinations were carried out six, twelve and twenty-four months after the completion of active treatment. Results revealed that treatment resulted in loss of clinical attachment in sites with initially shallow pockets, while sites with initially deep pockets gained clinical attachment. Regression analysis allowed “critical probing depths” to be calculated for the two methods of treatments used. It was found that the critical probing depth value for SRP (2.9 mm) was significantly smaller than for SRP with MWF (4.2 mm) (Figure 2). In addition, the surgical modality of therapy resulted in more attachment loss than the non-surgical approach when used in sites with initially shallow pockets. On the other hand, in sites with initial probing depths above the critical probing depth value more gain of clinical attachment occurred following MWF than following SRP.

The critical probing depth for NSPT/SRP [RPL] is 2.9 mm; below this PPD the site would lose clinical attachment as a result of therapy, whilst above this value clinical attachment gain will result. For the access flap therapy, this critical probing depth was 4.2 mm, therefore open flap debridement [OFD] is only beneficial when initial PPD is above this value, while below this value, attachment loss may result. When analysing the data for SRP and access flap surgery, another critical probing depth is 5.4 mm. Sites with initial PPD $\geq$5.4 mm will achieve more attachment gain with flap surgery whereas for sites 2.9-5.4 mm NSPT therapy is preferred.

A similar study from the same cohort of patient assessing healing responses (Lindhe et al., 1982b) revealed that PPD reduction was more pronounced in initially deep compared to initially shallow sites exposed to SRP alone. Initially deep sites also exhibited more clinical attachment gain than in shallow pockets. Significant loss of attachment did not occur following SRP but did occur at sites where MWF has been undertaken where initial PPD was <4 mm.

Critical probing depths of surgical and non-surgical periodontal therapy were again assessed in a meta-analysis more recently (Heitz-Mayfield and Lang, 2013). As with Lindhe et al (1982a) the critical probing depth represented a baseline probing-depth value above which the outcome of a therapy will result in attachment gain and below which the outcome of therapy will result in clinical attachment loss. Meta-analysis evaluation of six randomised controlled trials indicated that twelve months following treatment, surgical therapy resulted in 0.6 mm more PPD reduction and 0.2 mm more CAL gain than NSPT in deep pockets (>6 mm). In 4–6 mm pockets NSPT resulted in 0.4 mm more attachment gain and 0.4 mm less PPD reduction than surgical therapy. In shallow pockets (1–3 mm) NSPT resulted in 0.5 mm less attachment loss than surgical therapy. It was therefore concluded that both NSPT alone or combined with flap procedure are effective methods for the treatment of chronic periodontitis in terms of attachment level gain and reduction in gingival inflammation. In the treatment of deep pockets OFD results in greater PPD reduction and clinical attachment gain.

In a study investigating prognostic factors that affect NSPT response in the same East London population that is to be studied (Hughes et al., 2006b), the primary outcome measure for patient-specific analyses was poor response to treatment of deep pockets (>5 mm at baseline). As with previous studies, deep sites also had greater improvements with PPD reduction of 2.11 mm and attachment gain of 1.7 mm compared to shallow sites (initial PPD <3 mm) where mean PPD increased by 0.15 mm and attachment loss of 0.13 mm occurred.

A multivariable analysis evaluated factors that influence the outcome of NSPT (Tomasi et al., 2007) with ‘pocket closure’ as the primary outcome. When initial PPD was introduced into the model, the probability of ‘pocket closure’ for sites with initial PPD > 5 mm three months after treatment was 87%. With each millimetre increase of initial PPD, the log odds decreased by 1.14 ($p<0.0001$). Hence, the probability of closing a 6 mm pocket was 67%, and for a 7 mm pocket 40%. This study therefore highlighted that the higher the baseline PPD, then the chance of pocket closure reduces, regardless of other factors.

The impact of baseline probing depth on the PPD reduction was assessed as one of the site-associated variables that may affect NSPT (D'Aiuto et al., 2005). As shown in previous studies, it is generally agreed that deeper pockets have a greater potential for reduction. However as another author warned (Tu et al., 2005), there are potential issues with the effect of mathematical coupling in multivariate models incorporating baseline probing depths as an independent variable. To minimise the effects of this important variable and reduce the impact of mathematical coupling, baseline PPD measurements were therefore excluded from the model. Despite this, the analysis in this study concurs with the classical studies which indicate that greater improvement in mean PPD reduction is seen in deeper pockets.

Another study investigating factors that affect NSPT response (Jiao et al., 2017) determined that the effectiveness of NSPT is mainly influenced by baseline PPD, baseline attachment level, baseline mobility and tooth type. The most influential factor was determined to be baseline PPD at the site level. If baseline PPD was increased by 1 mm, according to multilevel linear regression models, PPD reductions of sites would have been 0.56 mm greater for all sites and 0.59 mm greater for sites with baseline PPD$\geq$5 mm. Despite potential issues with regression analysis (Tu et al., 2005), baseline PPD was included into the final multilevel model, firstly as baseline PPD was the most influential prognostic factor affecting the outcome of NSPT and coefficient of the multilevel regression analysis was consistent with clinical practice, and secondly as variations in PPD were better explained after baseline PPD was included. Jiao et al (2017) concluded that PPD at baseline did have a significant influence on the treatment outcomes of NSPT although the reliability of the effect was comprised by the ‘nature of regression’.

Meanwhile, a study analysing outcomes measures that can used for diagnostic predictability (Badersten et al., 1990) found that sites with residual PPD ≥7 mm increased from 9% at six months to 52% at sixty months, meaning at earlier observations this PPD seemed to be a poor indicator of the probing attachment level (PAL) at six months but at later intervals, half of sites had undergone attachment loss. Meanwhile an increase of PPD >1 mm showed a diagnostic predictability for progression of 62% after thirty six months and 78% at sixty months.

1.2.2 Furcation involvement at site

Presence of furcation involvement has been included in some studies as either a site level or tooth level prognostic factor. Classification of furcation involvement [FI] categorised furcations into three levels based on the level of horizontal attachment loss present at the furcation clinically (Hamp et al., 1975). A degree I furcation has “horizontal loss of periodontal tissue support less than 3 mm”, a degree II furcation has “horizontal loss of support exceeding 3 mm but not encompassing the total width of the furcation area”, whilst a degree III furcation describes a “horizontal ‘through and through’ destruction of the periodontal tissue in the furcation” (Hamp et al., 1975). This defining study compared different treatment modalities based on the extent of furcation involvement as described above, including both non-surgical and surgical approaches. Of all the teeth involved, 18% of all furcation involved molars were degree I and underwent SRP. Changes in PPD as an outcome variable at this furcation involves sites/ teeth was not reported however the degree I FI molar teeth treated with SRP reported a survival rate of 100% at the 5-year examination. As with all periodontal lesions, treatment of furcation involved teeth requires reduction of the bacterial load of subgingival biofilm, (Heitz-Mayfield et al., 2002), achieved firstly with OHI and NSPT aimed at removing calculus and disrupting the plaque biofilm from the affected root surfaces. However, it is clear that teeth with furcation involvement have a poorer long-term prognosis compared with single-rooted teeth and tooth with no furcation involvement (Hirschfeld and Wasserman, 1978, McFall, 1982). Furthermore, teeth with furcation involvement (FI) do not respond as favourably to NSPT compared to teeth without FI (Nordland et al., 1987, Nibali et al., 2016, Loos et al., 1988), due to the difficulty in cleaning inside the furcation, for both the clinician and patient (Lang et al., 1973, Fleischer et al., 1989).

In a study investigating the effectiveness of instrumentation on multi-rooted teeth (Fleischer et al., 1989), surgical access and a more experienced operator significantly enhanced calculus removal in molars with furcation invasion, whilst demonstrating that total calculus removal in furcations utilising conventional instrumentation may be limited.

A recent systematic review appraised the existing literature on periodontal furcation-involved teeth with respect to tooth loss based on initial diagnosis (no furcation/furcation grade I, II or III) and treatment carried out and to identify areas needing further research (Nibali et al., 2016). This review concluded that the presence of FI approximately doubles the risk of tooth loss for molars maintained in supportive periodontal therapy for up to 10–15 years. Despite this most molars even those with degree III furcation involvement can respond well to periodontal therapy, suggesting that every effort should be made to maintain these teeth when possible. A further clinical trial highlighted that FI increases the risk of molar loss in subjects not undergoing regular periodontal care and that increasing degrees of FI increases the risk of molar loss (Nibali et al., 2017). In fact, a study investigating risk factors associated with the longevity of multi rooted teeth (Salvi et al., 2014) demonstrated that molars with degree II or III FI represented a significant risk for the loss of multi-rooted teeth in subjects

treated for periodontitis, whereas degree I FI did not have any additional risk of tooth loss compared to molars with no furcation involvement. Another study investigating tooth loss in molars and prognostic factors for molar survival also found that degree III FI molars had the greatest mortality (Dannewitz et al., 2006).

The difficulty of effectively removing plaque and performing root debridement at molar furcation lesions has been investigated (Nordland et al., 1987). For sites with initial PPD >4 mm, molar furcation sites respond less favourably than non-furcation sites, as they had higher scores for BOP, less PPD reductions and loss of probing attachment after two years. The limited response to therapy in molars with FI is related to the anatomical configuration of the furcation sites. Poor accessibility to the fornix and concavities of the furcations limit the efficacy of root debridement (Bower, 1989, Waerhaug, 1980, Matia et al., 1986, Leon and Vogel, 1987). In addition to difficulties with achieving initial complete debridement, anatomical factors may limit adequate plaque control and may lead to microbial recolonisation (Mousqueegs et al., 1980, Magnusson et al., 1984, Lavanchy et al., 1987).

Whilst NSPT may be effective for degree I FI molars (Hamp et al., 1975), adequate biofilm removal degree II/III FI molars by the patient is more difficult unless the anatomy of the inter-radicular area is altered, such that despite NSPT of more advanced furcation involvements disease progression will likely occur eventually leading to tooth loss, as illustrated in lower survival rates of molars with residual FI after active periodontal therapy (Dannewitz et al., 2006, Carnevale, 2007).

With regards to assessing the effectiveness of NSPT (Park et al., 2009) in the treatment of FI molars, this was assessed as part of a larger systematic review examining the literature for survival rates of different periodontal therapies (Huynh-Ba et al., 2009). Two papers were found specifically relating to the effectiveness of NSPT; the first paper (Hamp et al., 1975) as previously discussed, demonstrated 100% survival at five years when SRP was applied, although these were only for degree I FI molars. In the second study (Dannewitz et al., 2006), out of 54 non-surgically treated furcation-involved molars (32/54 degree I, 18/54 degree II, 4/54 degree III), five were extracted, corresponding to a survival rate of 90.7% after an observation period ranging from 62 to 145 months (mean 107 months). Out of the five extracted teeth, three teeth had a degree III, one tooth had a degree II and one tooth a degree I furcation. Another study evaluating molar furcations reported similar treatment outcomes (Vertical CAL change, horizontal CAL change and PPD change) 12 months after OFD and SRP procedures (Kalkwarf et al., 1988). Meanwhile when examining if patients were “responding” to NSPT in an East London population, and what factors may influence this, no difference between the outcome of sites with furcation involvement was found (Hughes et al., 2006b).

As discussed, difficult access significantly impedes the ability to maintain such furcation involved molars, and FI is a predictor of tooth loss alongside tooth mobility, bone loss, mean pocket depth and age during supportive periodontal therapy (SPT) (Graetz et al., 2015). Maxillary molars have been shown to have a significantly higher risk of tooth loss than those in the mandible, although multivariable analysis (Graetz et al., 2015) could not attribute this risk increase solely to a higher prevalence of FI in upper (25–72%) than lower molars (16–50%) or that upper molars show advanced bone loss more often (Albandar et al., 1999, Dannewitz et al., 2006, Walter et al., 2011). However, the complex maxillary root anatomy and different bone densities in the maxilla may contribute to maxillary molars having a poorer survival rate (Ross and Thompson, 1980, Park et al., 2009).

In this review (Graetz et al., 2015), severe furcation involvement (FI- III) and bone loss were strong predictors of tooth loss, concurring with previous which also found a potential dose-

response between the degree of FI and tooth loss (Dannewitz et al., 2006, Salvi et al., 2014). As well as trying to treat furcations with NSPT, which has been shown to be effective for degree I FI molars (Hamp et al., 1975), conversion of degree II or III FI molars into Degree I involved FI molars may improve their prognosis in the long term as it has been shown that FI prognosis is similar to that of a molar without FI if maintained (Salvi et al., 2014).

1.2.3 Plaque present at site

Plaque presence at site level has rarely been considered as a prognostic factor affecting the outcome of NSPT in previous studies, with plaque scores at the patient level normally being used instead. In a study assessing plaque at both the patient and site level (Tomasi et al., 2007), the patients plaque score was not found to be a significant factor, however presence of plaque at individual sites was identified to have a significant negative effect (Figure 3). The authors do make clear that prior to the baseline examination however, thorough OHI was given to all patients meaning the mean baseline initial plaque score was only 26%. This comparatively low plaque score at the start of the study period made it feasible to explore the site-specific impact of plaque on the outcome variables describing treatment success. Another study evaluating the influence of plaque at the tooth site level (Axtelius et al., 1999) also demonstrated that this had a significant negative effect on the treatment outcome.

Conversely to these studies however, another study investigating effectiveness of NSPT in the East London population in fact found that the baseline presence of plaque at a site had no predictive ability to distinguish between responding or non-responding sites (Hughes et al., 2006b).

1.2.4 Bleeding on Probing (BOP) at site

Teeth with sustained BOP represent a significantly increased risk of for disease progression also have a 46 times greater risk of being lost compared to teeth without gingival inflammation (Schatzle et al., 2004, Lang et al., 1986) .

Although NSPT substantially reduced the number of bleeding sites, more sites that remained with BOP after treatment were sites with deeper initial PPDs (31% of PPDs 7-7.5 and 50% of PPDs $\geq$8 mm) compared to shallow sites (9-16% of PPDs 3-4.5 mm) at 24 months (Badersten, 1984). Persistent bleeding was also shown to be of high predictive value for disease progression, when combined with increase in PPD or loss of probing attachment (Claffey et al., 1990, Claffey, 1991).

1.3 Tooth level Factors

These include mobility (Hughes et al., 2006b, D'Aiuto et al., 2005, Jiao et al., 2017), tooth type (Axtelius et al., 1999), bleeding index (Jiao et al., 2017).

1.3.1 Mobility

With regards to mobility, it has been demonstrated an overall poorer outcome in sites from teeth with increased mobility for teeth with grade II or III mobility in an East London population (Hughes et al., 2006b), however there was only a small proportion of teeth with such mobility.

A multilevel analysis assessing factors that affect treatment response to NSPT (Jiao et al., 2017) also demonstrated that teeth with severe attachment loss and hypermobility (degree II or III) were associated with inferior treatment outcomes. The authors suggested that this indicates that NSPT on questionable or hopeless teeth is limited, and surgical intervention or extraction should be recommended to achieve better treatment outcomes.

Another multivariate study assessing the outcome of NSPT using PPD reduction as the outcome variable indicated that mobility had an overall negative impact on PPD reductions once confounders were accounted for (D'Aiuto et al., 2005). However, the univariate analysis indicated tooth mobility was associated with better reductions in PPD. Since mobile teeth are likely to have lost a significant amount of clinical attachment and present with deep pockets, they are likely to display better improvements in PPD due to the deep pockets. Overall, it was concluded that mobility in itself had a negative impact on the clinical responses following subgingival debridement.

1.3.2 Tooth type

In a systematic review (Heitz-Mayfield, 2005) comparing surgical therapy to NSPT, two studies were found that evaluated the treatment outcome in molar and non-molar teeth (Pihlstrom et al., 1984, Lindhe et al., 1982b). Although twelve months following surgery, initially deeper sites (PPD> 6 mm) showed greater probing pocket depth reduction for non-molar teeth than for molar teeth but little difference in mean attachment levels between tooth-types and treatment modalities.

Tooth type was also explored in a multlivariable analysis assessing factors affecting NSPT response, where this model compared single-rooted (incisors and premolars) and multi-rooted teeth (molars) (Tomasi et al., 2007). A lower proababilty of “pocket closure” was found for molars pockets compared to those at single-rooted teeth, demonstrating that the location PPDs present at molar sites alongside smoking habits and supragingival plaque contribute to an inferior outcome of NSPT (Fig. 4).

The poorer outcomes for molars after NSPT are also demonstrated by other studies (Axtelius et al., 1999, D'Aiuto et al., 2005) utilising multilevel analyses. One study found that premolars as well as molars responded less well to NSPT then incisors and canines, possibly due to the higher efficacy of subgingival instrumentation at single-rooted teeth. Furthermore, thinner gingival tissues associated with anterior teeth with PPDs are likely to

heal with more recession and hence more pocket reduction than in posterior teeth (D'Aiuto et al., 2005).

In contrast to the these studies, Tomasi et al. (2007) did not consider furcation involvement in their analysis. Despite this, molars still responded less well, indicating that the poorer response in molars is not purely due to presence of FI, but may also be caused by poorer accessibility for sub-gingival instrumentation. Furthermore, the interaction with plaque presence suggests that self-performed oral hygiene by the patient is very important for PPD reduction.

A more recent multilevel analysis (Jiao et al., 2017) assessing factors effecting NSPT response in terms of PPD reduction also found that tooth type (molars vs non-molars) significantly influenced PPD reduction for all sites and for sites >5 mm. Bleeding index was also found to be influenced tooth type. Another study assessing effectiveness of NSPT found no difference between sites from single rooted versus those from multirooted teeth in NSPT response (Hughes et al., 2006b).

1.3.3 Presence of an infrabony defect

The presence of an intra-bony defects was investigated and not found to have any significant effect on the outcome of NSPT (Tomasi et al., 2007).

1.4 Patient level Factors

These include age (Jiao et al., 2017, Axtelius et al., 1999, Van der Weijden and Timmerman, 2002), gender, frequency of periodontal maintenance (FPM), smoking status (Heasman et al., 2006, Labriola et al., 2005), diabetic status, baseline full mouth plaque/bleeding scores (Van der Weijden and Timmerman, 2002).

1.4.1 Effect of Ethnicity on NSPT response

Although the response to NSPT has been investigated in different populations (Wan et al., 2009, D'Aiuto et al., 2005, Jiao et al., 2017, Tomasi et al., 2007) there has not been an investigation assessing whether ethnicity may contribute to the prognosis of NSPT.

The prevalence of periodontal disease has however been assessed, with comparisons made between different ethnic groups. Between 1971–1974, the first National Health and Nutrition Examination Survey (NHANES I) was conducted in the United States, which included a large sample representative of 194 million Americans. This survey showed that the level of oral hygiene differed significantly by race and gender (Albander, 2002a), with poorer oral hygiene found in blacks than in whites. Furthermore, different racial and ethnic groups showed marked differences in the prevalence of periodontal diseases. The NHANES I survey found a much higher occurrence of periodontitis in blacks than in whites with a Periodontal Index score of 1.28 and 0.76 in the two groups, respectively (Albandar, 2002a). Similar findings were also described in other U.S. national surveys (Albandar & Tinoco, 2002). Among the three largest race-ethnicity groups in the USA, adult blacks showed the highest prevalence of periodontitis and the most loss of periodontal tissue, followed by Mexican-Americans, whereas whites show the least disease and tissue loss. The third National Health and Nutrition Examination Survey (NHANES III) from 1988 -1994 showed that blacks have a much higher probability of having attachment loss and increased probing depth, whilst Mexican-Americans have a moderately increased probability, compared to whites. The probabilities of having attachment loss of $\geq$3mm and of $\geq$5mm, respectively, were 26% and 73% higher for blacks, and 10% and 28% higher for Mexican-Americans compared to whites. A similar pattern was observed for other periodontal whereby, 3.31 times (or 331%) more teeth in blacks, and 85% more teeth in Mexican-Americans, had probing depth of $\geq$5mm than in whites (Albander 2002b; Borrell et al., 2005).

Another cross-sectional study (Grossi *et al*., 1994) assessing an American population found that among other race-ethnicity groups with higher risk for periodontal disease, Native Americans, Asians, or Pacific Islander subjects were positively associated with a more severe bone loss (OR=2.4).

Whilst such ethnic disparities whereby predominantly white populations exhibit better periodontal health then other ethnic groups are present in the USA, Europe and Australia (Hjern & Grindefjord, 2000, Dye *et al.,* 2007, Mejia et al., 2010, Elani et al. 2012), this contrasts with the United Kingdom (UK) (Delgado-Angulo et al., 2016). The UK national Adult Dental Health Survey was last carried out in 2009, which collected data on ethnicity for the first time since 1969. However, the number of participants from ethnic minority groups was relatively small, making comparisons by ethnic groups unreliable. Initial attempts to summarise the literature suggested that patients from minority ethnic groups in the UK do not necessarily have poorer oral health (Dhawan & Bedi. 2001; Watt & Sheiham 1999) since oral health was similar among ethnic groups from the same socioeconomic position (Watt & Sheiham 1999). This did not specifically relate to periodontal health variations so whether similar patterns could be found for periodontal conditions is unclear.

Despite this, an early epidemiological study among subjects aged between 15 and 19 from the West Midlands established that the prevalence of juvenile periodontitis was higher in the Afro-Caribbean (0.8%) and Asian groups (0.2%) than in the White group (0.02%) (Saxby 1987). Another study from the UK (Mandall et al., 1998) used a stratified, random sample of 14–15 years old schoolchildren in Manchester, finding that Asian children had higher periodontal treatment needs than whites. In such studies, ethnic minorities have been grouped into broader groups, e.g., Asian, Black. This method ignores the heterogeneity that exists between ethnic groups (Nazroo 2003; Marcenes et al. 2013). A more recent study based on ethnic minorities within London, that compared to White British individuals, demonstrated that subjects from Pakistani, Indian, Bangladeshi and other Asian backgrounds had more teeth with PPD≥ 4 mm at baseline (Delgado-Angulo et al., 2016).

When analysing these differences in ethnicities and their effect on the prevalence of periodontitis and its potential effect on NSPT, we must consider whether such differences may be due to race/ethnicity per se (i.e., genetic) or confounding variables related to both ethnicity and health (Dressler et al., 2005; Nazroo 2003). When analysing a potential biologic disposition, Schenkein *et al*. (1991) assessed the neutrophil chemotaxis response to N-formyl-methyl-leucylphenyl- alanine (fMLP) antigens in periodontally healthy persons and in periodontitis patients, comparing the responses in white and black patients. They found that there may be an increased risk for periodontal disease in black patients suggesting a potential biologic susceptibility. Furthermore, significant differences between European and Chinese populations in the prevalence of interleukin -1 (IL -1) genotypes have been demonstrated (Armitage et al., 2000) which may influence the prevalence of periodontitis in these populations.

It has otherwise been hypothesised in previous studies that socioeconomic position (SEP) may fully explain the variation in the severity and prevalence of periodontal disease between ethnic groups as minority ethnic groups are disproportionately overrepresented in the lower SEP groups (Craig et al. 2001,2003). Other studies however have reported the persistence of ethnic inequalities in periodontal disease after adjustment for SEP measures (Borrell et al., 2003; Sabbah et al., 2009; Borrell et al., 2004; Jimenez et al., 2009).

The effect of SEP influencing the prevalence has been investigated including education and neighbourhood socioeconomic status. Whilst after adjustment for cofounders, black patients were twice (1.58 to 2.53) as likely to have periodontitis as white patients, those with less than a high school education were twice (1.48 to 2.89) as likely to have periodontitis than those with more than a high school diploma (Borrell et al., 2006). Furthermore, Individuals living in a neighbourhood in the lowest socioeconomic scores were 1.81 times (1.36 to 2.41) more likely to have periodontitis than those living in a neighbourhood in the highest socioeconomic scores (Borrell et al., 2006).

When ethnicity was considered in conjunction with SEP factors, black patients with higher education and income levels had a significantly higher prevalence of periodontitis than their White and Mexican American counterparts, whilst interestingly high-income black patients exhibited a higher prevalence of periodontitis than did low-income black and high-income white patients (Borrell et al., 2004). Whilst African Americans exhibited a significantly higher prevalence than whites (29.8% vs 17.7%), the effect of race may be modified by dental check-up visit frequency: African Americans with dental check-ups at least once a year had almost a fourfold higher odds of established periodontitis than their white counterparts with dental check-ups at least once a year (the referent group); while African Americans with a dental check-up once every two years or less often, were more than fourfold less likely to have established periodontitis than their white counterparts in the referent group (Borrell et al., 2003).

Additionally, the composition of minority ethnic groups varies between countries, therefore preventing any generalisation of foreign findings to a UK context. It is possible that factors influencing the periodontal status of minority ethnic groups in one country may not be relevant to other settings. As well as finding that Pakistani, Indian, Bangladeshi and Asian others had more teeth with PPD ≥ 4 mm after adjustments for demographics and SEP measures, when exploring the role of SEP in ethnic inequalities in a London population, the association of ethnicity with periodontal disease was moderated by education, but not by socioeconomic classification (Delgado-Angulo et al., 2016). Whilst there is a significant amount of literature explaining variations in the prevalence of periodontitis amongst different populations abroad, the evidence in the UK is limited due to the lack of such surveys being undertaken. Furthermore, no evidence could be found which may demonstrate differences between ethnic groups in their response to NSPT, despite varying severities of disease being present.

1.4.2 Effect of diabetes mellitus on NSPT response

Numerous studies have clearly demonstrated that diabetes mellitus (DM) is an important and significant risk factor for developing periodontal disease (Cianciola et al., 1982, Rylander et al., 1987, Hugoson et al., 1989, Emrich et al., 1991, Thorstensson and Hugoson, 1993, Yalda et al., 1994, American Academy of Periodontology, 1996, Oliver and Tervonen, 1994, Löe, 1993).

The effect that diabetes has on the outcomes on NSPT has also been evaluated. Patients with well controlled diabetes mellitus (DM) can respond well to NSPT with reduced PPDs and attachment gain (Christgau et al., 1998). With appropriate maintenance their periodontal status can remain stable over time (Westfelt et al., 1996). However, in patients with poor glycaemic control, long diabetes duration, and other diabetic complications, response to periodontal therapy is likely to be unpredictable due to compromised wound healing and tissue repair (Tervonen and Karjalainen, 1997).

A pilot study assessed the variations of periodontal health status amongst individuals with type I diabetes, as well as assessing the healing and recurrence of periodontal disease after the hygienic phase of periodontal treatment, which included OHI and SPR (Tervonen and Karjalainen, 1997). In this study there were 36 subjects with DM and ten non-diabetic controls; the diabetic group was divided into three subgroups based on the levels of glycosylated haemoglobin (HbA_{1c}) over a three-year period and the presence of diabetic complications as follows: (DI) subjects with good metabolic control and no complications (n=13). (D2) subjects with varying metabolic control with/without retinopathy (n= 15) and (D3) subjects with severe diabetes, i.e., with poor long-term control and/or multiple complications (n= 8). The study demonstrated there was no statistically significant differences in periodontal health status between subjects with DM and the non-diabetic controls. Furthermore, the level of periodontal health in D1 and D2 DM was similar to the non-diabetic controls, however D3 DM subjects had a higher proportion of sites at baseline with attachment loss ≥2 mm and a faster recurrence of deep periodontal pockets (PPD ≥ 4 mm) compared to D1 and D2 subjects indicating that increased periodontal breakdown is a complication of DM in subjects with ‘severe diabetes’. The authors commented that their results agreed with a previous study who also found more alveolar bone loss in poorly controlled diabetic compared to controlled diabetics after periodontal treatment (Seppala et al., 1993).

Another study undertook a study aimed at monitoring the clinical, microbiological, medical, and immunological effects of NSPT in diabetics (insulin dependent [IDDM] and non-insulin

dependent [NIDDM]) with HbA_{1c} scores ranging from 4.4-10.6% and comparing these to healthy controls (Christgau et al., 1998). Periodontal examinations including PPD, PAL and BOP, as well as microbiological examinations (culture), and immunological examinations were performed at baseline, then two weeks following supragingival scaling and then four months after subgingival therapy. The median % per patient of pockets with deep pockets (PPD≥4 mm) decreased from 41.9% to 28.3% in diabetics compared to 41.6% to 31.8% in controls, whilst microbiologically there were similar reductions of periopathogenic bacteria found in both diabetics and controls. Furthermore, there was no signicantly difference between diabetics and controls with regards to the oxidative burst response of Polymorphonuclear leukocytes (PMNs). As with previous studies, the authors also concluded that metabolically well-controlled diabetics may also respond as well as healthy controls to NSPT.

Another study also compared the outcomes of periodontal treatment which included NSPT and MWF for residual sites (PPD >5 mm at 6-month reassessment) between patients who had moderate to advanced periodontal disease with diabetes (type 1/IDDM and type 2/NIDDM) to healthy control patients with periodontal disease. (Westfelt et al., 1996). These patients were followed up for five years during which time a plaque control programme was repeated every three months and re-examinations of plaque, gingivitis, PPD and PAL performed 12, 24 and 60 months after the baseline examination. This study again also found that diabetics and non-diabetics alike, treated for moderately to advanced forms of periodontitis were able to maintain healthy periodontal conditions. Thus, the frequency of sites which exhibited signs of recurrent disease was similar in both groups.

Another study analysed the response to NSPT in subjects with type II DM with a HbA_{1c} >7% and <9% using data from a multicentre Diabetes and Periodontal Therapy Trial (DPTT). (Michalowicz et al., 2014). Multiple regression models were used to evaluate patient level factors that affected the treatment response. This study indicated that baseline severity of periodontal diseases was associated with the clinical response as previously discussed, but with regards to DM, found that glycaemic control and diabetes duration were not useful predictors for outcomes of NSPT in these patients.

Another study (Chen et al., 2012) evaluated the effect of NSPT on the metabolic control of patients with DM and periodontal disease, noting the reciprocal association between periodontal disease and diabetes mellitus, firstly as diabetics are more likely to develop periodontal disease and periodontal infection may have an adverse effect on glycaemic control and the incidence of diabetes complications (Taylor and Borgnakke, 2008). Diabetes and periodontal disease also have similar polygenic backgrounds (Covani et al., 2009) and are relatively widespread prevalence in the general population, with similar clinical risk factors, such as smoking, age, psychologic stress (Xiao et al., 2009), and neutrophil functional alterations (Gursoy et al., 2008). Additionally, both can upregulate the systemic immune response (Nassar et al., 2007) with some degree of immunoregulatory dysfunction. Furthermore, it has been suggested that periodontal therapy, which decreases the intraoral bacterial load and reduces periodontitis-induced bacteraemia, may have a positive impact on systemic inflammatory status and therefore improve metabolic control in patients with diabetes (Taylor and Borgnakke, 2008). A systematic review (Simpson et al., 2015) confirmed there is evidence to support an improvement in glyacmic control measured by a reduction in HbA1c of 0.43% three to four after NSPT. This reduction was in HbA1c was 0.30% at six months and improved after 12 months to 0.50%.

1.4.3 Effect of cigarette smoking on NSPT response

A significant difference has been found for pocket closure after NSPT when comparing smokers and non-smokers (Labriola et al., 2005, Heasman et al., 2006). Other studies have also demonstrated a poorer response to treatment in smokers compared to never or former smokers (Ah et al., 1994, Kaldahl et al., 1996, Grossi et al., 1997, Kinane and Radvar, 1997, Tonetti et al., 1998, Trombelli et al., 2003, Stavropoulos et al., 2004, Paulander et al., 2004, Rieder et al., 2004, Sculean et al., 2005, Wan et al., 2009). Meta-analyses also demonstrate the effects of smoking on the outcome of periodontal therapy (Garcia, 2005, Labriola et al., 2005, Patel et al., 2012).

In a study investigating prognostic factors affecting NSPT (Tomasi et al., 2007) with 'pocket closure', as the main outcome, smoking was found to have a significant negative impact when compared to non-smokers (Figure 5), as smokers had a 0.5 mm higher PPD three months after treatment compared to non- smokers. The negative impact of smoking on was also more pronounced for deep sites than for shallow sites.

In a systematic Review evaluating the effect of smoking on NSPT (Labriola et al., 2005), the difference in full-mouth probing depth reduction after NSPT between smokers and non-smokers was assessed in six studies, five of which showed a better response in non-smokers equating to a 0.13mm greater PPD reduction in non-smokers than in smokers with no evidence of heterogeneity between these studies (Preber et al., 1995, Grossi et al., 1997, Haffajee et al., 1997, Machtei et al., 1998, Preshaw et al., 1999, Winkel et al., 2001).

A separate analysis (Labriola et al., 2005) was undertaken for sites which had an initial probing depth of ≥5 mm for which eight available studies were included (Preber et al., 1995, Grossi et al., 1997, Pucher et al., 1997, Renvert et al., 1998, Palmer et al., 1999, Ryder et al., 1999, Mongardini et al., 1999, Williams et al., 2001). A random effects meta-analysis indicated a weighted mean difference showed that non-smokers had 0.43 mm more PPD reduction than smokers, however for this parameter there was significant heterogeneity between studies (Figure 7).

Meta-analyses comparing PPD reduction in quit smokers to non-smokers, firstly for all sites based on three studies (Grossi et al., 1997, Haffajee et al., 1997, Preshaw et al., 1999), and then for sites initally ≥5 mm, based on two studies (Grossi et al., 1997, Ryder et al., 1999) both showed no statistically significant differences (Labriola et al., 2005).

Another study evaluating response to NSPT in the East London population also demonstrated that smoking status was statistically associated with response to treatment and emphasised smoking as a negative prognostic factor (Hughes et al., 2006b). A further multilevel analysis evaluating factors affecting the outcome of NSPT also confirmed that indicating that cigarette smoking negatively affects the outcome of NSPT, with smokers having 0.23 mm less PPD reduction than non-smokers (D'Aiuto et al., 2005). The authors also commented that although this seems like a small difference, it actually repsrents that smokers had 20% less PPD reduction compared to the overall population.

The effect of non-surgical treatment on periodontal pockets in smokers and non-smokers has been assessed in previous studies assessing PPD reductions (Preber and Bergström, 1986b) and the effect on gingival bleeding (Preber and Bergström, 1986a). NSPT can reduce PPD in smokers and non-smokers, however as demonstrated in other studies,

smokers have less PPD reduction (Preber and Bergström, 1986b, Tomasi and Wennstrom, 2004), with the greatest difference in this study noticed in the maxillary anterior region (Preber and Bergström, 1986b). Smokers also achieved less reduction in bleeding compared to non smokers, and it was noted that gingival inflammatory symptoms seem to be suppressed in patients who smoke (Preber and Bergström, 1986a). A further study also confirmed that smokers respond less favourably to non surgical and surgical periodotnal therapy in patients with moderate to advanced periodontitis, achieving less PPD reduction then non-smokers, less gain in PAL following active treatment. These differences were observed during each year of maintenance when a greater loss of horizontal attachemnt level was noticed (Ah et al., 1994). These findings were most pronounced for the deepest pockets (PPD > 7 mm). This concurs with a similar study which concluded that smoking is an important prognostic factor for periodontal treatment particularly in persistent and deeper pockets, where smokers had even less PPD reduction than non-smokers (Kinane and Radvar, 1997). Ah et al (1994) also observed that smokers had slightly more supragingival plaque and BOP. When undertaking a multivariate analysis evaluate the outcome of NSPT, smokers were again considered to have a limited repsonse, however plaque control and initial PPD ≥6 mm were also considered to be significant predictors for teeth requiring further treatment, leading the authors to suggest that deep sites in smokers should proceed straight to surgical intervention rather then NSPT first (Papantonopoulos, 1999a). Site specific analysis to evaluate the effect of smoking following non-surgical and surgical periodontal therapy has also demosntated that smokers respond less favourably compared with non-smokers, in particular at plaque-positive sites (Bunaes et al., 2015).

In a Chinese population where the response to SRP in smokers and non-smokers was evaluated over six months also found that smokers had a different healing response to non-smokers (Jin et al., 2000). Smokers and non-smokers exhibited significant decreases in PPD at one month, and a greater reduction of PPD was found in non-smokers than in smokers with the most notable difference in PPD reduction at three months (2.4 mm for non-smokers vs. 1.1 mm for smokers). Non-smokers showed a consistent gain of attachment throughout treatment up to six months whereas there was no significant gain of attachment for smokers until six months. PPD reduction was positively correlated with baseline PPD for both groups, but when only initial PPDs ≥ 5 mm were included, then only non-smokers had a significant correlation. A multilevel regression analysis investigated the factors to predict NSPT response, and also determined in a Chinese population that non-smokers, anterior teeth and sites with plaque at baseline were associated with a greater PPD reduction over 12 months, with smokers showing less favourable PPD reduction especially at deeper sites .(Wan et al., 2009).

A further study comparing the outcome of NSPT between non-smokers and smokers also investigated the microbiological effects of NSPT in both groups (Preber et al., 1995). Periodontal indices (plaque index, gingival index, PPD) were recorded and bacterial samples collected at baseline and two months after NSPT was performed by a dental hygienist. PPD was reduced in both groups, but as with the previous studies, PPD reduction was significantly smaller in smokers than non-smokers. Microbiologically, there was almost total eradication of A.a and P.g in both groups, however 9/11 eleven smokers and 5/10 non-smokers remained positive for Prevotella intermedia after treatment. This study again highlighted the less favourable clinical outcome of NSPT in smokers compared to non-smokers despite that NSPT was equally effective in reducing periodontal pathogens.

Another study also investigated the effect of cigarette smoking on patients clinical and microbiological responses to mechanical therapy (Grossi et al., 1997). When comparing ex-smokers to non-smokers, the healing and microbial response was similar. However, after four to six sessions of subgingival SRP and OHI, current smokers had less healing and reduction in subgingival Bacteroides forsythus and P.g after treatment compared to former and non-smokers, suggesting that smoking impairs periodontal healing. This study

compared former as well as current smokers to non-smokers and concluded that as the healing and microbial response of former smokers is comparable to that of non-smokers, smoking cessation may restore the normal periodontal healing response. However as with Preber et al. (1995) a further trial investigating the microbial response after NSPT in smokers and non-smokers found that whilst less PPD reduction was achieved for smokers, similar reductions in of P. g and P. intermedia/nigrescens and slight increases of A.a were found in both smokers and non-smokers. They therefore concluded that the microbiological response found in this study was not influenced by smoking habits (Renvert et al., 1998).

Conversely to the previous studies mentioned, a clinical trial evaluating the outcome of NSPT in non-smokers and smokers at nine months actually found that there was no significant differences between smokers and non-smokers comparing PPD, clinical attachment level, plaque index, BOP, and gingival index, indicating that smokers and non-smokers responded similarly (Pucher et al., 1997). The authors cite possible reasons for possible differences compared to other studies including that suboptimal initial periodontal therapy as only one hour of subgingival SRP was completed. Furthermore, only sites with moderate to severe periodontal disease (5-8 mm) were included, which narrowed the focus of the study to those that would respond to initial periodontal therapy, whereas other studies (Ah et al., 1994, Kaldahl et al., 1996) utilised full mouth measurements where data from sites with minimal PPD was pooled with PPDs from moderate to severe sites. A further study also agreed that smokers and non-smokers respond comparably to NSPT, as well as being comparable to ex-smokers (Preshaw et al., 1999) although smokers had deeper probing depths than non- and ex-smokers.

The most recent meta-analysis evaluating the impact of smoking on clinical outcomes of NSPT (Chang et al., 2021) based on differences in PPD and CAL gain included seventeen studies (AlAhmari et al., 2019, Guru et al., 2018, Türkoğlu et al., 2016, Dodington et al., 2015, Feres et al., 2015, Ardais et al., 2014, Preshaw et al., 2013, Eltas and Orbak, 2012, Wan et al., 2009, Hughes et al., 2006a, Apatzidou et al., 2005, D'Aiuto et al., 2005, Jin et al., 2000, Palmer et al., 1999, Renvert et al., 1998, Preber et al., 1995, Preber and Bergström, 1986b). PPD reduction was found to be smaller for smokers compared to non-smokers (weighted mean difference in PPD reduction: −0.33 mm, 95% confidence interval (CI): [−0.49, −0.17], $p < .01$), whilst CAL gain in smokers was also smaller than for non-smokers (weighted mean difference in CAL gain: −0.20 mm, CI: [−0.39, −0.02], $p < .01$). This study also confirmed that baseline PPD significantly affected the difference in PD reduction between two groups.

1.4.4 Baseline Plaque score at patient level

As previously discussed, presence of plaque at the individual tooth site had a significant negative effect, however the patient's plaque score at baseline did not have a significant impact on the outcome of 'pocket closure' (Tomasi et al., 2007). Furthermore, a multilevel analysis of factors influencing the six-month clinical outcome of subgingival debridement (D'Aiuto et al., 2005) also reported a non-significant effect of the full-mouth plaque score on both the final PPD and the change in PPD.

When used to assess prognostic factors for outcome of NSPT for treatment of generalised aggressive periodontitis in the East London population, the patient's plaque score on the subject level was not found to be associated with the outcome of initial cause-related therapy (Hughes et al., 2006b). Additionally, baseline plaque presence at site level did not have any predictive ability to distinguish between responding or non-responding sites.

The diagnostic predictability of plaque scores to predict attachment loss was 28% at 36 months assuming a plaque frequency of >75%, meaning that 28% of sites with supragingival plaque present at 75% or more of the examinations between 6-36 months had undergone probing attachment loss during 0-60 months after NSPT (Badersten et al., 1990) demonstrating that plaque presence is a poor indicator of progression of attachment loss.

1.4.5 Baseline Bleeding score at patient level

When used to assess prognostic factors for outcome of NSPT for treatment of generalised aggressive periodontitis in the East London population, the patient's bleeding score did not have any significant predicative ability despite the fact that there was a significant reduction in BOP following treatment in responding sites compared to non-responding sites (Hughes et al., 2006b).

Bleeding frequency ≥75% reached a diagnostic predictability of 23% at 36 months, meaning that 23% of sites which had BOP at 75% or more during examinations 6-36 months post NSPT had undergone probing attachment loss during 0-60 months. Furthermore, suppuration had diagnostic predictability of 20% however no site demonstrated suppuration more than three times during entire 18-60 month period so was difficult to use as a predictor (Badersten et al., 1990).

1.5 Operator level Factors

1.5.1 Hand vs machine driven instrumentation

Various instruments may be appropriate for subgingival instrumentation, demonstrating differences in the removal of soft and hard subgingival deposits (Lea et al., 2003, Leknes et al., 1994). Several recent systematic reviews support the efficacy of NSPT but found few differences between instrumentation types (Tunkel et al., 2002, Van der Weijden and Timmerman, 2002, Hallmon and Rees, 2003, Suvan et al., 2020). Hand, sonic, and ultrasonic instruments produce similar periodontal healing response measured by PPD reduction, BOP reduction and CAL gain (Badersten et al., 1981, Badersten et al., 1984, Lindhe and Nyman, 1985, Kalkwarf et al., 1989, Loos et al., 1987, Copulos et al., 1993, Obeid et al., 2004, Wennstrom et al., 2005, Christgau et al., 2006).

Furthermore, ultrasonic/sonic subgingival debridement requires less time than hand instrumentation (Tunkel et al., 2002). Additionally, sonic and ultrasonic scalers produce less root surface loss than hand instruments and cause less soft tissue trauma (Ritz et al., 1991, Busslinger et al., 2001, Schmidlin et al., 2001, Kawashima et al., 2007) whilst providing better access to deep pockets and furcation areas (Kocher et al., 1998, Beuchat et al., 2001). Ultrasonic or sonic scalers are also less operator-dependent and require less treatment time, while resulting in a rougher root surface (Breininger et al., 1987). Despite these benefits, tactile sensation is reduced, and contaminated aerosols are produced with powered instrumentation (Barnes et al., 1998, Harrel et al., 1998, Rivera-Hidalgo et al., 1999, Timmerman et al., 2004). Furthermore, hand instrumentation may result in smoother tooth surfaces and may remove more calculus deposits (Rateitschak-Pluss et al., 1992).

The most recent systematic review (Suvan et al., 2020), compared the effectiveness of powered instrumentation to hand instrumentation for subgingival debridement. Six randomised controlled trials (Ioannou et al., 2009, Laurell and Pettersson, 1988, Malali et al., 2012, Obeid et al., 2004, Petelin et al., 2015, Wennstrom et al., 2005) were found which specifically compared hand and sonic/ultrasonic instruments for subgingival treatment. Meta-

analysis was possible for PPD reduction and CAL gain, but not for any other outcomes. The findings of this review confirmed the findings of previous reviews (Drisko et al., 2000, Krishna and De Stefano, 2016, Tunkel et al., 2002) in that no significant differences were observed between treatment groups at any time point or for different categories of initial pocket depth.

It was however noted that a variety of different instruments in terms of manufacturer, design, and technology were used in the different studies, and there was therefore considerable heterogeneity amongst the studies, and further advances in the development of both types instrumentation may make the findings of such studies less relevant. An example of such advances include use of a slimline ultrasonic insert, which showed superior effectiveness compared with a standard ultrasonic insert, whereby 42% of the surfaces in pockets >6 mm were covered with residual calculus after instrumentation, compared to 34% after instrumentation using the slimline insert (Clifford et al., 1999). Additionally specially designed Gracey curettes were more effective in penetrating deep pockets compared to standard Gracey curettes (38% and 42% of surfaces with residual deposits, respectively) (Nagy et al., 1992), whist specially designed ultrasonic scaler tips for molars were more effective than standard tips for removal of deposits in maxillary molars (50.3 vs. 15.1% of surfaces with residual calculus) and mandibular molars (44.1 vs. 16.7% of surfaces with residual calculus) (Oda and Ishikawa, 1989). Furthermore, in clinical practice, operators will often combine the use of hand and power-driven instruments when undertaking PMPR and subgingival instrumentation.

1.5.2 Previous NSPT

It could be speculated that repeated instrumentation of root surfaces might increase the efficacy of treatment; however, it has been demonstrated that a single episode of ten minute NSPT using curettes was as effective as two episodes of ten minute each within a 24 h interval (Anderson et al., 1996). Furthermore, for patients who have had NSPT, only 11–16% of poorly responding sites may reach a successful end point following repeat NSPT. Furthermore, half of deeper sites (PPD >7 mm) remain as ‘non-successful sites’ in terms of limited PPD reduction or CAL gain (Wennstrom et al., 2005). It has therefore been shown that the probability of achieving pocket closure three months after repeat NSPT was ~45% overall, but only 12% for pockets over 6 mm (Tomasi et al., 2008). Pockets associated with molars, furcation sites, and angular bone defects have been shown to respond even less favourably to repeated NSPT (Axtelius et al., 1999, D'Aiuto et al., 2005, Tomasi et al., 2007).

1.5.3 Operator experience

Studies comparing the ability of ‘periodontal residents’ with periodontists or undergraduate dental students to remove calculus from root surfaces demonstrated that experience and skill significantly affect the outcome (Fleischer et al., 1989, Kozlovsky et al., 2018). Conversely, a previous study comparing a periodontist to dental hygienists found that operator experience had little influence on the outcome (Badersten et al., 1985a).

Training of the operator is a further parameter that affects the efficacy of root debridement, especially when using hand instruments. Studies demonstrated that less experienced periodontal residents or dental hygienists were not as effective at producing calculus- free surfaces in periodontal pockets as experienced periodontists (Breininger et al., 1987, Fleischer et al., 1989).
The effect of operator experience was demonstrated for both closed and open procedures, with trained periodontists removing significantly more calculus (19% of surfaces with residual calculus) than residents (66% surfaces with residual calculus) during a closed procedure on

pockets >6 mm using a combination of curettes and ultrasonic instruments (Breininger et al., 1987).

Subsequent studies in phantom heads that showed that approximately 15– 24% of all surfaces still had deposits after treatment by an inexperienced operator, compared with 13% residual deposits after root instrumentation by an experienced operator (Kocher et al., 1997, Ruhling et al., 2002).

1.5.4 Full mouth scaling/disinfection vs quadrant SRP

'Full mouth Scaling' (FMS) refers to completion of full mouth instrumentation within a 24-hour period whereas 'Full Mouth Disinfection' (FMD) supplements FMS with the use of chlorhexidine gluconate (CHX) for full mouth sensing, subgingival irrigation, tonsil spray and tongue brushing (Quirynen et al., 1995). Recent Cochrane and systematic reviews have found little difference between the effectiveness of FMS/FMD over quadrant SRP (Suvan et al., 2020, Eberhard et al., 2015). Whichever approach that is chosen is influenced by patient convenience (Eberhard et al., 2015). Regarding cost-benefit considerations, FMD/FMS results in comparable healing (% PPD <4 mm at six months after treatment) to conventional SRP (Wennstrom et al., 2005, Zanatta et al., 2006, Del Peloso Ribeiro et al., 2008). The efficiency of FMS, measured as the mean time to achieve a closed pocket, was three times better using an ultrasonic device alone than SRP using hand instruments (Wennstrom et al., 2005).

A recent systematic review specifically addressed the comparison between quadrant-wise and full mouth approaches for subgingival instrumentation (Suvan et al., 2020) which included thirteen randomised controlled trials (Apatzidou and Kinane, 2004, Del Peloso Ribeiro et al., 2008, Fonseca et al., 2015, Jervøe-Storm et al., 2006, Koshy et al., 2005, Loggner Graff et al., 2009, Meulman et al., 2013, Predin et al., 2014, Quirynen et al., 2006, Swierkot et al., 2009, Wennstrom et al., 2005, Zanatta et al., 2006, Zijnge et al., 2010). Meta-analysis was undertaken for PPD reduction, CAL gain and pocket closure; no significant differences were observed between treatment groups irrespective of time point or initial pocket depth.

1.6 Outcome measures in the Literature

When undertaking periodontal treatment, the ultimate goal is to avoid tooth loss which represents a deterioration of function and aesthetics, as well as quality of life (Araújo et al., 2010). Therefore 'tooth loss' represents a true outcome assessment of efficacy in clinical trials investigating periodontal treatment (Tomasi and Wennström, 2017).

True endpoints are outcomes that directly measure how a patient feels, functions or survives and are tangible to the patient (Hujoel, 2004). Other than tooth loss, true endpoints may also include subjective oral health-related quality-of-life measurements (Leao and Sheiham, 1996, Leake, 2002) or self-reported symptoms such as bleeding after brushing.

In studies evaluating the outcome on NSPT which normally are short term studies with short observation periods, 'tooth loss' rarely occurs and therefore not a discriminating variable to compare the efficacies of various therapeutic means. Surrogate outcome measures can instead be applied, which should be easy to measure, reproducible, sensible [i.e. reflecting changes potentially measurable after a short time], and valid [demonstrated by correlation with true outcome variables] (Tomasi and Wennström, 2017). Unlike true endpoints, surrogate endpoints are intangible to the patient's mind and include changes in Probing attachment level or gingival crevicular fluid markers (Hujoel, 2004). Surrogate endpoints are often objective because they can be measured by the clinician (rather than relying on self-report by patients) or by laboratory methods. Typical surrogate endpoints in periodontology include anatomic measures (e.g., probing pocket depth), measures of inflammation (plaque index, gingival index), microbiological measures, and immunologic measures (Hujoel, 2004).

Historically, PPD was the main outcome measure for periodontal treatment success as treatment was aimed at pocket elimination (Gottlieb, 1928), however between the 1970s until 1992, attachment levels became the gold standard as treatment focused on maintaining these (Goodson, 1986, Koch and Paquette, 1997, Ramfjord et al., 1973). More recently, PPD has again become the main outcome measure as it allowed site-specific antimicrobials to be evaluated (Hujoel, 2004).

In a recent systematic review evaluating the efficacy of subgingival instrumentation, NSPT was demonstrated to be efficacious in terms of PPD reduction, however disease resolution measured by pocket closure (PPD <4 mm or PPD=4 mm with no BOP), was less likely (Suvan et al., 2020). Shallow PPD and absence of BOP are reasonable endpoints for NSPT that represent an absence of clinical signs of inflammation (Loos and Needleman, 2020, Suvan et al., 2020). As discussed, PPD and clinical attachment level (PAL/CAL) are the most commonly reported outcomes in studies, however an ideal endpoint of therapy should be clinically meaningful and represent a clear and tangible benefit for patients, whilst being relevant to the goal of therapy that is being utilised. As the main aim of NSPT is to achieve infection control, measured by absence of clinical signs of inflammation and increased resistance to probing, PPD reductions as well as frequencies of closed pockets (probing pocket depth ≤ 4 mm and absence of BOP) were the outcomes utilised in this review.

1.6.1 Mean PPD

The decision to undertake periodontal treatment is based on the presence of pathological pockets i.e., deepened pockets and bleeding on probing. A successful clinical outcome for NSPT would therefore be shallow probing pocket depths without bleeding, which would indicate sufficient removal of biofilm/calculus and resolution of the inflammatory lesion (Tomasi and Wennström, 2017). Previous meta-analyses analysing periodontal treatment outcomes have often utilised PPD reductions as a surrogate outcome variable as increased

pocket depths are associated with disease progression and tooth loss (Badersten et al., 1990, Claffey and Egelberg, 1995, Lang et al., 2008b, Matuliene et al., 2008, Westfelt et al., 1998).

A study that included 172 subjects followed up for a mean of eleven years after active periodontal therapy (Matuliene et al., 2008) found that compared to a PPD of ≤ 3 mm, a remaining PPD of 5 mm represented a risk factor for tooth loss with an odds ratio of 7.7, The corresponding odds ratio for a remaining PPD of 6 mm and ≥ 7 mm was 11 and 64, respectively. This paper highlights how residual PPDs>5 mm can progress to tooth loss and therefore highlights the importance of using PPD as a surrogate outcome variable.

Many studies investigating prognostic factors that may have a significant impact on the outcome of NSPT have utilised PPD changes as a surrogate outcome variable, especially as tooth loss is not a feasible short-term variable for clinical trials (D'Aiuto et al., 2005, Tomasi et al., 2007, Wan et al., 2009, Jiao et al., 2017). PPD has also been used as a primary outcome variable in studies examining the effectiveness of NSPT (Badersten et al., 1981, Badersten et al., 1984, Axtelius et al., 1999, Grossi et al., 1997, Winkel et al., 2001, Westfelt et al., 1996, Renvert et al., 1998, Lindhe et al., 1982b).

1.6.2 Bleeding on Probing

The long-term influence of the variable “bleeding on probing” (BOP) on tooth loss was addressed in a 26-year longitudinal study (Schätzle et al., 2004), and revealed that teeth that at all examinations were positive for bleeding on probing had a 46-time higher risk of being lost compared to teeth not showing gingival inflammation. Furthermore, the absence of bleeding on probing (BOP) following treatment is a strong negative predictor of further clinical attachment loss (98% predictive value; Lang et al., 1990).

BOP is not always included as a surrogate variable analysis of prognostic factors of NSPT but is often measured during clinical examination of the patients included in these studies (Tomasi et al., 2007, Badersten et al., 1981, Badersten et al., 1984, Wan et al., 2009). A recent systematic review analysing the effect of subgingival instrumentation found that such treatment caused a mean BOP reduction of 62% (Suvan et al., 2020) and it has also been used as a secondary outcome measure in assessing the efficacy of periodontal treatment (Claffey and Egelberg, 1995, Cobb, 2002, Haffajee et al., 1997).

1.6.3 Mean CAL change/ Loss of attachment

Clinical attachment level (CAL) has also previously been used as a surrogate outcome in previous studies analysing effectiveness of NSPT or factors affecting NSPT (Claffey and Egelberg, 1995, Haffajee et al., 1995, Haffajee et al., 1997, Grossi et al., 1997, Badersten et al., 1981, Badersten et al., 1984, Axelsson and Lindhe, 1981, Winkel et al., 2001, Westfelt et al., 1996, Trombelli et al., 2015, Lindhe et al., 1982b) and is useful as it can be linked to tooth loss in the long term.

Loss of CAL after treatment may reflect failure to prevent disease progression or damage to the supporting tissues caused by treatment. However as NSPT is unlikely to form new connective tissue attachment, any gains in CAL may actually only reflect the increased resistance to probing as a result of resolution or reduction of the inflammatory lesion in the tooth-bordering soft tissues (Tomasi and Wennström, 2017).

Previous studies have also shown that the magnitude of potential CAL gain is related to initial PPD, such that the deeper the baseline PPD, the greater the potential for reduced probe penetration following successful NSPT and therefore for increased CAL gain

(Badersten et al., 1984). It has therefore ben suggested that any reported benefit of a supposed adjunct to NSPT based solely on reported differences in the magnitude of mean CAL gain is highly doubtful and provides limited if any support for clinical decisions since its validity in relation to the true outcome variable and ultimate goal of treatment is not proven (Tomasi and Wennström, 2017).

1.6.4 Pocket closure (site level)

The goal of periodontal treatment is to obtain shallow probing pocket depth (PPD<4 mm) and absence of bleeding, indicating sufficient removal of biofilm/calculus and subsequent resolution of the inflammatory lesion (Lang and Lindhe, 2015, Loos and Needleman, 2020). Pocket closure is an essential outcome variable as it demonstrates a reduced risk of disease progression (Westfelt et al., 1998, Badersten et al., 1990, Claffey and Egelberg, 1995, Lang and Tonetti, 2003, Matuliene et al., 2008). The clinical value of this variable is also validated by data demonstrating (i) lower risk for disease progression in patients with non-bleeding shallow pockets (Badersten et al., 1990, Claffey, 1991, Claffey and Egelberg, 1995, Lang and Tonetti, 2003), (ii) the effectiveness of pocket reduction in changing subgingival environmental conditions and microbial composition (Mombelli et al., 1995), and (iii) the risk of attachment loss in sites with PPD>6 mm (Westfelt et al., 1998).

In a recent systematic review investigating the outcome of NSPT (Suvan et al., 2020), pocket closure was therefore considered an important component to evaluate treatment efficacy. However, pocket closure was not consistently reported and was defined in different ways, that is with or without the measure of BOP. In this review, an overall proportion of pocket closure of 74% at 6-8 months after subgingival instrumentation was observed (Suvan et al., 2020)

Pocket closure represents a clinical endpoint of periodontal treatment success and is defined as firstly having no BOP and secondly achieving “closed pockets”, where PPD achieves a depth of ≤ 4 mm. Using pocket closure as a clinical endpoint following various treatment approaches provides information that is more easy to interpret and compare than commonly reported mean PPD or CAL differences of fractions of a millimetre between procedures, and directly transferable to daily practice and communication with the patient (Tomasi and Wennström, 2017). Further, when this outcome is applied with the use of a multilevel logistic analysis it allows a comprehensive and clear assessment of factors that may influence treatment outcome (Tomasi and Wennström, 2017).

Pocket closure (PPD<4 mm) was the main clinical endpoint used in a multilevel study investigating factors influencing the outcome of NSPT at three-month reassessment (Tomasi et al., 2007). The authors commented that using this outcome measure implies the restriction of the initial sample to the sites >5 mm depth at baseline, however these are the sites that require root debridement. By using this outcome variable, a prediction table for different variables that can affect the clinical outcome of initial phase of NSPT could be created. They concluded that the probability of achieving ‘‘pocket closure’’ three months after subgingival debridement at a site with an initial PPD of 6 mm was at best 84% (single rooted tooth without plaque at baseline in a non-smoker), and decreased markedly for greater initial PPD, presence of plaque at baseline, location at a multi-rooted tooth and/or if the patient was a smoker (Tomasi et al., 2007).

1.6.5 Pocket closure (patient level)

Periodontal stability at the patient level based on the new classification can be defined as having PDDs<4 mm, with no sites that have PPD of 4 mm also having BOP, and BOP being less then 10% (Lang and Bartold, 2018, BSP, 2018).

Although this has not been specifically set as a clinical endpoint for periodontal treatment as it is difficult to achieve in clinical practice, another study has previously utilised a patient specific outcome measure analysing the response to treatment of deep PPDs at baseline (Hughes et al., 2006b). Hughes et al (2006b) determined the percentage of sites for each patient that were designated as non-responding (i.e., showed no improvement following treatment). The study group were dichotomised into ‘‘responding’’ and ‘‘non-responding’’ patients, nonresponding patients being defined as those with a minimum of 30% of their deep sites that did not respond to the treatment provided. The authors note that this 30% value was chosen arbitrarily as a clinically significant poor response to treatment.

1.6.6 Gingival and Plaque Indices

Both gingival indices and plaque indices are often used as secondary outcome measures when the outcome of NSPT is being assessed (Isidor et al., 1984, Jiao et al., 2017, Preber and Bergström, 1986b, Lindhe et al., 1982b, Axelsson and Lindhe, 1981, Badersten et al., 1981, Badersten et al., 1984, Hughes et al., 2006b)

1.6.7 Recession

Recession is not always specifically specified as an outcome measure for NSPT but is often measured in such studies (Badersten et al., 1981, Badersten et al., 1984, Isidor et al., 1984). However since the reduction/elimination of the inflammatory lesion in the gingiva will cause retraction of the gingival margin with potential aesthetic inferences, assessment and description of soft tissue, recession may be considered (Tomasi and Wennström, 2017).

1.6.8 Patient Related Outcome Measures (PROMs)

PROMs allow clinical outcomes to be related to patient’s perceptions and expectations (Tomasi and Wennström, 2017), however are rarely reported in studies evaluating the outcome of NSPT.

CHAPTER 2: SUMMARY AND PROJECT PLANS

2.1 Hypotheses

Primary hypothesis
NSPT outcomes are poorer in South East Asian Ethnicity (SEA [Indian, Pakistani and Bangladeshi]) groups compared to other ethnic groups in patients attending postgraduate specialist clinic at The Royal London Dental Hospital in East London

Secondary hypothesis
Changes in mean PPD following NSPT may be influenced by multiple factors including:

- Smoking status
- Diabetes
- Plaque scores
- Stress
- Family History of Periodontitis
- Sex
- Frequency of attendance
- Operator factors including number of treatment sessions
- Type of Toothbrush used
- Time between treatment and Reassessment
- Baseline disease severity (Mean PPD)

2.2 Aim

The aim of this hospital-based retrospective study is to evaluate factors influencing the effectiveness of NSPT in patients referred for periodontal treatment at Royal London Dental Hospital using data extracted from their clinical records.

2.3 Objectives

Primary Objectives

- To describe the baseline population of patients referred to the Royal London Dental Hospital for periodontitis treatment
- To determine which factors may affect the response to NSPT using bivariate analysis
- Using multivariable analysis, to determine the relative influence of each independent predictor variable on the dependent outcome variable (PPD reduction/pocket closure)

Secondary Objectives

- To describe the levels of severity of periodontitis in an East London population receiving secondary NHS care
- To examine what risk factors may be more common in the South-East Asian ethnic group compared to all other groups combined

CHAPTER 3: MATERIALS & METHODS

3.1 Study population

Patients who have received NSPT in the Department of Restorative Dentistry, Royal London Dental Hospital and have had at least one periodontal re-evaluation record from January 2017 to January 2021 will be included in this retrospective study. Patients were identified consecutively from previous 'new patient' consultant clinic lists, where patients had been referred to by their general dental practitioners specifically in relation to their periodontal health (PR, NG, NM, ND).

The study was approved by local ethics committee (EDGE ID: 128856).

Inclusion criteria are as follows:

- Patients > 18 years old
- Patients with either aggressive or chronic periodontitis (Armitage, 1999) or Stage I, II, III, IV periodontitis severity (Papapanou et al., 2018).
- Patients with complete baseline data (including completed periodontal new patient assessment form- Appendix 2) and a follow-up of at least 6 weeks (on periodontal reassessment proforma – Appendix 3)
- Patients who had initial NSPT by postgraduate periodontology students, restorative registrars (e.g., SPR), periodontology consultants, or research fellows (e.g. ITI scholars)

Exclusion criteria:

- Patients referred for treatment for mucogingival conditions e.g. gingival recession, and who did NOT have Stage I, II, III, IV periodontitis severity (Papapanou et al., 2018).

3.2 Sample size calculation

There is a lack of studies reporting data on demographic information in highly mixed populations. Therefore, this study was deemed exploratory. For detecting a difference of 0.15 mm of probing depth change between the test and reference group (e.g. ethnic groups), and assuming a pooled standard deviation of 0.28 mm, the study required a sample size of 55 for each group (total of 110), to achieve a power of 80% and a level of significance of 5% (Dhand, 2014). Also, a patient sample of 110 would allow inclusion of up to five predictors in a multivariable analysis. Due to the limitations of data collection described, a convenience sample of 108 subjects was obtained given that this study was primarily exploratory.

3.3 Periodontal examinations and treatments

All periodontal examinations and treatments were performed by qualified dentists undergoing postgraduate training in periodontology (DClinDents/ Academic Clinical Fellows) or speciality registrars in restorative dentistry (SPRs) or dental hygiene/therapists with supervision from specialist level periodontists.

Initial periodontal examination included medical history, history of present complaint, social history (including smoking, use of tobacco, alcohol, stress), information on oral hygiene (including toothbrushing, interdental aids, mouthwashes), dental history (including previous

periodontal care, oral hygiene, orthodontic treatment) as well as baseline six point pocket chart where probing pocket depth (PPD), recession, bleeding on probing and plaque are recorded at six sites per tooth and for each tooth mobility (Miller, 1950) and furcation involvement (Hamp et al., 1975) has been assessed and recorded. This information was normally recorded on the "Periodontal New Patient Assessment Form" (Appendix 2).

NSPT was performed after the initial examination. Treatment would include oral hygiene instruction (OHI) and root surface debridement (RSD) using ultrasonic scalers &/or hand instruments. Extractions were sometimes required as part of initial therapy for hopeless prognosis teeth or for teeth that were irrational to treat (McGuire, 1996, Kwok and Caton, 2007)

After a follow up period of at least six weeks to allow healing and reattachment of long junctional epithelium (Listgarten, 1979) the data recorded at baseline, including a full six-point pocket chart, was reassessed to evaluate the response to treatment using the "Periodontal Reassessment Proforma" (Appendix 3). Only the first reassessment after the first round of NSPT was included in this study. Subsequent six-point pocket charts were not being assessed. However, data was recorded on how many additional rounds of NSPT were undertaken prior to any periodontal surgery or periodontal stability achieved.

3.4 Data extraction

Patient level, tooth level and site level parameters extracted from patient's records for analysis are shown (see Figure 1 & Appendix 8).

Patient level	Tooth level
Age	Tooth mobility
Gender	Tooth type
Ethnicity	Presence of apical pathology
Country of Birth	Endodontic periodontal lesion present clinically/ radiographically
London Borough	Number extracted during initial NSPT
Relevant medical history	Retention of hopeless teeth
Family history of	Periodontal abscesses
Smoking status	
Dental attendance	
Oral hygiene	
Previous periodontal therapy	
Distribution of periodontitis	
Site stability	
Number of missing teeth (before and after treatment)	
Site level	**Operator level**
Probing pocket depth (PPD)	Grade of operator
Bleeding on probing (BOP)	Instruments used
Horizontal Furcation involvement in molars (Grade I, II, III))	Time spent during NSPT

Figure 2 Patient- level, Tooth-level, Site Level, Operator-level factors that may affect the outcome of NSPT

Data was collected from the clinical records, using the following online databases:

1. Medical and social history (Appendix 4)
2. Dental/periodontal history and related factors (Appendix 5)
3. Non-surgical periodontal treatment and related factors (Appendix 6)
4. Diagnosis (Appendix 7)

Clinical periodontal information for each patient, was also collected in an excel spreadsheet (Appendix 9) with three tabs: 'Baseline', 'Reassessment NS1' and the 'Difference B vs NS1' which was automatically calculated. These spreadsheets enabled the recording of missing teeth, pocket depths, bleeding on probing, recession, plaque at six sites per tooth, tooth mobility (Miller, 1950), and furcation involvement (Hamp et al., 1975).

The surveys and spreadsheets were first piloted on a group of six patients to validate their use and data availability. This also allowed us to ensure that the data was being collected and uploaded properly, i.e. that there was no technical issues with the surveys.

Data collection was undertaken by two people as there was crossover with a corresponding study assessing baseline information to determine risk factors.

Initial data collection was based on a list of patients that was generated showing who had attended a consultant-led new patient clinic in the time period above. Each patient was allocated a study ID to allow the logging of which information had been recorded for each patient. No patient identifiable information was kept on this database. This database also identified the ethnic group, postcode and borough of residence for each patient.

3.5 Limitations of data collection

When undertaking the data collection, some issues were encountered:

- Poor record keeping such that the first periodontal reassessment could not be found either on the periodontal assessment proforma or on any separate six-point pocket chart sheet.
- Sometimes multiple reassessments had been completed and were not always dated so it was not clear which was for the first reassessment. In these cases, the case was excluded.
- Some data was missing from the clinical records or not recorded properly, e.g., plaque scores, recession, furcation involvement. Occasionally a single value was recorded for recession rather than three values for the buccal or lingual aspects of each tooth. Other times furcations were only recorded once on the palatal aspect of maxillary teeth and it was not clear if this related to the disto-palatal or mesio-palatal furcations or both.
- It was not possible to ascertain the experience of the clinicians in terms of years since qualification.
- Oral hygiene instruction was sometimes documented as being undertaken in an appointment, but what was specifically done was not always recorded, or how toothbrushing or interdental aids were instructed was not detailed sufficiently.

3.6 Data Analysis

3.6.1 Statistical analysis

- The data collected was entered into a computer and analysed using the statistical software package (SPSS).
- Once all data from the 108 patient records was extracted, data outlining characteristics of the study population was extracted, including the levels of severity of disease at baseline, baseline mean PPD, baseline number and percentage of stable sites, baseline number and percentage of unstable sites (PPD >4 mm with BOP/PPD>4 mm) and baseline percentage of sites with BOP.
- A sister study on the same individuals also examined what risk factors may be more common in the South-East Asian ethnic group compared to all other groups combined. A one-tailed test was used throughout for all chi-squared tests performed as we are testing in the alternative hypothesis whether South East Asian (SEA) ethnic groups have higher proportions compared to all other ethnic groups (presumption was that SEA population are more likely to have higher levels of the risk factor in question. Where two tailed tests are used the direction in the null hypothesis was not assumed (e.g., sex – no established sex differences in NS treatment response.
- For comparing the difference in the healing response between South East Asian (SEA) Ethnic groups and other ethnic groups at the subject level, the primary outcome measure was change in mean PPD.
- In order to do compare mean PPD changes, these changes were ranked from best (highest PPD reduction) to worst (lowest PPD reduction) in terms of mean PPD change. The 108 study members were then divided into two groups, with the top 50% with the highest PPD reduction allocated to the “High responders” group and those 50% with the worst response allocated as “low-responders”
- The significance level was set at p=0.05 within groups or between groups. Variables were dichotomised to assess differences between groups and tested by the Chi Squared test.
- Differences between pre- and post-treatment outcomes will be tested by bivariate analysis to identify potential factors involved in site/teeth/patient response to NSPT (positive vs non or negative response)
- Further analysis utilising PPD as a continuous variable was also undertaken to assess if NSPT was effective overall. In order to use this data we had to assess if it was normally distributed or not. This was undertaken with Shapiro Wilk analysis. Based on this, paired T test or non-parametric alternative (Wilcoxon Sign- rank test) could be used to compared PPD change to baseline PPD, and PPD change to baseline plaque and plaque change.
- Furthermore, ANOVA or non-parametric alternative (Kruskal Wallis test) were used to compare mean PPD change amongst ethnic groups as well as between SEA and non SEA groups.
- Demographic and health characteristics (Risk Factors) that were explored with bivariate analyses with regards to mean PPD change after NSPT included:
    - Comparison between SEA group (n=29 [27%]) and non-SEA group (n=79 [73%])
    - Comparison between smoking exposure group (ex and current smokers) group (n=41 [38%]) and Never smokers’ group (n= 67 [62%])

- Comparison between current smoking group group (n=9 [8%]) and Never/ex smokers' group (n= 99 [92%])
- Comparison between Diabetes group (n=16 [15%]) and non- diabetes group (n=92 [85%])
- Comparison between group with Baseline (B) High plaque at baseline PS score >50% (n= 77 [71%]) vs Baseline (B) Plaque score <50% (n= 31 [29%])
- Comparison between group with High Plaque at Reassessment (NS1) PS >50% (n= 21 [19%]) vs Reassessment (NS1) PS <50% (n= 87 [81%])
- Comparison between group with Reassessment (NS1) Plaque score >20% (n= 68 [63%]) vs group showing compliance with OH, i.e. Reassessment (NS1) Plaque score <20% (n= 40 [37%])
- Comparison between group demonstrating Plaque score improvement (>10%) (n= 85 [79%]) vs group not demonstrating PS improvement (n= 23 [21%])
- Comparison between high stress group (scored 6-10 on stress scale) (n=23 [21%]) and low stress group (scored 1-5 on stress scale) (n= 85 [79%])
- Comparison between Group with family history of Periodontitis (n=20 [19%]) and those without Family history of periodontitis (n=88 [81%])
- Comparison between males (n=48 [44%]) and females (n=60 [56%])
- Comparison between Regular attenders (n=86 [80%]) and Irregular attenders (n=22 [20%])
- Comparison between group undergoing 1 or treatment sessions (n=26 [24%]) vs 3 or more (n=82 [76%])
- Comparison between group using electric toothbrush (n=77 [71%]) and manual brush (n=31 [29%])
- Comparing time between treatment and Reassessment at 90d(3m) vs. 120d(4m)
- Comparing top 50% of individuals with highest baseline disease severity (Mean PPD) (n=54 [50%]) vs 50 % of individuals lowest baseline disease severity (n=54 [50%])

- To compare between ethnicities and SE group and others, Secondary outcome measures included BOP% and pocket closure (% of unstable sites at baseline that at reassessment have PPD>4 mm)
- Other operator factors were not included as operator experience could not always be determined, and most treatments were performed by PG students using combination of hand and power instrumentation. Experience of operators could not be determined from notes.

3.6.2 Multilevel analysis

Multivariable analysis (multivariate linear regression) was used to determine the relative importance of five most significant patient-level independent predictor variables as identified in the bivariate analysis using mean PPD reduction as the dependent outcome variable.

The Five Predictor (Independent variables) used all had dichotomous outcomes:

- Baseline Disease Severity
    - 50% of population with highest mean PPD (Most current disease)
    - 50% of the population with lowest mean PPD (Least current disease)
- Reassessment period longer than 4 months
    - Reassessment visit >4 months
    - Reassessment visit <4 months
- At least a 10% improvement in plaque score
    - Plaque score <10% improvement at reassessment relative to baseline
    - Plaque score >10% improvement at reassessment relative to baseline
- Poor plaque score at baseline (>50%)
    - Plaque score >50% at baseline
    - Plaque score <50% at baseline
- Sex
    - Male
    - Female

CHAPTER 4: RESULTS

4.1 Defining the cohort population

Data was successfully collected for 108 patients. Several observations can be made

N=108 (58 Male, 92 Female)

Age 47.3 years +/- 12.9 (SD)

Age (Years)	n (%)
0-19	4 (3.7%)
20-29	6(5.6%)
30-39	13(12.0%)
40-49	20(18.5%)
50-59	38(35.2%)
60-69	22(20.4%)
70-79	4(3.7%)
80-89	1(0.9%)

Table 1: Age distribution of sample collected so far (n=108)

	N	Baseline (B) Mean PPD (mm)	B mean no. stable sites	B mean no. unstable sites	Mean B no. of sites BOP	Mean B no. of sites with Plaque
All Patients	108	3.73	95.85	59.58	38.51	58.49
	Mean age (50.14 years old; range 18-81)		50.63%	31.48%	20.34%	30.90%

Table 2: All Patients Baseline mean data: mean baseline (B) PPD (mm), mean number and % sites stable, unstable, BOP and with plaque at baseline. Average age of study population 50.14 years old (range 18-81)

Gender	N	%	Baseline (B) Mean PPD (mm)	B mean no. stable sites	B mean no. unstable sites	Mean B no. of sites BOP	Mean B no. of sites with Plaque
Male	48	44.44%	3.74	95.48	59.58	38.76	58.66
				50.43%	31.48%	20.48%	30.99%
Female	60	55.56%	3.73	95.85	59.69	38.54	58.49
				50.63%	31.53%	20.36%	30.90%

Table 3: Male and Female. Baseline mean data: mean baseline (B) PPD (mm), mean number and % sites stable, unstable, BOP and with plaque at baseline.

	Number of erupted teeth	n (%)
No periodontal tooth loss (n=, %)	32	8 (7.4%)
	31	5 (4.6%)
	30	10 (9.3%)
	29	10 (9.3%)
	28	22 (20.4%)
<5 missing teeth (n=, %)	27	7 (6.5%)
	26	16 (14.8%)
	25	6 (5.6%)
	24	6 (5.6%)
	23	4 (3.7%)
>5 missing teeth (n=, %)	22	5 (4.6%)
	21	2 (1.9%)
	20	2 (1.9%)
	19	1 (0/9%)
	18	1 (0.9%)
	17	1 (0.9%)
	16	1 (0.9%)
	15	0 (%)
	14	1 (0.9%)
		108 (100%)

Table 4: Number of erupted teeth

Table 1 shows the age distribution of the cohort (n=108); most of the cohort were ages between 30-69 (n=93), representing 86% of the total cohort. The largest group was aged between 50-59 (n= 38) representing 35% of the cohort. The mean age was 50.14 years old, with subject ages ranging from 18-81 years old.

Table 2 shows that the mean baseline PPD for the entire cohort was 3.73 mm, whilst the mean percentage of stable sites and unstable sites at baseline was 50.63% and 31.48% respectively. Furthermore 20.34% of baseline sites presented with BOP whilst 30.9% presented with plaque. Table 3 shows that males represented 44.44% of the sample whilst females represented 55.56%, whilst barely any difference in baseline mean PPD, % of stable and unstable sites, % BOP and % sites with plaque.

Table 4 shows that the majority (n=55, 51%) of patients had not lost teeth due to periodontal tooth loss and had at least >28 erupted teeth, although 39 (36%) of the cohort had lost < 5 teeth. Very few individuals relative to the overall cohort had lost more than 5 teeth (n=14, 9%). This tallies with table 5 which shows that more individuals were diagnosed as stage III (77%) rather than stage IV (20%) periodontitis based on the EFP classification, which distinguishes between grade III and IV based on whether ≥4 teeth were lost due to periodontitis. Interestingly when assessing the staging based on the BSP classification where radiographic bone loss is used to determine the stage, most cases are stage IV (59%) rather than stage III (5%). The extent of disease for most individuals was generalised where more than 20% of sites are affected whilst for both BSP and EFP classifications, most

patients were documented as grade C, which identifies the rate of progression. Table 6 shows that mean PPD at baseline ranged between 2 mm and 5 mm.

Prevalence of Periodontitis (n=108)	**N**	**%**
Generalised Periodontitis (>30 sites affected)	85	79%
Localised Periodontitis (<30 sites affected)	22	20%
Molar- Incisor	1	1%
EFP Staging	**N**	**%**
Stage I	0	0%
Stage II	3	3%
Stage III	83	77%
Stage IV	22	20%
BSP Staging	**N**	**%**
Stage I	0	0%
Stage II	1	1%
Stage III	5	5%
Stage IV	64	59%
Not stated	38	35%
EFP Grading	**N**	**%**
Grade A	3	3%
Grade B	27	25%
Grade C	78	72%
BSP Staging	**N**	**%**
Grade A	0	0%
Grade B	6	6%
Grade C	64	59%
Not stated	38	35%

Table 5: Extent, Staging, grading of Periodontitis in sample (n=108)

4.1.1 Periodontal pocket depth at baseline

Mean PPD (mm) = 3.73 +/-10

Mean PPD (mm)	**n**
0-1	**0 (0%)**
1-2	**0 (0%)**
2-3	**24 (22.2%)**
3-4	**51 (47.2%)**
4-5	**22 (20.4%)**
5-6	**8 (7.4%)**
6-7	**1 (0.9%)**
7-8	**2 1.9%)**

Table 6: Periodontal Pocket depth at baseline. Mean PPD (mm) frequency and percentage

4.1.2 Ethnic groups

Ethnicity	N	%	Baseline (B) Mean PPD (mm)	B mean no. stable sites	B mean no. unstable sites	Mean B no. of sites BOP	Mean B no. of sites with Plaque
White - English, Welsh, Scottish, Northern Irish or British (N / %)	44	40.74%	3.73	96.09	58.88	38.55	58.26
				50.76%	31.10%	20.37%	30.77%
White- any other white (N / %)	6	5.56%	3.66	96.26	56.09	37.70	58.82
				50.85%	29.63%	19.92%	31.07%
Asian- Bangladeshi, Indian or Pakistani (N / %)	29	26.85%	3.70	97.09	57.05	38.77	59.26
				51.29%	30.14%	20.48%	31.31%
Asian- Any other Asian background (N / %)	9	8.33%	3.73	96.24	59.19	38.36	58.68
				50.84%	31.27%	20.26%	31.00%
Black African or Caribbean (N / %)	15	13.89%	3.71	96.92	57.29	38.67	59.08
				51.20%	30.26%	20.43%	31.21%
Any Other Black, African or Caribbean backgrounds (N / %)	2	1.85%	3.35	105.20	46.68	36.25	60.39
				55.57%	24.66%	19.15%	31.90%
Mixed- (N / %)	2	1.85%	3.17	119.50	30.50	35.63	51.92
				63.12%	16.11%	18.82%	27.42%
Other (N / %)	1	0.93%	3.21	131.00	37.00	6.32	51.15
				69.20%	19.54%	3.34%	27.02%
Not stated (N / %)	0	0	0	0	0	0	0
				0.00%	0.00%	0.00%	0.00%

Table 7: Ethnicities of sample group (n=108). Baseline mean data: mean baseline (B) PPD (mm), mean number and % sites stable, unstable, BOP and with plaque at baseline.

Ethnic Group		n (%)
White (n=67, 44.7%)	White British/Irish	44 (40.7%)
	White Other	6 (5.6%)
Asian or Asian British (n=52, 34.7%)	Indian	10 (9.3%)
	Pakistani	6 (5.6%)
	Bangladeshi	13 (12.0%)
	Chinese	2 (1.9%)
	Any other Asian background	7 (6.5%)
Black, Black British, Caribbean, African (n=30, 20%)	Black African	11 (10.2%)
	Black Caribbean	4 (3.7%)
	Any other Black, Black British or Caribbean background	2 (1.9%)
Mixed	Mixed	2 (1.9%)
Other ethnic group (n=1, 0.7%)	Arab	0 (0.0 %)
	Any other ethnic group	1 (0.9%)
	Total	**108 (100%)**

Table 8: Combined ethnic groups 1; frequency and percentage

Combined Ethnic groups	n
White	50 (46.3%)
Black	17 (15.7%)
South-East Asian	29 (26.9%)
Other	12 (11.1%)
Total	**108 (100%)**

Table 9: Combined ethnic groups 2; frequency and percentage

Combined Ethnic groups	n
South-East Asian	29 (26.9%)
Other	79 (73.1%)
Total	**108 (100%)**

Table 10: Combined ethnic groups 3; frequency and percentage of South East Asian group and others

Tables 7-10 show the number and percentage proportions for the different ethnic groups. South East Asians represent 26.9% of the cohort population (n=29), which includes patients of Indian, Pakistani and Bangladeshi ethnicity. This compares to the “other” group, which makes up 73.1% of the cohort (n=79), within which white British/Irish was the largest group (n=44, 40.7%). Table 7 also shows that baseline (B) mean data for all ethnicities including mean PPD (mm), mean number/percentage of stable sites, unstable sites, sites with BOP and with plaque.

4.2 Risk Factors

4.2.1 Baseline Plaque control

Mean baseline FMPS was 58.5% +/- 17.48 (SD). At baseline only 1/108 (0.9%) patient was considered compliant in relation to oral hygiene (i.e., <20% plaque score). 107/108 (99.1%) patients were considered non-compliant (i.e., >20% plaque scores).

	% Plaque score	n
Compliant (Plaque score <20%) N=4	0-9	0 (0.0%)
	10-19	1(0.9%)
Non-compliant (Poor) – 20-49% n=41	20-29	3 (2.8%)
	30-39	10 (9.3%)
	40-49	17 (15.7%)
Non-compliant Very poor (>50%) N=105	50-59	31 (28.7%)
	60-69	22 (20.4%)
	70-79	8 (7.4%)
	80-89	8 (7.4%)
	90-100	8 (7.4%)
	Total	108 (100%)

Table 11: Plaque scores and compliance. Baseline complaint patients with FMPS <20%, non-complaint (poor FMPS 20-49%) and non-compliant very poor (FMPS >50%)

4.2.2 Smoking

	n
Current smoker	**9 (8.3%)**
Ex-smoker	**32 (29.6%)**
Never smoker	**67 (62.0%)**
Total	**108 (100%)**

Table 12: Smoking status current smoker, ex-smokers and never smokers

	n
Smoking exposure	**41 (38.0%)**
Never smoker	**67 (62.0%)**
Total	**108 (100%)**

Table 13: Smoking status smoking exposure (current smoker and ex-smokers) vs never smokers

Tables 12 and 13 show the proportion of current smokers to ex and never smokers, and the number of those exposed to smoking to never smokers, respectively. There were only 9 smokers in this cohort (8.3%), however 41 patients (38%) were exposed to smoking.

4.2.3 Diabetes

	n (%)	Diabetes type	n (%)
Diabetes	**16(%)**	**Type 1**	**0 (0.0 %)**
		Type 2	**16 (14.8 %)**
No Diabetes	**92 (%)**		**92 (85.2 %)**
Total			**108 (100%)**

Table 14: Diabetes status of cohort; diabetes (type 1 and 2) vs non diabetes

4.2.4 Stress (self-declared stress levels 0-10)

	n (%)
Low stress (0-5)	**85 (78.7%)**
High Stress (6-10)	**23 (21.3%)**
Total	**108 (100%)**

Table 15: Self-reported stress levels: Diabetes status of cohort; diabetes (type 1 and 2) vs non diabetes

4.2.5 Irregular dental attendance (Self-declared)

	n(%)
Irregular attender	**21 (19.4%)**
Regular attender	**87 (80.6%)**
Total	**108 (100%)**

Table 16: Dental attendance

Table 14 shows that only a minority (n=16, 14.8%) of patients had diabetes and all of these were type 2. When grouped into low stress and high stress groups based on self-reported levels in documented notes, table 15 shows that most patients had low stress, i.e., between 0 and 5 (n=85, 78.7%). Table 16 shows that the prevalence of regular attenders was significantly higher than irregular attenders, however these were also based on self-declarations.

4.3 NSPT treatment response as an outcome Variables

Primary outcome to assess treatment response was the change in mean probing pocket depth. In order to do this the mean PPD change between baseline and at reassessment were determined and then ranked from best (highest PPD reduction) to worst (lowest PPD reduction) in terms of PPD change. The 108 study members were then divided into two with the top 50% with the highest PPD reduction allocated to the "High responders" group and those 50% with the worse response allocated as "low-responders"

4.3.1 Ethnic Group

		NS treatment response		
		High responders	**Low responders**	**Total**
Ethnic group	**South-East Asian**	16	13	**29**
	All Others	38	41	**79**
		54	**54**	**108**

Table 17: NSPT treatment response; high vs low responders in SEA group and others group

Chi-squared = 0.4243, z= 0.6514, p=0.2574 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.147 (0.7408-1.663), Odds Ratio = 1.328 (0.5655-3.063)

There was no significant difference in NS treatment response in the SEA population compared to other groups.

4.3.2 Smoking exposure (Ex or current smoker)

		NS treatment response		
		High responders	**Low responders**	**Total**
Smoking Exposure	**Yes**	22	19	**41**
	No	32	35	**67**
		54	**54**	**108**

Table 18: NSPT treatment response; high vs low responders comparing smoking exposure groups

Chi-squared = 0.3538, z= 0.5948, p=0.2760 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.123 (0.7562-1.627), Odds Ratio = 1.266 (0.5761-2.843)

Smoking history at baseline does NOT significantly affect initial NS treatment response

4.3.3 Current Smoker

		NS treatment response		
		High responders	**Low responders**	**Total**
Current Smoker	**Yes**	4	5	**9**
	No	50	49	**99**
		54	**54**	**108**

Table 19: NSPT treatment response; high vs low responders comparing current smokers and non/ex smokers

Chi-squared = 0.1212, z= 0.3482, p=0.3639 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.136 (0.6496-2.724), Odds Ratio = 1.276 (0.3509-4.346)

Being a self-reported current smoker at baseline did NOT significantly affect initial NS treatment response (P=0.3639).

4.3.4 Diabetes

		NS treatment response		
		High responders	**Low responders**	**Total**
Diabetes	**Yes**	7	9	**16**
	No	47	45	**92**
		54	**54**	**108**

Table 20: NSPT treatment response; high vs low responders comparing Diabetics and non-diabetics

Chi-squared = 0.2935, z= 0.5417, p=0.2940 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.150 (0.6562-1.718), Odds Ratio = 1.343 (0.4682-3.554)

A diabetes diagnosis at baseline does NOT significantly affect initial NS treatment response
NB There was no indication of diabetic control in these results.

4.3.5 High Plaque at baseline (>50%)

		NS treatment response		
		High responders	**Low responders**	**Total**
High Plaque (>50%)	**Yes**	44	33	**77**
	No	10	21	**31**
		54	**54**	**108**

Table 21: NSPT treatment response; high vs low responders comparing high (>50%) and low PS (<50%) at baseline

Chi-squared = 5.475, z= 2.340, p=0.0096** (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.581 (1.083-2.235), Odds Ratio = 2.8 (1.197-6.905)

Better treatment outcomes (Mean PPD reduction) were found with patients with higher levels of plaque (>50%) at baseline

4.3.6 High Plaque at Reassessment (>50%)

		NS treatment response		
		High responders	**Low responders**	**Total**
High Plaque (>50%)	**Yes**	9	12	**21**
	No	45	42	**87**
		54	**54**	**108**

Table 22: NSPT treatment response; high vs low responders comparing high (>50%) and low PS (<50%) at reassessment

Chi-squared = 0.5320, z= 0.7294, p=0.2329 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.207 (0.7619-2.179), Odds Ratio = 1.429 (0.5484-3.538)

No association of reassessment plaque levels and NS treatment outcome

4.3.7 Compliance with OH (PS<20%) at reassessment

		NS treatment response		
		High responders	**Low responders**	**Total**
Compliant plaque levels at reassessment	**Yes**	15	25	**40**
	No	39	29	**68**
		54	**54**	**108**

Table 23: NSPT treatment response; high vs low responders comparing compliant (<20%) and non-compliant PS (>20%) at reassessment

Chi-squared = 3.971, z= 1.993, p=0.0232* (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.529 (1.007-2.461), Odds Ratio = 2.241 (1.000-4.941)

At reassessment only 40/108 (37.0%) of patients were compliant in terms of plaque score <20%. Interestingly compliant patients had poorer non-surgical responses compared to compliant patients.

4.3.8 Plaque score improvement (10%)

		NS treatment response		
		High responders	**Low responders**	**Total**
Plaque score improvement of at least 10%	**Yes**	46	39	**85**
	No	8	15	**23**
		54	**54**	**108**

Table 24: NSPT treatment response; high vs low responders comparing subjects with good (>10%) and poor PS improvement

Chi-squared = 2.707, z= 1.645, p=0.05* (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.421 (0.9277-2.011), Odds Ratio = 2.212 (0.8959-5.773)

Patients achieving at least a 10% improvement in plaque between baseline and reassessment showed better non-surgical treatment outcomes (P-0.05*)

4.3.9 High Stress (Self-declared)

		NS treatment response		
		High responders	**Low responders**	**High responders**
High Stress	**Yes**	10	13	**23**
	No	44	41	**85**
		54	**54**	**108**

Table 25: NSPT treatment response; high vs low responders comparing high stress and low stress subjects

Chi-squared = 0.4972, z= 0.7051, p=0.2404 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.191 (0.7630-2.087), Odds Ratio = 1.395 (0.5726-3.706)

There was no difference in NS treatment outcomes between those patients with self-declared high stress (rated 6-10) compared to those declaring low stress (rated 0-5). All patients were asked to self-declare stress on a scale of 0-10 at baseline before treatment

4.3.10 Family History of Periodontitis

		NS treatment response		
		High responders	**Low responders**	**High responders**
Family History of Periodontitis	**Yes**	12	8	**20**
	No	42	46	**88**
		54	**54**	**108**

Table 26: NSPT treatment response; high vs low responders comparing those with and without family history of periodontitis

Chi-squared = 0.9818, z= 0.9909, p=0.1609 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.257 (0.7764-1.819), Odds Ratio = 1.643 (0.6081-4.476)

There was no difference in NS treatment outcomes between those patients with a self-reported positive family history of periodontitis at baseline compared to those without

4.3.11 Gender

		NS treatment response		
		High responders	**Low responders**	**High responders**
Gender	**Male**	29	19	**48**
	Female	25	35	**60**
		54	**54**	**108**

Table 27: NSPT treatment response; high vs low responders comparing males and females

Chi-squared = 3.750, z= 1.936, p=0.0528 (ns) (2-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.450 (0.9955-2.130) Odds Ratio = 2.137 (1.000-4.494)

There were more favourable NS treatment responses observed in males compared to female patients – borderline statistical significance

4.3.12 Attendance

		NS treatment response		
		High responders	**Low responders**	**High responders**
Attendance	**Regular**	42	45	**87**
	Irregular	12	9	**21**
		54	**54**	**108**

Chi-squared = 0.5320, z= 0.7294, p=0.23329 (ns) (1-tailed test)

Table 28: NSPT treatment response; high vs low responders comparing regular and irregular attenders

Effect size and 95% CIs shown:

Relative Risk = 1.184 (0.7262-1.729), Odds Ratio = 1.429 (0.5484-3.538)

There was no difference in treatment response between regular and irregular attendees (self-reported at baseline)

4.3.13 Number of treatment sessions (1 or 2 vs 3 or more)

		NS treatment response		
		High responders	**Low responders**	**High responders**
Number of NS visits	**1 o 2**	42	40	**82**
	3 or more	12	14	**26**
		54	**54**	**108**

Table 29: NSPT treatment response; high vs low responders comparing those who had 1-2 sessions with those who had >2 sessions as part of initial NSPT

Chi-square = 0.2026, z= 0.4501, p=0.3263 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.110 (0.7319-1.850), Odds Ratio = 1.225 (0.4848-2.863)

There was no difference in NS treatment response in comparing patients receiving 2 or less visits compared to those having 3 or more visits.

4.3.14 Electric vs. Manual Toothbrush

		NS treatment response		
		High responders	**Low responders**	**High responders**
Toothbrush	**Electric**	36	41	**77**
	Manual	18	13	**31**
		54	**54**	**108**

Table 30: NSPT treatment response; high vs low responders comparing subjects using an electric toothbrush versus those using a manual brush

Chi-square = 1.131, z=1.064, p=0.1438 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.242(0.8211-1.784), Odds Ratio = 1.577(0.6966-3.823))

There was no significant difference between electric toothbrush users compared to those using manual brushes in NS treatment response.

4.3.15 Time between treatment and Reassessment

Comparing 90 days (3 months) vs. 120 days (4 months)

Reassessment at 3 months

		NS treatment response		
		High responders	Low responders	High responders
Time to reassessment	**<90 days**	31	32	**63**
	>90 days	23	22	**45**
		54	**54**	**108**

Table 31: NSPT treatment response; high vs low responders comparing time to reassessment before and after three months (90 days)

Chi-square = 0.03810, z=0.1952, p=0.4226 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.039 (0.7132-1.551), Odds Ratio = 1.079(0.5022-2.332)

There was no significant difference in NS treatment response in comparing patients reassessed at <90 days (n=63) compared to those reassessed at >90 days (n=45)

Reassessment at 4 months

		NS treatment response		
		High responders	Low responders	High responders
Time to reassessment	**<120 days**	44	36	**80**
	>120 days	10	18	**28**
		54	**54**	**108**

Table 32: NSPT treatment response; high vs low responders comparing time to reassessment before and after four months (120 days)

Chi-square = 3.086, z=1.757, p=0.0395* (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.540 (0.9568-2.737), Odds Ratio = 2.2(0.9084-5.521)

There was a significant difference in NS treatment response in comparing patients reassessed at <120 days (n=80) compared to those reassessed at >120 days (n=28).

Waiting > 4 months to reassess was associated with poorer treatment outcomes.

4.3.16 Baseline disease severity (Mean PPD)

If we compare the baseline Mean PPD of the patients with the lowest 50% to the highest 50% at baseline in terms of whether they fall into high or low responders

		NS treatment response		
		High responders	**Low Responders**	**Total**
Baseline Mean PPD(mm)	**Lowest mean baseline PD 50%**	17	37	**54**
	Highest mean baseline PPD (50%)	37	17	**54**
		54	**54**	**108**

Table 33: NSPT treatment response; high vs low responders comparing subjects with the lowest 50% of mean baseline to the highest 50% of mean baseline PPD

Chi-squared =14.81, z=3.849, p=0.0001*** (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 2.176 (1.444-3.418), Odds Ratio = 4.737 (2.105-10.83)

Patients with the highest mean PPD at baseline responded better to NS therapy compared to those with lower mean PPD at baseline (p=0.0001***)

Patients with more disease at baseline (mean PPD) respond with greater amounts of PPD reduction compared to those with less disease.

4.4 PPD as continuous outcome variable

	N	Range	Minimum	Maximum	Mean	Std. Deviation
Baseline PPD	108	5.2	2.01	7.21	3.7312	0.9964
Reassessment (NS1) PPD	108	5.08	1.76	6.84	3.2072	0.76463
PPD change (B-NS1)	108	4.81	-0.98	3.83	0.5239	0.66347

Table 34: Range, minimum, maximum, mean, standard deviation for Baseline PPD, Reassessment PPD and change in PPD

This table shows the mean PPD at baseline was 3.73 mm (range 2.01-7.21 mm) whilst at NS1 this was 3.21mm (range 1.76-6.84 mm) and mean PPD change was 0.52 mm (-0.98 – 3.83 mm).

Further tests to establish if the PPD change, Baseline PPD and PPD at reassessment (NS1) data was normally distributed is shown below

Tests of Normality							
	Shapiro-Wilk			Skewness		Kurtosis	
	Statistic	df	Sig.	Statistic	Standard error	Statistic	Standard error
Basline PPD	0.936	108	<.001	1.084	0.233	1.762	0.46
Reassessment (NS1) PPD	0.911	108	<.001	1.376	0.233	3.841	0.46
PPD change (B-NS1)	0.915	108	<.001	1.411	0.233	4.947	0.46

Table 35: Assessment of normality for Baseline PPD, Reassessment PPD and change in dependent PPD variables

The Shapiro Wilk significance figures in all three cases demonstrates that the data was not normally distributed. This was because we assumed the null hypothesis for this test of normality that the data are normally distributed. As the P value was <0.05 we rejected the null hypothesis. Furthermore, when assessing the skewness and kurtosis of the data for PPD B, PPD NS and PPD change, the Z values (statistic/standard error) are not all within +/-

1.96. As the data was not normally distributed, we therefore opted to use non parametric tests for comparisons.

	Mean PPD change	N	Std. Deviation	Minimum	Maximum
Low Responder group	0.0489	54	0.27732	-0.98	0.39
High Responder group	1.0028	54	0.58373	0.46	3.83
Total	0.5258	108	0.66066	-0.98	3.83

Table 36: Mean PPD change, range of PPD change, standard deviation for low and high responder groups

The table above shows the mean PPD change in the responder group was 0.049 mm (range -0.98-0.39 mm) compared to the responder group which had a mean PPD reduction of 1.003 mm (range 0.46-3.83 mm). in the non-responding group 19 patients had no improvement (6) or increasing in PPD depths after NSPT (13). Overall PPD reduction for the whole group was 0.526 mm.

As the PPD change variables are non-parametric, Kruskal-Wallis tests which are utilised as a non-parametric alternative to ANOVA to compare changes over time in the means of two or more groups, to firstly compare all the ethnic groups separately and then compare the SEA group to the non-SEA group. Both tests showed no differences in PPD change between groups.

Null Hypothesis	Test	Sig.[a]	Decision
The distribution of PPD change is the same across all categories of Ethnicity.	Independent-Samples Kruskal-Wallis Test	0.538	Retain the null hypothesis
a. The significance level is .050.			
Null Hypothesis	**Test**	**Sig. [a]**	**Decision**
The distribution of PPD change is the same between SEA and non-SEA Ethnic groups	Independent-Samples Kruskal-Wallis Test	0.252	Retain the null hypothesis.
a The significance level is .050.			

Table 37: Hypothesis testing (Kruskal-Wallis Tests) comparing the distribution of mean PPD change between al ethnic groups and between the SEA group and non-SEA group

In order to assess the significance of B PPD on PPD change, a Wilcoxin Signed Rank test was undertaken, again as the data was non parametric and variables were continuous and paired. A similar test was carried out between Baseline Plaque and PPD change as well as plaque change and PPD change.

Baseline PPD vs PPD change Wilcoxin signed Rank test	
Z	-9.021[b]
Asymp. Sig. (2-tailed)	<.001
a. Wilcoxon Signed Ranks Test b. Based on negative ranks.	

Table 38: Wilcoxin signed Rank test of significance for Baseline PPD vs PPD change.

As $p<0.05$ ($p<0.001$) we may assume that the baseline PPD had a significant effect on PPD change. Similar effects on PPD change were seen for baseline plaque levels (FMPS-B) and FMPS change.

PPD changes vs Baseline plaque scores and plaque score changes PPD change Wilcoxin signed Rank test		
	Baseline FMPS vs. PPD change	FMPS change vs. PPD change
Z	-8.979[b]	-7.771[b]
Asymp. Sig. (2-tailed)	<.001	<.001
a. Wilcoxon Signed Ranks Test		
b. Based on negative ranks.		

Table 39: Wilcoxin signed Rank test of significance for Baseline FMPS vs PPD change and FMPS change vs PPD change.

Further investigations of secondary outcome variables in relation to assess differences between ethnic groups included change in plaque score changes, BOP and changes in the number of unstable sites (PPD >4 mm). For all these variables, there was no significant difference between ethnicities or between the SEA group and non-SEA group.

Hypothesis Test Summaries (Kruskal Wallis tests)				
	Null Hypothesis: The distribution of the variable is the same across categories of Ethnicity.		Null Hypothesis: The distribution of the variable is the same between SEA group and non SEA group	
Variable	Sig.[a,b]	Decision	Sig.[a,b]	Decision
Baseline BOP	0.248	Retain the null hypothesis.	0.197	Retain the null hypothesis.
Reassessment BOP	0.931	Retain the null hypothesis.	0.766	Retain the null hypothesis.
BOP change	0.075	Retain the null hypothesis.	0.141	Retain the null hypothesis.
Unstable sites (PPD>4 mm) at baseline	0.661	Retain the null hypothesis.	0.190	Retain the null hypothesis.
Unstable sites (PPD>4 mm) at Reassessment	0.843	Retain the null hypothesis.	0.897	Retain the null hypothesis.
Unstable sites change	0.166	Retain the null hypothesis.	0.316	Retain the null hypothesis.
Baseline plaque scores (FMPS)	0.228	Retain the null hypothesis.	0.146	Retain the null hypothesis.
Reassessment FMPS	0.361	Retain the null hypothesis.	0.903	Retain the null hypothesis.
FMPS change	0.236	Retain the null hypothesis.	0.226	Retain the null hypothesis.
a. The significance level is .050.				
b. Asymptotic significance is displayed.				

Table 40: Hypothesis testing (Kruskal-Wallis Tests) comparing the distribution of Baseline BOP, Reassesment BOP, OP change, unstable sites at baseline, unstable sites at reassessment, unstable sites change, FMPS at baseline reassessment and FMPS change

4.5 Multiple regression models

Periodontal Non-surgical treatment outcome

Model 1

Logistic Regression

Dependent Outcome variable = Mean PPD reduction

Dichotomous (binary) outcome:

- 50% largest PPD reduction (High Responders)
- 50% smallest PPD reduction (Low responders)

As we have 108 in the sample – 5 predictor variables will be entered into the model (approximately 1 variable for 20 observations) – chosen based on the univariate analysis

5 Predictor (Independent variables) – all with dichotomous outcomes:

- Baseline Disease Severity
  - 50% of population with highest mean PPD (Most current disease)
  - 50% of the population with lowest mean PPD (Least current disease)
- Reassessment period longer than 4 months
  - Reassessment visit >4 months
  - Reassessment visit <4 months
- At least a 10% improvement in plaque score
  - Plaque score <10% improvement at reassessment relative to baseline
  - Plaque score >10% improvement at reassessment relative to baseline
- Poor plaque score at baseline (>50%)
  - Plaque score >50% at baseline
  - Plaque score <50% at baseline
- Sex
  - Male
  - Female

Enter 5 predictor variables (Univariate p<=0.2) into the binary logistic regression model

Predictors							95% CI for EXP(B)	
Factor	B	S.E	Wald	df	Sig	Exp(B)	Lower	Upper
High baseline disease (Mean PPD)	1.361	0.447	9.267	1	0.002*	3.898	1.623	9.361
Reassessment period > 4 months	-0.841	0.502	2.809	1	0.094	0.431	0.161	1.153
<10% plaque improvement	0.665	0.551	1.454	1	0.228	1.944	0.660	5.725
Poor plaque control at baseline (>50%)	0.783	0.498	2.466	1	0.116	2.188	0.823	5.811
Sex	-0.520	0.445	1.368	1	0.242	0.594	0.248	1.422
Constant	--1.265	0.680	3.459	1	0.063	0.282		

Table 41: Multivariable analysis comparing effect of 5 Predictor factors (high baseline disease, reassessment after 4 months, <10% plaque improvement, poor plaque control at baseline, sex) with dichotomous outcomes on Mean PPD reduction after NSPT

*All variables entered into the equation

Model Summary

- Model chi-square = 24.030 p<0.001 – significant improvement compared to null model– good model
- Hosmer and Lemeshow Test chi-square 4.978 p=0.547 ns – indicates goodness of fit of model i.e. a non-significant p>0.05 result here shows a good model
- Nagelkerke R-square = 0.266 (model explains 26.6% of the variation in the non-surgical treatment response)
- Classification Accuracy of model - Model successfully predicts 74.1% of treatment response. The model predicts a poor treatment response and good treatment response outcome equally well (74.1%)
- Only baseline disease level (Mean PPD) was a significant predictor of treatment response
- B = regression weights for model – not really intuitive to understand
- SE = Standard errors
- Wald = Ratio of regression weight (B) to SE i.e. B/S.E
- Df = degrees of freedom
- Easier to understand Exp(B) = Odds Ratio – Change in odds for every unit change of predictor variable
    - Patients presenting with higher levels of disease (Highest 50% percentile) at baseline (Mean PPD) are 3.9 times more likely to have a good non-surgical treatment response (more pocket depth reduction) compared to those with lower levels of disease (Lowest 50% percentile)

- All other variables were non-significant in this model (many factors significant in the univariate analysis became non-significant when combined into the same binary logistic regression model.

4.6 Risk Factors research question: Are there certain established periodontitis risk factors more common in the South-East Asian ethnic group compared to all other groups combined?

A one-tailed test was used throughout for all chi-squared tests performed as we are testing in the alternative hypothesis whether SEA ethnic groups have higher proportions compared to all other ethnic groups (presumption was that SEA population are more likely to have higher levels of the risk factor in question. Where two tailed tests are used the direction in the null hypothesis was not assumed (e.g., sex – no established sex differences in NS treatment response.

4.6.1 Diabetes

Diabetes in this analysis was a diagnosis of either Type 1 or Type 2 diabetes (No reference was made to amount of control).

Research Question: Is diabetes prevalence higher in SEA ethnic groups compared to other ethnic groups?

		Diabetes		
		Yes	**No**	**Total**
Ethnic group	**South-East Asian**	6	23	**29**
	All Others	10	69	**79**
		16	**92**	**108**

Table 42: Presence of diabetes in SEA group and other ethnic group

Chi-squared = ,1.084 z= 1.041, p=0.1489 ns (1-tailed test).

Effect size and 95% CIs:

Relative Risk = 1.634 (0.6555-3.887), Odds Ratio = 1.800 (0.5620-5.363)

Conclusion: Diabetes does not occur at significantly higher levels in the SEA ethnic group compared to all other groups combined (p=0.1489).

4.6.2 Age <40 years in cohort

Research Question: Within the periodontitis cohort referred for Specialist assessment, Is the proportion of patients <40 years greater in the SEA ethnic group i.e. Do a higher proportion of the SEA ethnic groups get referred at a younger age?

		Age <40 years		
		Yes	**No**	**Total**
Ethnic group	**South-East Asian**	9	20	**29**
	All Others	14	65	**79**
		85	**23**	**108**

Table 43: Proportion of older (>40 years old) to younger patients (<40 years old) in SEA group and others group

Chi-squared = 2.243, z= 1.498, p=0.0671 ns (1-tailed test).

Effect size and 95% CIs shown:

Relative Risk=1.751 (0.8417-3.482), Odds Ratio=2.089 (0.8169-5.521)

A larger proportion of the SEA ethnic group get referred at a younger age (<40 years) compared to other ethnic groups i.e. There are more younger SEA (<40 years) in the treated cohort.

This narrowly misses the 5% significance level (p=0.0671).

4.6.3 Smoking exposure (Ex or current smoker)

Research question: Is smoking exposure higher in the SEA ethnic group compared to all other groups combined?

		Smoking exposure		
		Yes	**No**	**Total**
Ethnic group	**South-East Asian**	10	19	**29**
	All Others	31	48	**79**
		41	**67**	**108**

Table 44: Smoking exposure in SEA group and others group

Chi-squared = 0.2039, z= 0.4515, p=0.3258 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 0.8788 (0.4805), Odds Ratio = 0.8149 (0.3250-1.970)

There was no significant difference in the proportion of patients with a smoking history in the SEA population compared to all other ethnic groups combined.

4.6.4 Gender

Research question: Are there higher proportions of a particular gender in SEA ethnic groups compared to all other ethnic groups combined?

		Gender		
		M	**F**	**Total**
Ethnic group	**South-East Asian**	14	15	**29**
	All Others	34	45	**79**
		48	**60**	**108**

Table 45: Gender proportions in SEA group and others group

Chi-squared = 0.2357, z= 0.4855, p=0.6273 (2-tailed test – used as no presumed direction of effect)

Effect size and 95% CIs shown:

Relative Risk = 1.122(0.6878-1.710), Odds Ratio = 1.235 (0.5117-2.943)

There was no significant difference in proportion of presenting Male/females in the SEA ethnic group compared to all other groups combined (p=0.6273)

4.6.5 Plaque (Poor plaque control >50%)

Research question: Are there higher proportions of patients with poorer plaque control in SEA ethnic groups compared to all other ethnic groups combined?

		Poor plaque control >50%)		
		Y	**N**	**Total**
Ethnic group	**South-East Asian**	21	8	**29**
	All Others	56	23	**79**
		77	**31**	**108**

Table 46: Proportion of subjects with poor plaque control (>50%) in SEA group and others group

Chi-squared = 0.02419, z= 0.3546, p=0.4382 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.022 (0.7470-1.298), Odds Ratio = 1.078 (0.4074-2.785)

There was no significant difference in the proportion of patients with baseline poor plaque control (>50% score) in the SEA ethnic group compared to all other groups combined (p=0.3614). Plaque levels are not significantly different in SEA vs other groups.

4.6.6 Family History of periodontal disease (self-declared)

Research question: Are there higher proportions of patients with a FH of periodontitis in SEA ethnic groups compared to all other ethnic groups combined?

		FH of periodontal disease		
		Y	**N**	**Total**
Ethnic group	**South-East Asian**	6	23	**29**
	All Others	14	65	**79**
		20	**88**	**108**

Table 47: Family History of periodontal disease (self-declared) proportions in SEA group and others group

Chi-squared = 0.1239, z= 0.3519, p=0.3624 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 1.167 (0.4930-2.599), Odds Ratio = 1.211 (0.4061-3.587)

There was no significant difference in the proportion of patients with a known family history of periodontal disease in the SEA ethnic group compared to all other groups combined (p=0.2769).

4.6.7 Interdental brush use

Research question: Are there higher proportions of patients using interdental brushes in SEA ethnic groups compared to all other ethnic groups combined?

		Interdental brush use		
		Y	**N**	**Total**
Ethnic group	**South-East Asian**	13	16	**29**
	All Others	38	41	**79**
		51	**57**	**108**

Table 48: Interdental brush use proportions in SEA group and others group

Chi-squared = 0.09122, z= 0.3020, p=0.3813 (ns) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 0.9313 (0.5641-1.424), Odds Ratio = 0.8766 (0.3765-2.060)

There was no significant difference in the proportion of patients using interdental brushes in the SEA ethnic group compared to all other groups combined (p=0.3813).

4.6.8 Irregular dental attender (Self-declared)

Research question: Are there higher proportions of irregular dental attenders in the SEA ethnic groups compared to all other ethnic groups combined?

		Irregular attender		
		M	**F**	**Total**
Ethnic group	**South-East Asian**	12	17	**29**
	All Others	9	70	**79**
		21	**87**	**108**

Table 49: Self-declared Irregular attendance proportions in SEA group and others group

Chi-squared = 12.18, z= 3.490, p=0.0002 (***) (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 3.632 (1.722-7.567), Odds Ratio = 5.490 (1.911-15.03)

There was a significantly higher levels of irregular dental attendance in the SEA ethnic group compared to all other groups combined (p=0.0002).

4.6.9 Stress (Self-reported on scale 0-10)

Research question: Are there higher amounts of self-reported stress in the SEA ethnic groups compared to all other ethnic groups combined at baseline?

		Stress Levels		
		High (6-10)	**Low (0-5)**	**Total**
Ethnic group	**South-East Asian**	3	26	**29**
	All Others	20	59	**79**
		23	**85**	**108**

Table 50: Self-reported stress level proportions in SEA group and others group

Chi-squared = 2.837, z= 1.684, p=0.0461** (1-tailed test)

Effect size and 95% CIs shown:

Relative Risk = 2.447 (0.8815-7.453), Odds Ratio = 2.938 (0.8087-9.948)

There are significantly lower levels of self-reported stress in the SEA ethnic group compared to all other groups combined (p=0.0461**).

4.7 Results Summary

4.7.1 Risk Factors in South East Asian (SEA) community

- A larger proportion of the SEA ethnic group get referred at a younger age (<40 years) compared to other ethnic groups i.e. There are more younger SEA (<40 years) in the treated cohort compared to the other ethnic groups. This narrowly misses the 5% significance level (p=0.0671).
- There was a significantly higher level of irregular dental attendance in the SEA ethnic group compared to all other groups combined (p=0.0002)
- There are significantly lower levels of self-reported stress in the SEA ethnic group compared to all other groups combined (p=0.0461**)
- All other risk factors showed no difference between SEA and other ethnic groups

4.7.2 Treatment Outcomes (Univariate analysis)

- Better treatment outcomes (Mean PPD reduction) were found with patients with higher levels of plaque (>50%) at baseline
- No association of reassessment plaque levels and NS treatment outcome
- At reassessment only 40/108 (37.0%) of patients were compliant in terms of plaque score <20%. Interestingly compliant patients had poorer non-surgical responses compared to compliant patients.
- Patients achieving at least a 10% improvement in plaque between baseline and reassessment showed better non-surgical treatment outcomes (P= 0.05*)
- There were more favourable NS treatment responses observed in males compared to female patients – borderline statistical significance
- There was a significant difference in NS treatment response in comparing patients reassessed at <120 days (n=80) compared to those reassessed at >120 days (n=28).
- Waiting > 4 months to reassess was associated with poorer treatment outcomes.
- Patients with the highest mean PPD at baseline responded better to NS therapy compared to those with lower mean PPD at baseline (p=0.0001***) i.e. Patients with more disease at baseline (mean PPD) respond with greater amounts of PPD reduction compared to those with less disease.

4.7.3 Binary logistic Regression model

- Baseline Disease severity was only significant predictor of good treatment response in the model which explained 26.6% of the variability in the dependent outcome variable

4.7.4 Multiple Level Logistic regression model

- Patients presenting with higher levels of disease (Highest 50% percentile) at baseline (Mean PPD) are 3.9 times more likely to have a good non-surgical treatment response (more pocket depth reduction) compared to those with lower levels of disease (Lowest 50% percentile)

- All other variables were non-significant in this model (many factors significant in the univariate analysis became non-significant when combined into the same binary logistic regression model.

CHAPTER 5: DISCUSSION AND FUTURE WORK

5.1 Discussion

Previous studies have generally demonstrated the effectiveness of NSPT (Van der Weijden and Timmerman, 2002, Trombelli et al., 2015, Suvan, 2005, Badersten et al., 1984, Suvan et al., 2020) and have also undertaken univariate and multilevel analyses of factors that may influence the outcome of NSPT (Tomasi et al., 2007, Wan et al., 2009, Jiao et al., 2017, D'Aiuto et al., 2005). Recent data from a large sample of a local East London population has suggested that periodontitis is more severe in minority ethnic groups, especially Pakistani, Indian and Bangladeshi groups who all had more PPD >4mm compared wo white British patients based on 1925 adults (Delgado-Angulo et al., 2016). Despite the potential for increased level of periodontial disease amongst such patients, their response to NSPT has not previously been investigated in the literature. As the Royal London Dental Hospital (RLDH) in Tower Hamlets is one of the most ethnically diverse boroughs in the UK, with 69% belonging to minority ethnic groups, of which 41% are Asian groups and 32% are Bangladeshi (Tower-Hamlets, 2018). This represents a population where we could investigate the impact of ethnicity on NSPT outcome, especially as it has been previously established that these groups are frequently under-represented in clinical outcome studies compared to Caucasian populations (Jiao et al., 2017). Although prognostic factors for NSPT outcome were previously assessed, ethnicity was not reported (Hughes et al., 2006b). Therefore, there was a need to better understand the factors influencing non-surgical success in our local East London population.

The effectiveness of NSPT on patients with chronic periodontitis was proved by this hospital-based retrospective study in patients at RLDH. The overall mean patient level PPD reduction was 0.52 mm (range: -0.98 - 3.83 mm). These PPD reductions are slightly lower than reported for previous studies. A Chinese retrospective study (Jiao et al., 2017) analysing 10,789 patients found PPD reductions of 0.62 mm and 0.65 mm at patient and site level respectively. Furthermore, another study also had higher weighted mean PD reduction of 0.64 mm although these were in pockets initially $\geq$ 5 mm (Van der Weijden & Timmerman 2002). A recent systematic review found that shallow sites (4-6 mm) achieved a mean PPD reduction of 1.5 mm at 6/8 months whilst deeper sites (≥7 mm) achieved an even better PPD reduction of 2.6 mm (Suvan et al., 2020), confirming the PPD reductions in this study were conservative compared to previous studies (Badersten et al., 1984, D'Auito et al., 2005). When the cohort was split into high and low responders, the low responders' groups achieved a mean PPD reduction of 0.0489 mm whilst the high responders group achieved 1.0028 mm, which was still a poorer NSPT response compared to previous studies.

When addressing the primary hypothesis of whether NSPT outcomes are poorer in SEA groups compared to other ethnic groups, no differences between ethnic groups were found. Firstly, with the Chi squared analysis where outcome was dichotomised according to good responders and bad responders, there was no significant difference in NSPT treatment response in the SEA population compared to other groups. Moreover, when mean PPD change was utilised as an outcome variable, Kruskal-Wallis tests were utilised as a non-parametric alternative to ANOVA to compare changes over time in the means of i) all the ethnic groups separately and ii) the SEA group to the non-SEA group. Both tests showed no differences in PPD change between groups. In order to make comparisons in this study, ethnic minorities were merged into broader groups, e.g., SEA and non-SEA, which may ignore the heterogeneity that exists between ethnic groups (Nazroo 2003; Marcenes et al. 2013). However due to the lack of patients within each ethnicity, this was not possible. Furthermore, we chose to focus on the Indian, Pakistani and Bangladeshi ethnic minority groups in this study as these groups are highly representative of Tower Hamlets and the

area surrounding RLDH. Furthermore, as discussed, data from a large sample of our local East London population previously suggested that periodontitis is more severe in these minority ethnic groups (Delgado-Angulo et al., 2016). Despite attempting to analyse potential differences in treatment outcome between ethnic minorities, we did not collect any genetic samples or attempt to analyse the data that may have contributed to such potential differences. It would therefore have been difficult to establish if such differences were due to a genetic/biologic preposition or due to socioeconomic cofounders.

When investigating factors within the SEA population that may increase their risk of periodontal disease, it was found that a larger proportion of the SEA ethnic group get referred at a younger age (<40 years) compared to other ethnic groups. Although narrowly missing the 5% significance level (p=0.0671), there were more young (<40 years) South East Asians in the treated cohort compared to the other ethnic groups. Furthermore, a significantly higher level of irregular dental attendance in the SEA ethnic group compared to all other groups combined (p=0.0002). There were however considerably lower levels of self-reported stress in the SEA ethnic group compared to all other groups combined (p=0.0461**), although it is unrealistic to extrapolate this to all SE populations in the UK. However, when interpreting these results, tremendous care must be taken as it is not clear that we have a representative sample of the population and there is a considerable risk of bias. Firstly, this is because of the nature of how the specific cohort for the study was recruited with limited numbers, and secondly since such patietns are referred for periodontal treatment at RLDH and are likely to have more severe disease than is present in the general population.

No other risk factors, including smoking and diabetes, showed any difference between SEA and other ethnic groups. This contradicts previous studies regarding the prevalence of diabetes which have shown that type 2 diabetes is up to six times more common in SEA groups (Khunti et al., 2009). With regards to plaque control, there was no significant difference in the proportion of patients with baseline poor plaque control (>50% score) in the SEA ethnic group compared to all other groups combined (p=0.3614). Plaque levels are not significantly different in SEA versus other groups. This was assessed with a 2 by 2 square analysis of dichotomous outcomes by splitting the group into high and low plaque groups. Chi squared tests showed no significant differences (p=0.4382). Furthermore, a Kruskal Wallis analysis comparing all ethnicities, as well as comparing the SE group to others found no difference in baseline plaque, reassessment plaque scores and plaque changes. BOP also showed no differences between these groups in this analysis, neither at baseline nor at reassessment.

The effectiveness of NSPT was demonstrated to be mainly influenced by baseline PPD. Those patients with the highest mean PPD at baseline responded better to NSPT compared to those with lower mean PPD at baseline (p=0.0001). This therefore showed that patients with more disease at baseline (mean PPD) respond with greater amounts of PPD reduction compared to those with less disease. This was confirmed in the multilevel analysis which showed that patients presenting with higher levels of disease (Highest 50% percentile) at baseline (Mean PPD) are 3.9 times more likely to have a good non-surgical treatment response (more pocket depth reduction) compared to those with lower levels of disease (Lowest 50% percentile). Some caution may be necessary with such conclusions however as this analysis was based on patients being dichotomised at baseline into higher and lower levels of disease as well as for the outcome. The binary logistic regression model demonstrated that baseline disease severity was the only significant predictor of good treatment response in the model, explaining 26.6% of the variability in the dependent outcome variable. Previous studies have also shown that baseline PPD influenced NSPT outcome. According to multilevel linear regression models (Jiao et al., 2017), by increasing initial PPD by 1 mm led to an increased site PPD reductions at site level of 0.56 mm and 0.59 mm for sites with baseline PD ≥ 5 mm.

Furthermore, the initial probing depth was also found to negatively affect the predictability of the treatment outcome when pocket closure was utilised as the main outcome variable (Tomasi et al., 2007). Such a response where deeper initial pockets show more PPD reduction has been questioned by previous studies (Wan et al., 2009, Tomasi et al., 2007, D'Auito et al 2005). They considered this may be due to ''mathematical coupling'' (Tu et al., 2002, 2005; Tu & Gilthorpe 2007) and such an association between baseline PPD and PPD reduction should be interpreted with caution. However, a recent multilevel analysis (Jiao et al., 2017) included baseline PPD in their multilevel model as it was the most influential factor on the outcome of NSPT, as in this study, and it helped to explain the variance components model. They did however comment that despite PPD at baseline having a significant influence on the treatment outcomes of NSPT, the reliability of the effect was comprised by the nature of regression. Another study also found that baseline PPD had a significant impact on PPD reduction with deeper pockets showing a greater potential for reduction. However, again the authors warned about "mathematical coupling" in multivariate models (D'Auito et al., 2005), such that baseline PPD was excluded from the model.

In this study, smoking was not found to have a significant effect on the outcome of NSPT. This was demonstrated both when current and ex-smokers were grouped (smoking exposure) or when current smokers were compared to non-smokers (ex and non-smokers), however there were few current smokers in this cohort (n=9). Due to the very small chort size for smokers, the findings from this retrospective are unlikely to be representative of the population, therefore such a conclusion that smoking does not affect NSPT should not be elicited.

Previous studies have in fact determined that smoking does have a negative impact on the outcome of NSPT (Ryder et al., 1999, Meinberg et al., 2001, Kamma & Baehni, 2003, Trombelli et al., 2003, Cortellini & Tonetti, 2004, Fardal et al., 2004, D'Aiuto et al., 2005, Labriola et al., 2005). The previous multilevel analysis in a Chinese population (Wan et al., 2009) found that smokers showed less favourable responses after non-surgical therapy, presenting with a significantly higher percentage of residual pockets at twelve months. Furthermore, this influence of impaired PPD reduction was found both at all sites and those initially >5 mm. Despite this, when utilising probing attachment level (PAL) gain as the outcome variable for NSPT, no statistically significant difference was found in PAL gain between smokers and non-smokers. This corroborates a systematic review on the effect of smoking on non-surgical therapy (Labriola et al., 2005). However, a review of the literature suggests that most studies show that clinical attachment gain in response to periodontal therapy is impaired in smokers (Heasman et al., 2006). When pocket closure was evaluated as the main outcome variable (Tomasi et al., 2007) for NSPT, smoking habits were again determined to be a significant factor leading to inferior outcomes. This study utilised a multilevel model to obtain predictions which suggested that that the magnitude of difference in terms of the chance to obtain ''pocket closure'' was about 30% lower in a smoker. Furthermore, their continuous model revealed an interaction between smoking and initial PPD such that the negative effect of smoking was more evident in initially deep pockets. This finding has also been reported in previous studies comparing the effect of cause-related therapy in smokers and non-smokers (Kinane & Radvar 1997, Tomasi & Wennstrom 2004). Conceivable explanations for an inferior treatment outcome in smokers have been previously posited (Biddle et al., 2001); it has been suggested this poorer response to NSPT may be caused by reduced probe tip penetration within the tissue of smokers as less inflammation is present, and a lower height of the supra-bony connective tissue portion, especially where PPDs >5 mm. There is therefore less potential for reduction in probing after successful resolution of inflammation.

Another explanation provided describes the ecological environment of deep periodontal pockets in smokers being more difficult to alter by mechanical instrumentation. This is given

further credence as studies have observed that periodontally untreated and treated smokers harbour a subgingival microflora containing a higher prevalence of periodontal pathogens e.g. Bacteroides forsythus, than non-smokers (Zambon et al., 1996, Darby et al., 2000, Bostrom et al., 2001, Haffajee & Socransky 2001, van Winkelhoff et al., 2001). A study undertaken on the same east London population investigating prognostic factors for NSPT in aggressive periodontitis patients (Hughes et al., 2006) confirms the observation that smoking is a major negative prognostic factor in the outcome of NSPT. Despite no differences in age, severity of disease, (based on the number of deep sites [PPD > 5 mm]), or plaque levels between smokers and non-smokers, the smokers had a poorer outcome with less overall mean PPD reduction. Smokers also had more non-responding sites compared with non-smokers. Half of the non-responders in this study were smokers whilst only 25.4% of non-smokers were non responders; the authors calculated the risk of non-response in smokers had an odds ratio of 2.9. At site level, smokers had an increased risk of 40% of sites not responding to treatment, with an odds ratio of 5.9 of non-response. Unlike previous possible suggested mechanisms for smoking effecting PPD measurements, this study supported the idea that there is a direct effect on periodontal healing rather than influencing the levels of plaque or type of pathogens within plaque. Regardless, all these studies contradict the finding in our study that smoking is not a factor that may affect the outcome of NSPT, most likely due to the number of smokers being small in our cohort, but this may also be influenced by the 'quality' of probing by the examiners.

With regards to plaque control, better treatment outcomes in terms of mean PPD reduction were found with patients with higher levels of plaque (>50% FMPS) at baseline, however there was no association between reassessment plaque (FMPS) levels and NSPT outcome. Not only did higher baseline FMPS scores have superior PPD reduction, but counter intuitively, compliant patients had poorer non-surgical responses compared to compliant patients, of which only at reassessment only 40/108 (37.0%) of patients were compliant in terms of FMPS <20%. These potentially controversial findings contradict previous studies and may have been influenced by potential limitations of the size of the cohort, levels of disease within this cohort and the impact of COVID-19 during NSPT affecting the time between instrumentation and reassessment.

Other studies have conversely shown that initially diseased sites with plaque absence at baseline underwent greater PPD reduction after NSPT (Wan et al., 2009). Another study demonstrated in a multilevel analysis that the aggregated variable of plaque score at subject level was not significant, but the presence of plaque at single sites was significant (Tomasi et al., 2007). However, initial mean FMPS was only 26% as OHI was given prior to baseline examination, explaining the subject and site level variations for the impact of plaque on outcome variables. Meanwhile when analysing pre and post treatment plaque scores as prognostic factors, plaque was found not to be associated with NSPT outcome in generalised aggressive periodontitis cases (Hughes et al., 2006). A previous classic study (Axtelius et al., 1999) has evaluated the influence of plaque on the tooth site level, establishing a significant negative effect on the treatment outcome. Although in this study poorer plaque control patients obtained better outcomes, patients that managed to achieve at least a 10% improvement in FMPS between baseline and reassessment did however show significantly better non-surgical treatment outcomes (P=0.05*).

Previous studies have shown that patients with poor glycaemic control, long diabetes duration, and other diabetic complications are likely to have an impaired response to periodontal therapy due to compromised wound healing and tissue repair (Tervonen and Karjalainen, 1997). This study did not demonstrate a significant difference in treatment response between diabetics and non-diabetics. However, we did not determine the diabetic

control with these patients, therefore it is difficult to establish if this lack of observed differences in diabetics was confounded by this factor.

There were slightly more favourable treatment responses observed in males compared to female patients. This is despite previous studies demonstrating that men may exhibit worse periodontal conditions (Brown *et al*., 1989, Albandar, 2002, Susin *et al*., 2004) as a result of women performing better oral hygiene (OH) (Hugoson *et al*., 1998; Christensen *et al*., 2003).

This study also demonstrated that there was a significant difference in NSPT response in comparing patients reassessed at <120 days (n=80) compared to those reassessed at >120 days (n=28), with those waiting >4 months to reassess having poorer treatment outcomes. This observation contrasts to another long-term study assessing the outcomes of NSPT where further reductions were observed after long periods of reassessment (Badersten et al., 1984), with further PPD reductions being observed for up to six months. However in this study regular OHI and RSD every three months was undertaken. Despite the findings in this study, there are potential issues with the significant variation in the time between NSPT and reassessment. This was especially the case as several patients' treatment was affected by the COVID-19 pandemic, which increased the time between assessments. This is likely to have influenced plaque control and as well as the extent of PPD reduction as during this time no OHI or instrumentation was performed.

In this study we focused on specific patient level factors, and the multilevel analysis predictor variables that were introduced into the model were chosen based on the univariate analysis. When analysing patient, site, and tooth level factors affecting NSPT, an earlier multilevel investigation established from 94 systemically healthy subjects (D'Auito et al., 2005) with severe generalised periodontitis that the major source of variability in decreasing PPD following standardised NSPT (oral hygiene instructions and mechanical subgingival debridement) was attributable to predominantly site level factors, (80%). Tooth and patient level factors explained 12% and 8% of the variance respectively. Our study only focused on patient level outcome and assessed patient level dependent variables, therefore several factors may have not been considered in the multilevel analyses which may be true contributing factors.

Tooth type has previously been investigated (Jiao et al., 2017) showing that molars had inferior treatment outcomes and worse prognosis due to anatomic factors (such as presence of furcation, concavities on the root surfaces and cervical enamel projections) and more posterior position in the arches (Heitz-Mayfield et al. 2002, Matthews & Tabesh 2004, Angst et al. 2013). Other multilevel analysis studies have also shown that PPD reduction was less in molars than non-molar teeth as a result of poorer accessibility for sub-gingival instrumentation (Tomasi et al., 2007, Wan et al., 2009). Presence of furcation involvement was also considered to be a contributing factor for reduced pocket closure in molars (Tomasi et al., 2007), however, tooth sites associated with furcation involvement were not included in the multilevel analysis. The observation that incisors and canines responded better than premolars and molars has also been interpreted in terms of both a higher efficacy of subgingival instrumentation at single-rooted teeth as well as higher likelihood of thin gingival tissues associated with pockets in anterior teeth (D'Auito et al., 2005). Such teeth with thin periodontal phenotype are likely to heal with more recession and hence more pocket reduction than in posterior teeth. They also found that furcation involvement did not have a significant impact on PPD reduction as previously reported (Loos et al.,1988,1989, Nordland et al., 1987). Again, furcation involvement at site level was not specifically considered a prognostic factor but was instead interpreted as a tooth-level variable. Site level analysis did however show that greater PPD reductions were observed for interdental sites compared to facial and/or lingual and palatal sites.

Furthermore, severe mobility (degree II or III mobility (Miller, 1950) has been demonstrated to be associated with reduced PPD reduction after NSPT (Jiao et al., 2017, D'Auito et al., 2005). However, despite demonstrating the negative impact of tooth mobility on PPD reductions in their multivariate analysis, the univariate analysis indicated that better PPD reductions were observed in teeth with a high degree of mobility (D'Auito et al., 2005). This was considered to be plausible as severely mobile teeth have normally lost a significant amount of clinical attachment and normally present with deep pockets, whilst deep pockets are more likely to display better PPD reductions. However, after correcting for confounders, mobility in itself had a negative impact on the clinical responses following subgingival debridement.

Another patient level factor not considered was frequency of periodontal maintenance (FPM)(Jansson & Hagstrom 2002, Leininger et al. 2010).Descriptive analysis at patient level (Jiao et al 2017) as well as multilevel analysis at site level has demonstrated greater bleeding index (BI) and PPD reductions in patients with who attend FPM regularly. This factor could not easily be assessed in this study as patients are referred to RLDH for active treatment and discharged back to the general dentist for maintenance.

The outcome measure utilised in this study was PPD change. This outcome variable has been utilised in many studes previously when assessing the efficacy of NSPT and factors that may contribute to treatment success (Badersten et al., 1990, Claffey and Egelberg, 1995, Lang et al., 2008b, Matuliene et al., 2008, Westfelt et al., 1998, D'Aiuto et al., 2005, Tomasi et al., 2007, Wan et al., 2009, Jiao et al., 2017). An alternative outcome measure that may be more representative as a clinical endpoint of periodontal treatment success is pocket closure, defined as having no BOP and achieving "closed pockets", where PPD achieves a depth of ≤ 4 mm. This clinical endpoint measurement also may also be easier to interpret than mean PPD changes and maybe therefore make the findings more transferable to daily practice allowing better communication with the patient (Tomasi and Wennström, 2017). Further, when this outcome is applied with the use of a multilevel logistic analysis (Tomasi et al., 2007) it allows a comprehensive and clear assessment of factors that may influence treatment outcome (Tomasi and Wennström, 2017). Unfortunately, in our study, our data collection did not allow for such an outcome variable to be utilised however this may have provided more representative findings. Another study instead tried to identify patients who responded to treatment or became more stable (Hughes et al., 2006b) by dichotomising patients into ''responding'' and ''non-responding'' patients, nonresponding patients being defined as those with a minimum of 30% of their deep sites that did not respond to the treatment provided. In our study we split the study cohort by dichotomising subjects into two groups, high and low responders based on the highest 54 and lowest 54 mean PPD changes. This may introduce some bias as the difference in PPD change between the lowest 25 responders and highest 25 non responders is only 0.62 mm, ranging in these 50 individuals between 0.75 mm to 0.13 mm respectively. There was therefore very little difference in the outcome between some non-responders and responders however these were dichotomised into these groups solely based on the top performing 54 subjects.

There are several limitations to this study which may have contributed to these findings. Firstly, due to challenges described during the data collection, such as poor record keeping, missing six point charts or specific data on charts, the number of cases that could be included was reduced. This also meant that comparing different groups of individuals, the numbers of some groups, e.g., current smokers (n=9) were very small and made comparisons difficult and possibly unreliable. Therefore, the study is very likely to be under powered making the findings of this study, and lack of differences between certain groups potentially unreliable. Furthermore, this lack of data made it more difficult for tooth and site level comparisons to be made and for other outcome variables to be considered. Instead, as discussed, outcome was essentially based on mean PPD, and individuals split into high and

low responders. Not only was the sample size relatively small for a retrospective analysis, but this cohort has significant severity of periodontal disease with most individuals having stage III or IV disease. This could potentially mean PPD reduction may be limited especially given that compliance with oral hygiene at baseline was relatively poor. Conversely if patients had more deeper sites, these areas theoretically may show more PPD reduction. Such limitations with respect to the cohort size may therefore introduce a type 2 error, where we failed to show significance when significance is indeed present and create issues of being under powered for the multilevel analysis. Rather then choosing the high responders and low responders based on the highest 54 and lowest 54 mean PPD change individuals respectively, a threshold could have been established in which a 'good' response was defined. If we used Suvan et al (2020) as a reference for mean PPD reduction, we could set the minimum PPD change as 1.7 mm, which was the weighted mean PPD reduction at 8/8 months in this systematic review. However, by using this method, only 4 individuals (3.7%) from the cohort would satisfy these criteria as 'high' responders. This follows on from the previous discussion regarding the mean PPD change overall being lower than other studies assessing outcomes of NSPT, which could be due to operator or patents factors that may be confounding in this cohort.

Furthermore, although operator factors were not assessed, these may of course influence the potential impact of treatment. This is particularly relevant as there was no calibration of examiners, both intra operatively and inter operatively. Given the nature of such a retrospective study, such limitations also extended to a lack of calibration in how NSPT was undertaken, which may therefore have introduced variations in instruments used, time spent overall, time spent with machine and hand instrumentation, levels of oral hygiene prior to commencing NSPT, whether previous NSPT was undertaken. Such factors may clearly have confounded the outcomes, as previously discussed, operator experience may also contribute to the effectiveness of NSPT, whilst time spent may also be a factor, and whether repeat instrumentation has been undertaken. Another consideration was that patients that also received systemic or local antimicrobials was not specified or excluded.

As with another multilevel retrospective multilevel analysis (Jiao et al., 2017), this study shares limitations of observational and retrospective studies. Differences in characteristics of the population and confounding factors may lead to bias and threaten the validity of the treatment outcomes (Concato, 2012). This specific cohort as well as having poor compliance from baseline, were also patients that were referred to the hospital for severe levels of disease. Not only this, but there is considerable variation in the levels of disease and compliance with attendance as well as with oral hygiene that may affect NSPT outcome. With regards to operator factors, these were not assessed other than time of reassessment, mainly because treatment itself between individuals is unlikely to vary significantly. The protocol for most operators is to use hand and ultrasonic instruments for NSPT and perform OHI prior and during treatment. Again, comparisons could not be made for such factors as time taken for instrumentation, type of Cavitron™ tip, methods used, extent of OH in terms of brushing and Tepes™ advised, was often not documented in the notes. Whilst treatment methods are likely to have been similar, the experience of clinicians could not be easily assessed and was not recorded, again making such comparisons difficult.

5.2 Future Work

A similar study could be undertaken again however a larger study (sample size) with more participants to increase study power for primary outcomes would be beneficial. As previously discussed, a Chinese multilevel retrospective study (Jiao et al., 2017) with similar design took information from 10,789 records. Furthermore, as part of a larger study, more site and tooth level assessments of prognostic factors can be assessed and introduced into a multivariable linear regression model. Alternatively, a prospective longitudinal clinical trial could be instead considered (D'Auito et al., 2005) with stricter inclusion criteria so group sizes are more similar, and comparisons can be made more easily. Additionally, timelines to reassessment can be more readily controlled and follow up studies so that comparisons over time can also be made as classical studies have done (Badersten et al., 1984). Furthermore, operator factors could also be compared such as types of instrumentation, and even full mouth versus quadrant protocols which have previously been assessed and no differences found (Suvan et al., 2020). Due to multiple examiners, there was increased variability in probing and so reducing probing consistency in our study. For future studies, the operators and examiners should be calibrated to minimise the variation. Additionally, examiners can be blinded to the treatment method provided therefore helping reduce bias, although double or triple blinding where the operator and patient is blinding would be difficult.

This study unfortunately had a poorly compliant cohort at baseline with many remaining uncompliant throughout. Ideally a more compliant cohort would be considered. This could be controlled by ensuring adequate OH before proceeding with subgingival instrumentation. Again, a prospective clinical trial, with possible randomisation of operator components may help to investigate this, assuming we ensure it would be appropriately powered for primary outcome. Randomised control trials are superior in the hierarchy of evidence compared to this retrospective study. Although there are clear benefits with a prospective clinical trial compared to a retrospective study, such a clinical trial may introduce "artificial study conditions" which may vary from what can be seen in real world settings.

CHAPTER 6: CONCLUSIONS

This study was significantly under powered to demonstrate any clinically significant findings. Despite this, the effectiveness of NSPT was again shown in this hospital-based retrospective study, although no differences were found between different ethnic groups, or between south east Asian patients and others. The outcomes of NSPT were mainly influenced by baseline pocket depths. Further research may be required to establish the relative effect of different patient, tooth, site and operator level factors, including whether ethnicity is indeed a prognostic factor. This may require a prospective study with more specific inclusion, exclusion criteria and calibration of operators and examiners.

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APPENDICES

Appendix 1 -Abbreviation list

A.a	Aggregatibacter actinomycetemcomitans
ANOVA	Analysis of Variance
B	Baseline
BI	Bleeding Index
BOP	Bleeding on Probing
BSP	British Society of Periodontology and Implant Dentistry
CAL	Clinical Attachment Loss
CHX	Chlorhexidine Gluconate
DM	Diabetes Mellitus
DPTT	Diabetes and Periodontal Therapy Trial
EFP	European Federation of Periodontology
FI	Furcation involvement
fMLP	N-formyl-methyl-leucylphenyl- alanine
FMBS	Full Mouth Bleeding Score
FMD	Full Mouth Disinfection
FMPS	Full Mouth Plaque Score
FMS	Full Mouth Scaling
FPM	Frequency of Periodontal Maintenance
GCF	Gingival Crevicular Fluid
HbA1C	Glycosylated Haemoglobin
IL- 6-174 G/C	G/C promoter polymorphism at nt (-174) of the interleukin-6
ITI	International team of Implantology
mm	millimetres
MWF	Modified Widman Flap
NHANES	National Health and Nutrition Examination Survey
no.	Number of:
NS	Non-Surgical
NS1	Reassessment 1
NSPT	Non-Surgical Periodontal Treatment/Therapy
OH	Oral Hygiene
OHI	Oral Hygiene Instructions
OFD	Open Flap Debridement
PAL	Probing Attachment Level
P.g	Porphyromonas gingivalis
PMNs	Polymorphonuclear Leukocyte
PPD	Probing Pocket Depth
PRF	Plaque Retention Factor
PROM	Patient Related Outcome Measure
PS	Plaque score
RLDH	Royal London Dental Hospital
RPL	Root Planing
RSD	Root surface Debridement
SD	Standard Deviation
SE	Standard Error
SEA	South East Asian
SEP	Socioeconomic position
Sig	Significance level
SPSS	Statistical Package for the Social Sciences

SRP	Scaling & Root Planing
T.d	Treponema denticola
USA	United States of America
UK	United Kingdom

Appendix 2: Periodontal Baseline Data Collection Proforma

NHS
Barts Health
NHS Trust

Dental Hospital

Periodontal Baseline Data Collection Proforma

First Name: ..... Surname: .....

Hospital No: ..... Date of birth: .....

Address: .....

.....

DATE: **SUPERVISOR NAME:**

DATE OF INITIAL CONSULTATION: **CONSULTANT IN CHARGE:**

GDP DETAILS: **LAST GDP APPOINTMENT:**

HISTORY:

Presenting complaint/s:

Bleeding Gums	Recurrent Abscesses	Swelling/soreness
Bad Taste/Bad Breath	Food Impact	Sensitivity
Loose Teeth	Drifting Teeth	Poor Appearance
Difficulty in Chewing	Others	

History of Presenting Complaint:

Treatment already received: Local anaesthetic used (circle)
Yes No

Medical History Update:

Medications:

Family/Social History:

Occupation:

Stress: (none) 0 1 2 3 4 5 6 7 8 9 10 (high)

Smoker: No

Yes: For How Long: How Many: What:

Oral Hygiene:	Manual Toothbrush	Electric Brush	No of times/day
	Additional Aids:	Floss	Interdental Brush
		Mouth rinse	Other
Ex-smoker:	When Stopped:	How Many:	What:

CLINICAL EXAMINATION

Extra-oral:	TMJ	Lymphadenopathy	Facial Asymmetry
	Incompetent Lips	Smile Line	Other
Intra-oral:			
Soft Tissues:	Lips & Cheeks	Tongue & Mucosa	Floor of Mouth
	Other		

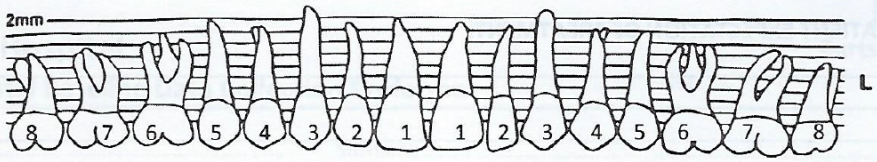
Restorations and Caries:

Tooth Wear															
Caries															
Restorations															
	8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
Restorations															
Caries															
Tooth Wear															

Occlusion:

UPPER

BUCCAL



P.D															
R															
BOP															
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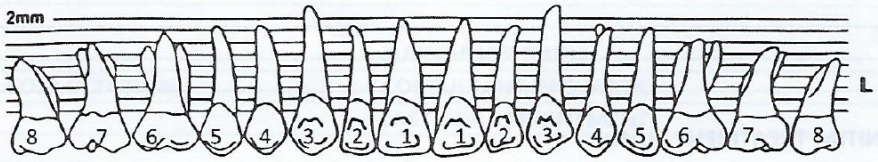
PALATAL



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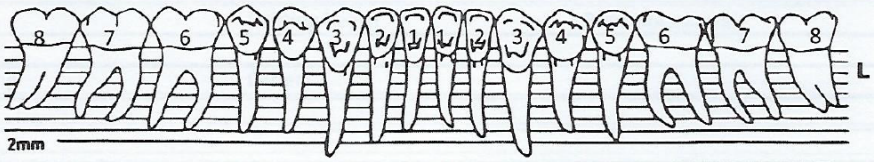
LOWER

LINGUAL



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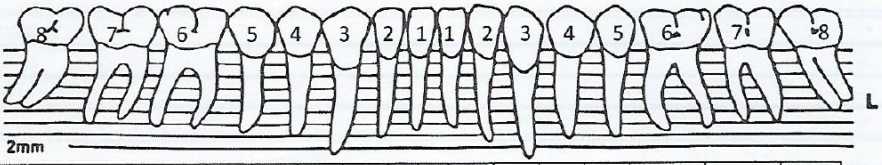

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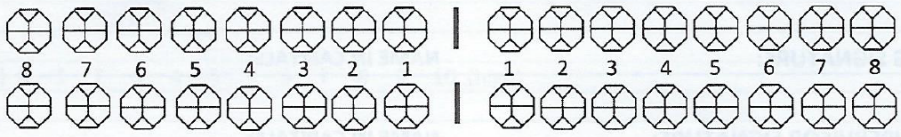
PLAQUE



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RADIOGRAPHIC FINDINGS & VITALITY TESTS:

Other																
Cold																
EPT																
BL%																
IBD																
Apical area																
Endo																
Def Rest																
Caries																
Tooth	8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
Caries																
Def Rest																
Endo																
Apical area																
IBD																
BL%																
EPT																
Cold																
Other																

OTHER FINDINGS:

PATIENT EXPECTATION OF TREATMENT:

GOAL OF TREATMENT:

INITIAL TREATMENT PLAN:

ADDITION LETTER WITH TREATMENT PLAN TO GDP/GP REQUIRED: YES NO

PG SIGNATURE: **NAME IN CAPITALS:**

SUPERVISOR SIGNATURE: **NAME IN CAPITALS:**

DATE:

THA2261

Appendix 3: Periodontal Reassessment Proforma

NHS
Barts Health
NHS Trust

Dental Hospital

Periodontal Reassessment Proforma

First Name: ..... Surname: .....

Hospital No: ..... Date of birth: .....

Address: .....

.....

DATE:

HISTORY:

Presenting Complaint:

Treatment provided since last assessment:

Medical History Update:

Smoking History:

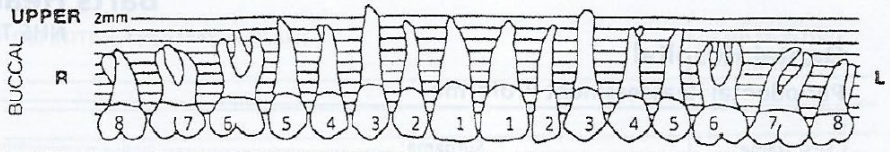
Stress: (none) 0 1 3 4 5 6 7 8 9 10 (high)

PLAQUE SCORE AT LAST VISIT:

BLEEDING SCORE AT LAST VISIT:

UPPER

BUCCAL



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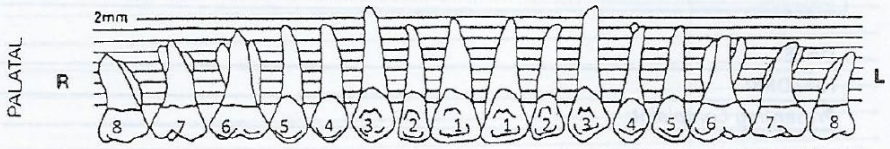

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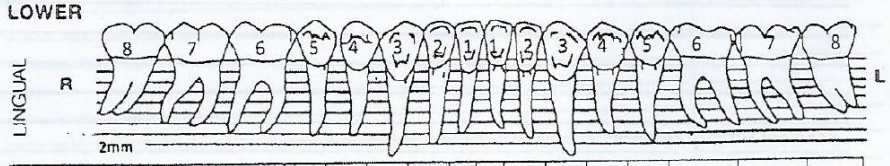

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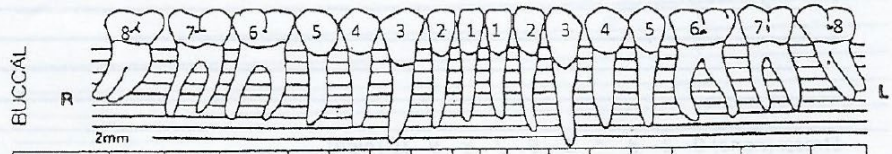

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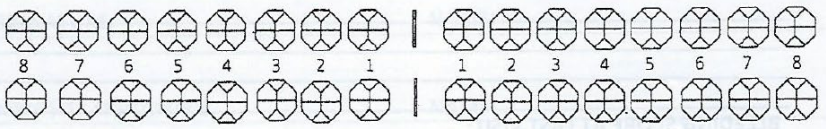

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RESPONSE TO INITIAL TREATMENT: GOOD POOR

REASONS FOR POOR RESPONSE:

TOOTH-BY-TOOTH PROGNOSIS:

OBJECTIVES OF TREATMENT:

OVERALL PROGNOSIS:

DEFINITIVE TREATMENT PLAN:	DATE WHEN PERFORMED

LETTER TO GDP:

PG SIGNATURE: NAME:

SUPERVISOR SIGNATURE: NAME:

THA2232

Appendix 4: Survey 1: Patient Notes 1/3 (Medical and Social History

PERIO 2020: PATIENT NOTES 1/3 (Medical and Social History)

0% complete

Page 1: General Information

1. Study ID Number * Required

2. Initials person extracting the information * Required

3. Date Data Collection started (Today) * Required

7. Borough of Residence (If unknown write Not Stated) * Required

8. Postcode (If unknown write Not Stated) * Required

9. Date Initial Consultation in Clinic * Required

(dd/mm/yyyy)

10. Date of Initial Assessment (Not CC- 1st appointment with treating clinician) Optional

Dates need to be in the format 'DD/MM/YYYY', for example 27/03/1980.
The date should be on or after 01/01/2000.

(dd/mm/yyyy)

11. Date 1st Periodontal Re-Assessment

of England and Wales] Use the option "Other" when Ethnicity was not provided but there is evidence to suggest the ethnicity of this patient. Please explain the reason in the text box * Required

- Not Stated (Data Not Available)
- White - English, Welsh, Scottish, Northern Irish or British
- White- Irish
- White- Gypsy or Irish Traveller
- White- any other white
- Mixed - White and Black Caribbean
- Mixed- White and Black African
- Mixed- White and Asian
- Any other Mixed or Multiple ethnic background
- Asian- Indian
- Asian- Pakistani
- Asian- Bangladeshi
- Asian- Chinese
- Asian- Any other Asian background
- Black- African
- Black Caribbean
- Any Other Black, African or Caribbean backgrounds
- Other- Arab
- Any other ethnic group

(dd/mm/yyyy)

10. Date of Initial Assessment (Not CC- 1st appointment with treating clinician) Optional

Dates need to be in the format 'DD/MM/YYYY', for example 27/03/1980.
The date should be on or after 01/01/2000.

(dd/mm/yyyy)

11. Date 1st Periodontal Re-Assessment Optional

Dates need to be in the format 'DD/MM/YYYY', for example 27/03/1980.
The date should be on or after 01/01/2000.

(dd/mm/yyyy)

Next >

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Patient's Information

4. Date of Birth * Required

(dd/mm/yyyy)

5. Gender * Required

- Male
- Female
- Other

6. Ethnicity [According to the 2011 Census of England and Wales] Use the option "Other" when Ethnicity was not provided but there is evidence to suggest the ethnicity of this patient. Please explain the reason in the text box * Required

- Not Stated (Data Not Available)
- White - English, Welsh, Scottish, Northern Irish or British
- White- Irish
- White- Gypsy or Irish Traveller

age 2: Medical History

2. Study ID Number * Required

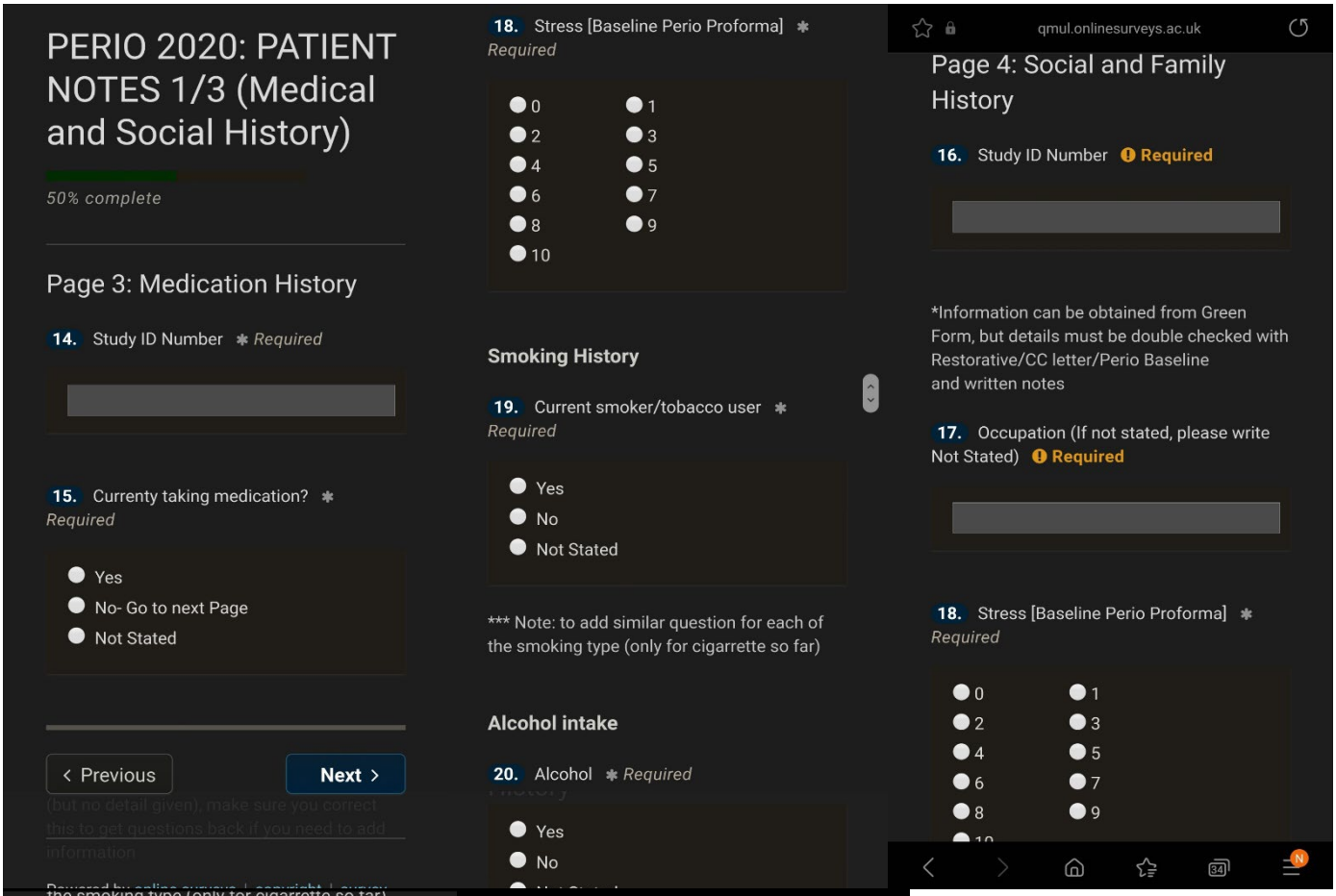
arts Health Medical History (Green orm)- front page

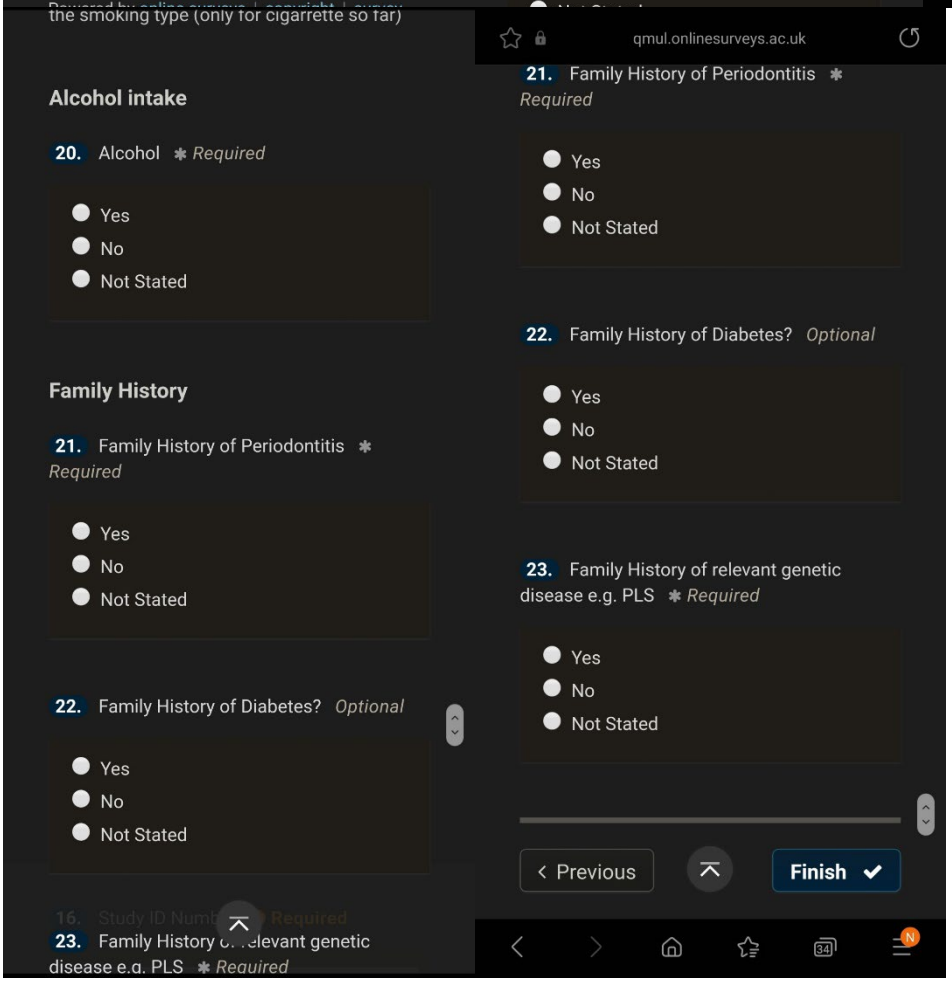
his section follows the structure of the Green orm but it should not be based on this formation only. Please correct if further formation is given in other parts of the otes.

ter each section, you are given specific uestions relevant to this study. These uestions dissapear if you anser No or Yes ut no detail given), make sure you correct is to get questions back if you need to add formation

3. Is the patient generally fit and well? * equired

- Yes- All clear. There is no need to complete Medical History form
- No- There are items in MH to extract
- Other- Add notes without completing MH form (do not use to add medical information)


PERIO 2020: PATIENT NOTES 1/3 (Medical and Social History)
50% complete
Page 3: Medication History
14. Study ID Number * Required
15. Currenty taking medication? * Required
Yes
No- Go to next Page
Not Stated
< Previous
Next >
18. Stress [Baseline Perio Proforma] * Required
0 1 2 3 4 5 6 7 8 9 10
Smoking History
19. Current smoker/tobacco user * Required
Yes
No
Not Stated
*** Note: to add similar question for each of the smoking type (only for cigarette so far)
Alcohol intake
20. Alcohol * Required
Yes
No
Not Stated
qmul.onlinesurveys.ac.uk
Page 4: Social and Family History
16. Study ID Number Required
*Information can be obtained from Green Form, but details must be double checked with Restorative/CC letter/Perio Baseline and written notes
17. Occupation (If not stated, please write Not Stated) Required
18. Stress [Baseline Perio Proforma] * Required
0 1 2 3 4 5 6 7 8 9


the smoking type (only for cigarette so far)
Alcohol intake
20. Alcohol * Required
Yes
No
Not Stated
Family History
21. Family History of Periodontitis * Required
Yes
No
Not Stated
22. Family History of Diabetes? Optional
Yes
No
Not Stated
23. Family History of relevant genetic disease e.g. PLS * Required
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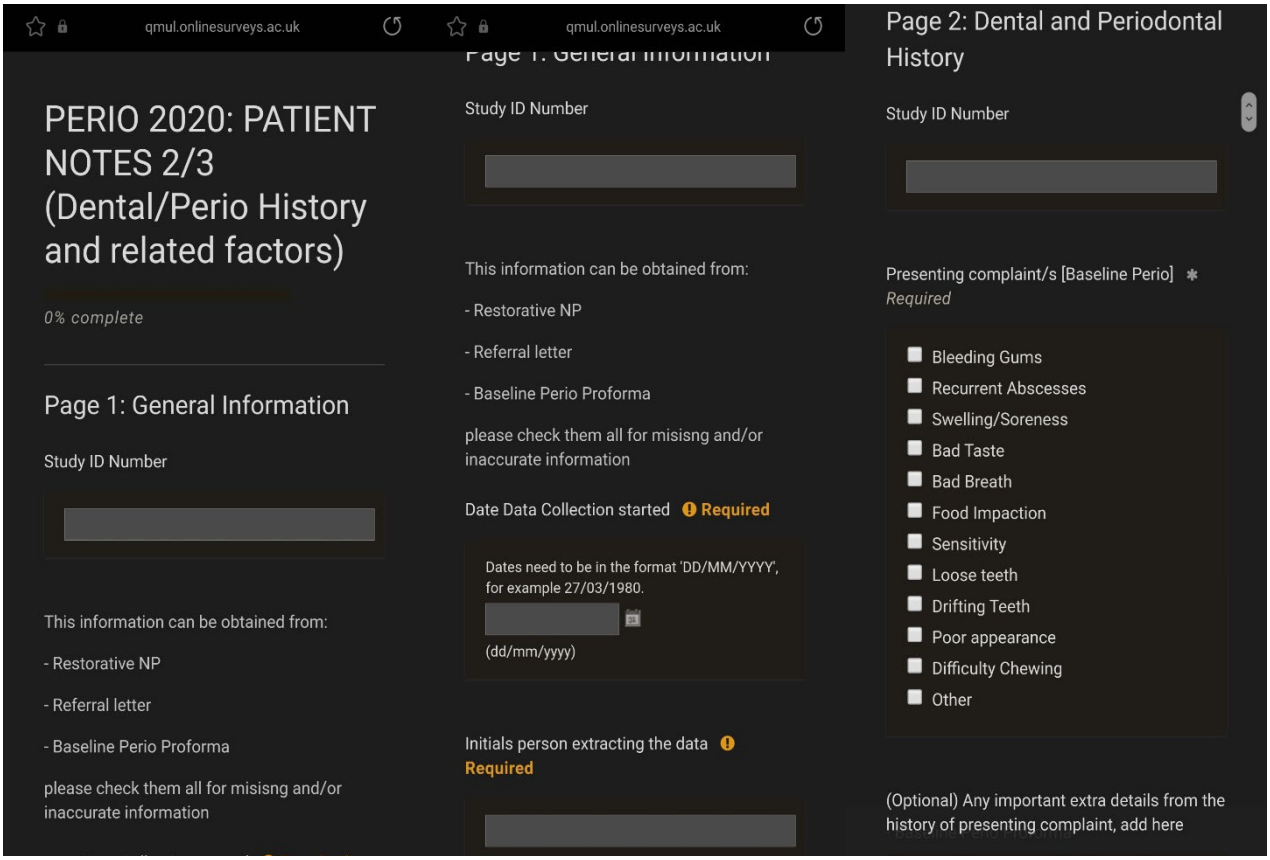
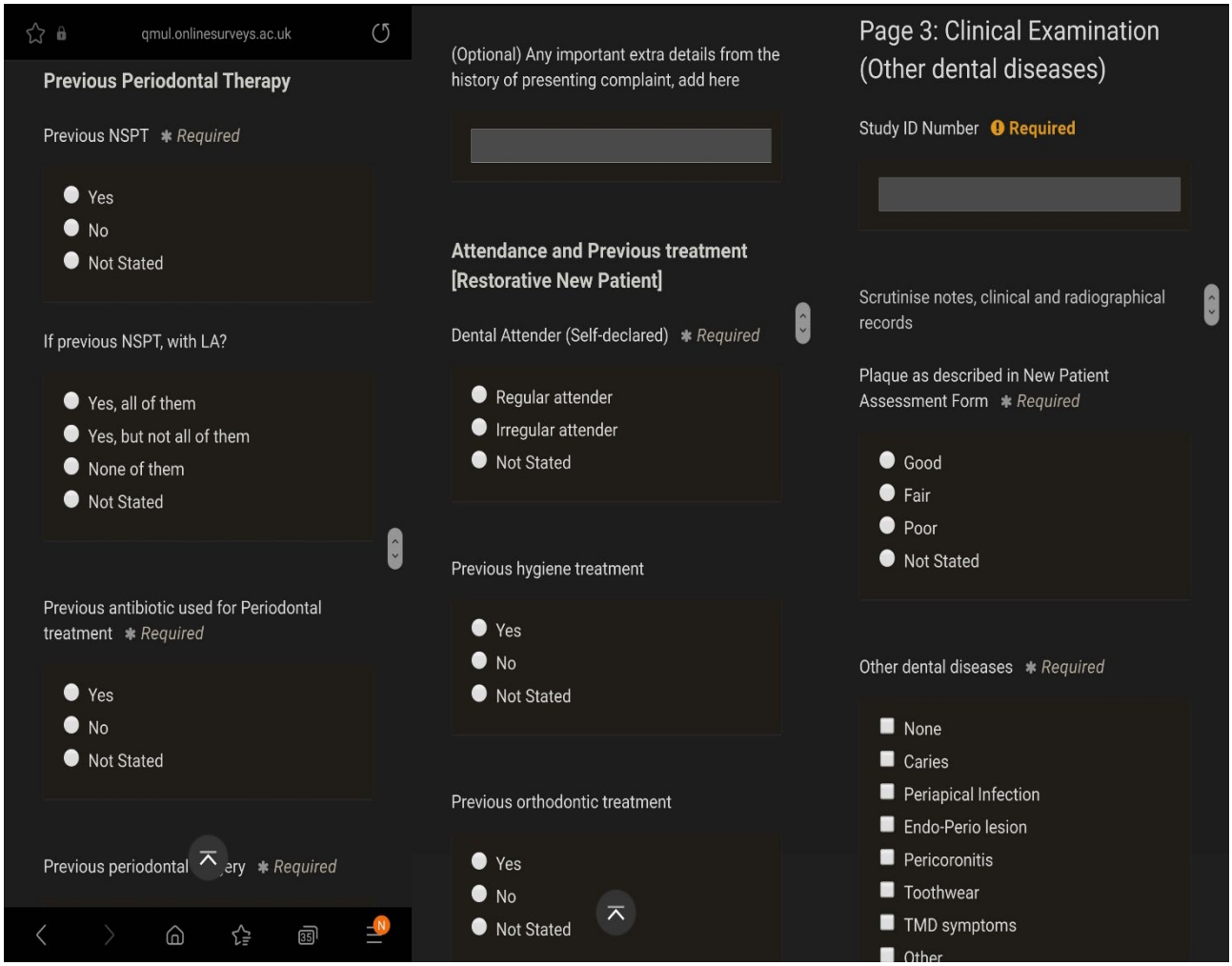
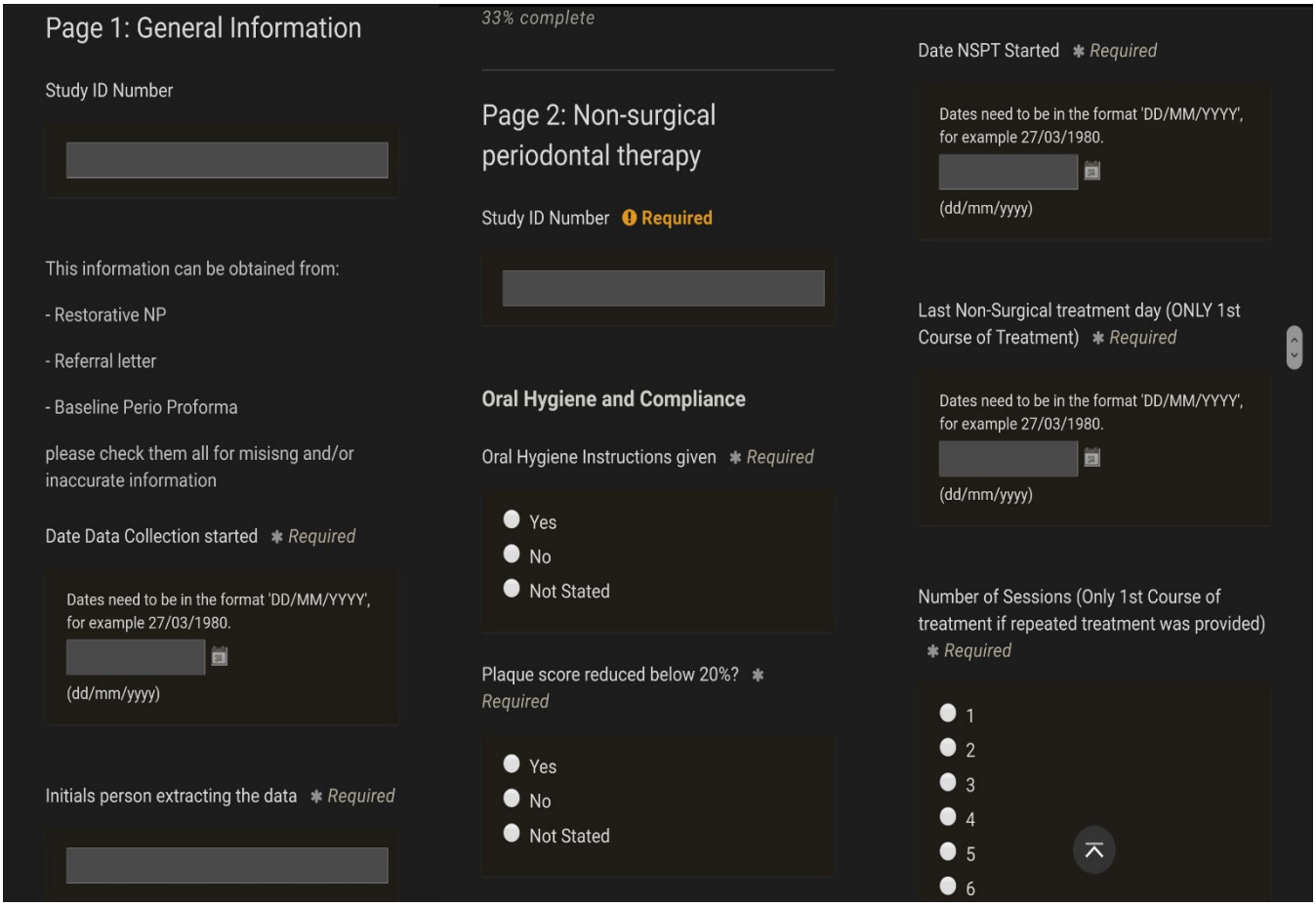
21. Family History of Periodontitis * Required
Yes
No
Not Stated
22. Family History of Diabetes? Optional
Yes
No
Not Stated
23. Family History of relevant genetic disease e.g. PLS * Required
Yes
No
Not Stated
< Previous
Finish

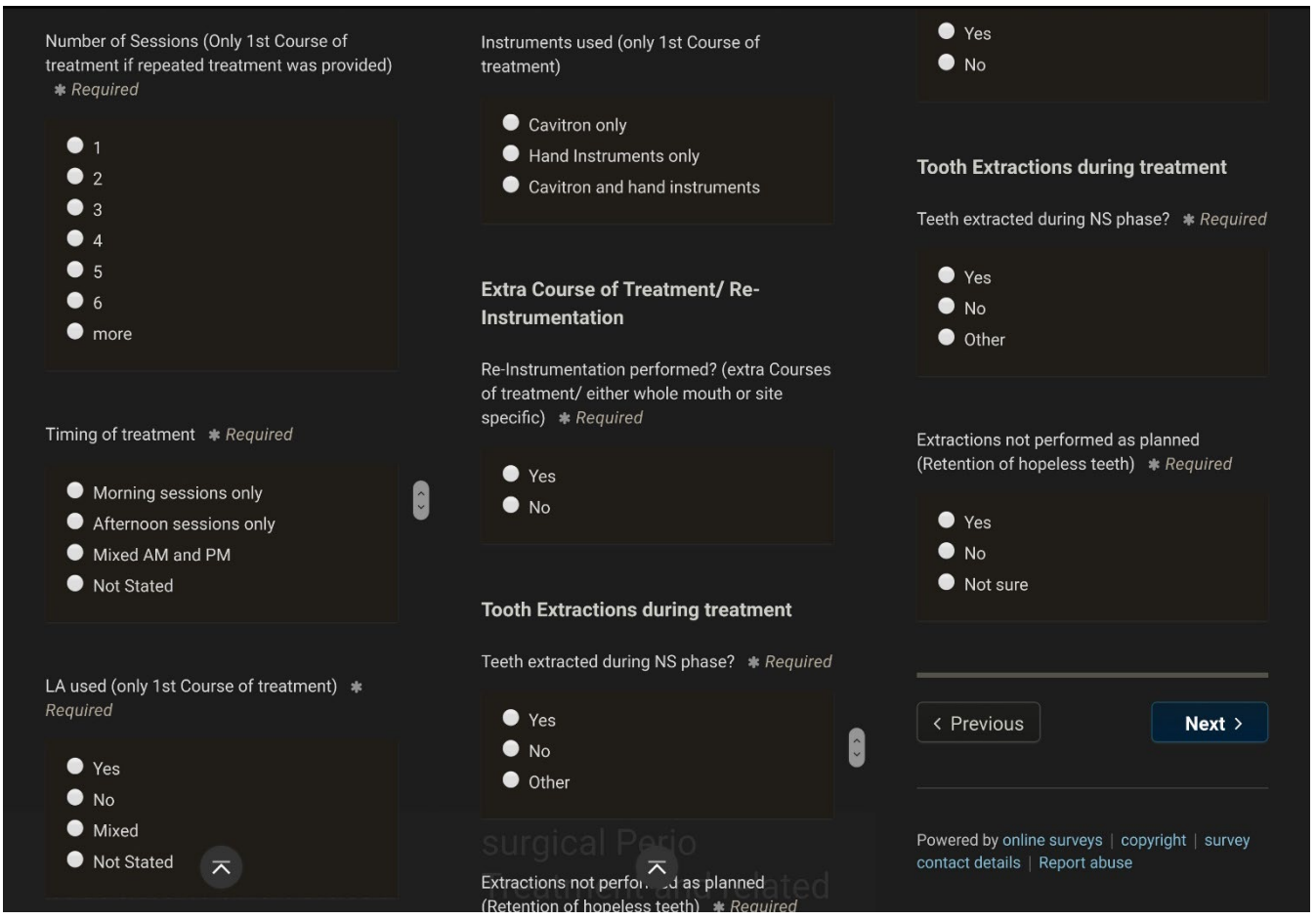
Appendix 5: Survey 2: Patient Notes 1/3 (Dental/Perio History and related factors)

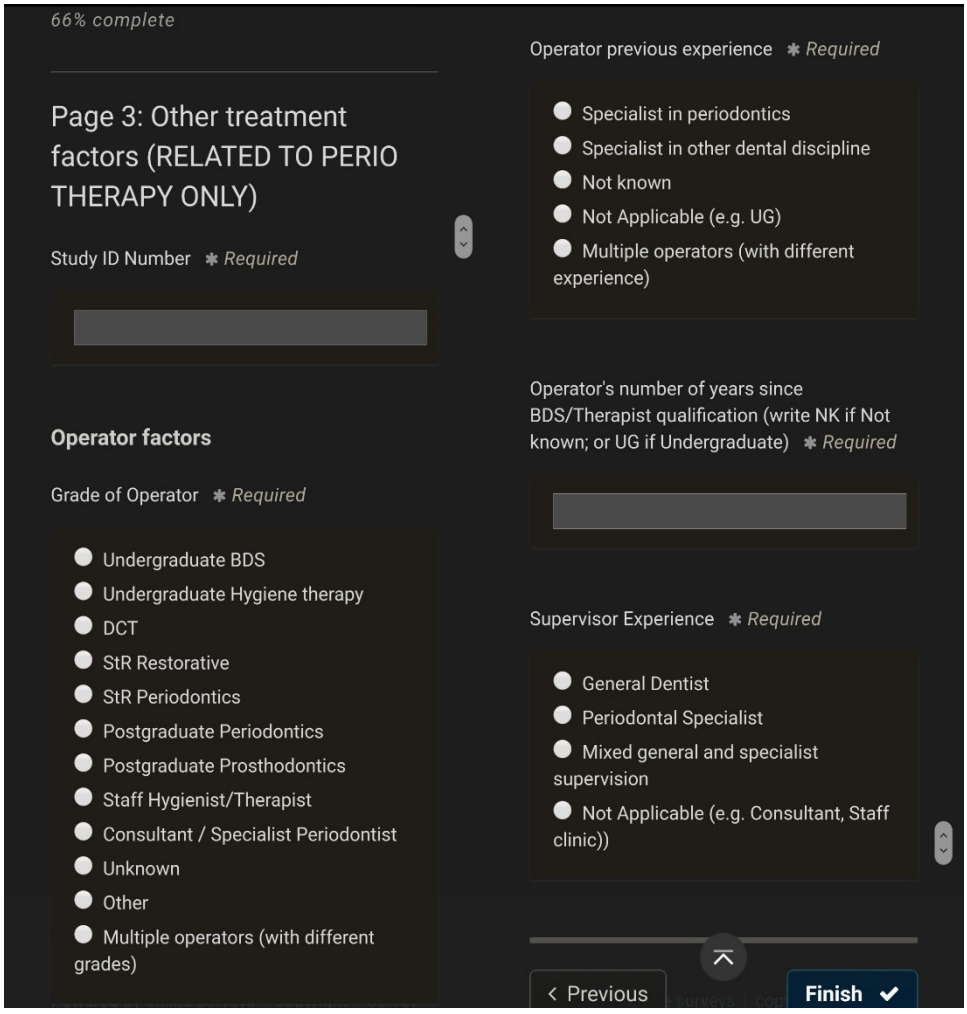




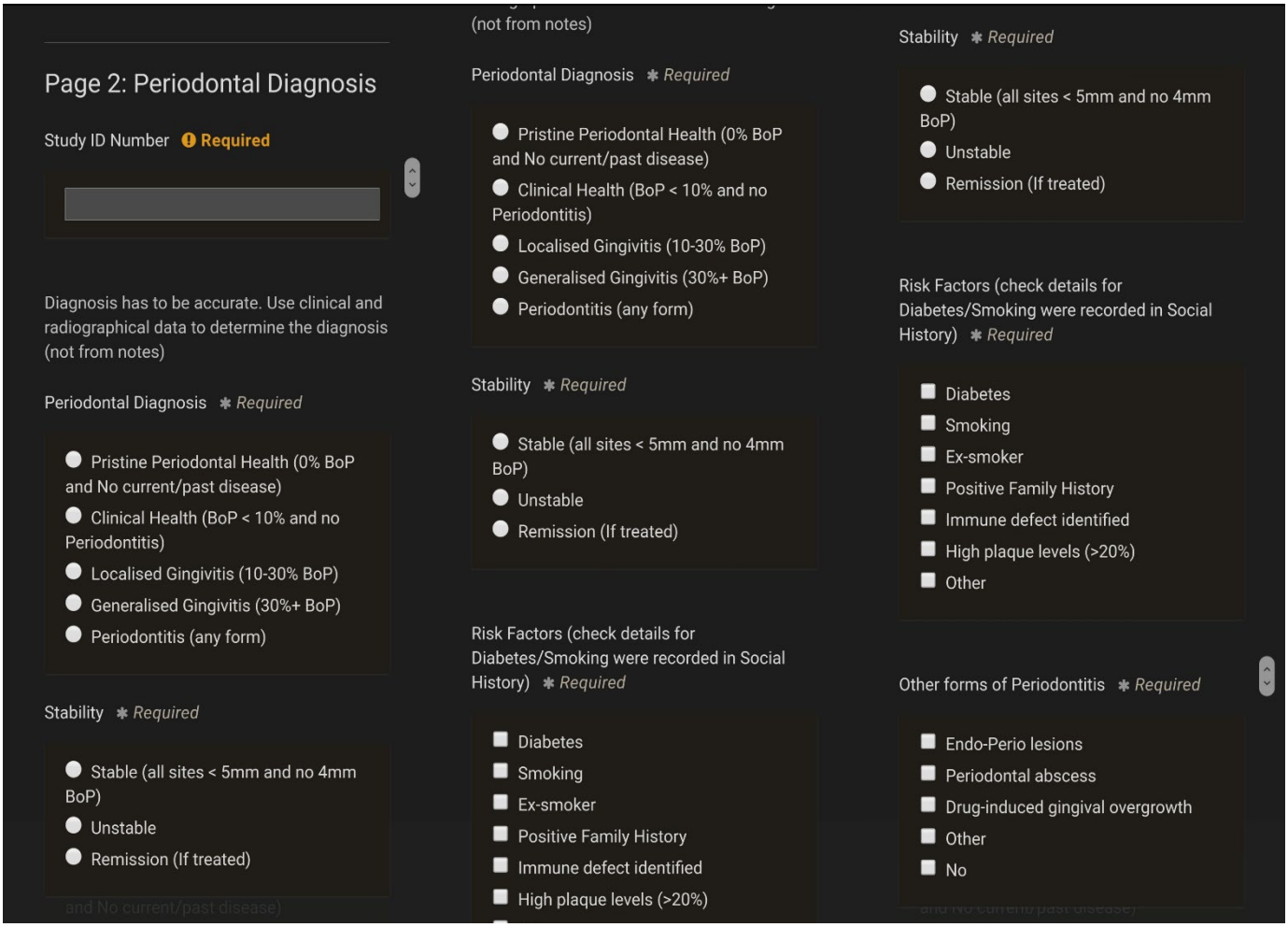
Appendix 6: Survey 3: Patient Notes 1/3 (Non Surgical Perio Treatment and related factors)






66% complete
Page 3: Other treatment factors (RELATED TO PERIO THERAPY ONLY)
Study ID Number * Required
Operator factors
Grade of Operator * Required
Undergraduate BDS
Undergraduate Hygiene therapy
DCT
StR Restorative
StR Periodontics
Postgraduate Periodontics
Postgraduate Prosthodontics
Staff Hygienist/Therapist
Consultant / Specialist Periodontist
Unknown
Other
Multiple operators (with different grades)
Operator previous experience * Required
Specialist in periodontics
Specialist in other dental discipline
Not known
Not Applicable (e.g. UG)
Multiple operators (with different experience)
Operator's number of years since BDS/Therapist qualification (write NK if Not known; or UG if Undergraduate) * Required
Supervisor Experience * Required
General Dentist
Periodontal Specialist
Mixed general and specialist supervision
Not Applicable (e.g. Consultant, Staff clinic))
Previous
Finish

Appendix 7: Survey 4: Perio Diagnosis



Page 2: Periodontal Diagnosis

Study ID Number ! Required

Diagnosis has to be accurate. Use clinical and radiographical data to determine the diagnosis (not from notes)

Periodontal Diagnosis * Required

- Pristine Periodontal Health (0% BoP and No current/past disease)
- Clinical Health (BoP < 10% and no Periodontitis)
- Localised Gingivitis (10-30% BoP)
- Generalised Gingivitis (30%+ BoP)
- Periodontitis (any form)

Stability * Required

- Stable (all sites < 5mm and no 4mm BoP)
- Unstable
- Remission (If treated)

and No current/past disease)

(not from notes)

Periodontal Diagnosis * Required

- Pristine Periodontal Health (0% BoP and No current/past disease)
- Clinical Health (BoP < 10% and no Periodontitis)
- Localised Gingivitis (10-30% BoP)
- Generalised Gingivitis (30%+ BoP)
- Periodontitis (any form)

Stability * Required

- Stable (all sites < 5mm and no 4mm BoP)
- Unstable
- Remission (If treated)

Risk Factors (check details for Diabetes/Smoking were recorded in Social History) * Required

- Diabetes
- Smoking
- Ex-smoker
- Positive Family History
- Immune defect identified
- High plaque levels (>20%)

Stability * Required

- Stable (all sites < 5mm and no 4mm BoP)
- Unstable
- Remission (If treated)

Risk Factors (check details for Diabetes/Smoking were recorded in Social History) * Required

- Diabetes
- Smoking
- Ex-smoker
- Positive Family History
- Immune defect identified
- High plaque levels (>20%)
- Other

Other forms of Periodontitis * Required

- Endo-Perio lesions
- Periodontal abscess
- Drug-induced gingival overgrowth
- Other
- No

Appendix 8: Data extraction data to be extracted

The following patient-related parameters assessed at the initial visit (T0) and the last evaluation (T1) will be extracted from the paper records for analysis:

- Patient level:
  - Age
  - Gender (male versus female);
  - Ethnicity
  - Country of Birth
  - London Borough
  - Relevant medical history including systemic conditions
    - Diabetes mellitus, controlled or uncontrolled, family history of DM
    - Obesity
    - Immunosuppressed, e.g. HIV
    - Blood dyscrasias, e.g. Papillon Lefevre syndrome
    - Rheumatoid arthritis, osteoarthritis
    - Rare conditions that can manifest with periodontal breakdown, e.g. Squamous cell carcinoma
    - Alzheimer's or Dementia
    - Mental Health problems including stress, depression, learning difficulties
    - Pregnancy
  - Medications
    - Bone mass changing medications, i.e. bisphosphonates
    - Immunosuppressants, e.g. methotrexate, Infliximab
    - Gingival enlargement inducing medications e.g. amlodipine, phenytoin
  - Family history of periodontal disease
  - Smoking status (non-smoker classified as patients who has never smoked, ex-smoker classified as patients who quit more than 5 years before initial assessment and smoker classified as patients who have smoked within the last 5 years);
    - If tobacco user, type of tobacco use to be recorded
    - Number per day, number of years smoked to be recorded to establish number of pack years
  - Regular or irregular dental attender (self-reported)
  - Full mouth plaque score (FMPS) – (before treatment, at first reassessment, after treatment)
  - Full mouth bleeding score (FMBS) - (before treatment, at first reassessment, after treatment)
  - Oral hygiene
    - Use of manual or electric toothbrush, how many times per day
    - Use of interdental aids, which sizes?
    - Use of mouthwash
  - Previous periodontal therapy
    - Previous NSPT with or without local anaesthetic
    - Who performed this NSPT?
    - How many rounds of previous NSPT?
    - When was NSPT last performed?
    - Previous periodontal surgery/ regeneration
  - Observation period between T0 and T1
  - Observation period between initial treatment visit and date of re-evaluation of periodontal indices
  - Percentage of sites BOP (before and after treatment)

        - % change in BOP (Pre vs. Post treatment)
    - Probing depths (PPD) (before and after treatment)
        - Highest PPD in charting (mm)
        - Mean PD (mm)
        - No. of unstable sites 4mm + BoP or greater .
        - % of unstable sites at baseline
    - Localised, generalised or molar incisor distribution of periodontitis
    - Site stability
        - Change in Mean probing depth (mm)
        - Percentage of sites changing from unstable to stable
        - Percentage of sites changing from stable to unstable
        - Percentage of sites remaining stable
        - Percentage of sites remaining unstable
    - Number of missing teeth (before and after treatment)
        - Reasons for missing teeth (if known)
- Tooth level:
    - Tooth mobility (0–III) before and after treatment (Miller, 1950)
        - Number and % of non-mobile teeth
        - Number and % of grade I mobile teeth
        - Number and % of grade II mobile teeth
        - Number and % of grade III mobile teeth
        - Mobility changes (Pre vs. Post treatment)
    - Tooth type (molars versus non-molars);
    - Endodontic periodontal lesion present clinically/ radiographically
    - Number extracted during initial NS phase
    - Retention of hopeless teeth
    - Periodontal abscesses
- Site level:
    - Probing pocket depth (PPD) measured at six sites – highest PPD, mean PPD and number of PPD sites >4mm, before and after treatment
    - Bleeding on probing (BOP) measured at 6 sites per tooth (before and after treatment)
    - Horizontal Furcation involvement in molars (Grade I, II, III-(Hamp et al., 1975)) before and after treatment
- Operator level:
    - Grade of operator, i.e. consultant, postgraduate student
    - Instruments used, i.e. Cavitron, hand, both
    - Number of sessions for NSPT- full mouth scaling, full mouth disinfection or quadrant scaling
    - Differences in timing of treatment AM vs PM
    - Time spent during NSPT

Tooth and site level data from the third molars and teeth lost during NSPT were excluded.