Keywords
Orthodontic arch wire, nickel–titanium alloy, Superelasticity; Tensile strength, microbiologically influenced corrosion, oral biofilm, Streptococcus mutans.

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Biomechanical Degradation of Nickel-Titanium Archwires Induced by Oral Streptococcal Biofilms: Effects on Superelastic Behavior and Force Delivery.

Abstract

Background: Nickel-titanium (NiTi) archwire is a fundamental component of fixed orthodontic appliances with superelasticity required for effective tooth movement. After insertion within the oral cavity, streptococcal species produce organic acids and extracellular polysaccharides that accelerate archwire. However, exposure to streptococcal biofilm changes the mechanical properties of NiTi archwires inadequately addressed.

Objective: To assess the effect of oral streptococcal species (Streptococcus mutans, Streptococcus salivarius, and Streptococcus parasanguinis) on the mechanical behavior of (NiTi) orthodontic archwires and to demonstrate the implications of these changes for orthodontic force delivery.

Materials and Methods: Forty-eight superelastic NiTi archwires were allocated to a test group (n = 24), immersed for four weeks in sterile medium inoculated with the three streptococcal species, and a control group (n = 24), immersed in sterile medium alone. Tensile tests to failure and cyclic tensile tests (1, 5, and 10 cycles) were performed at 37 °C on a universal testing machine, and the transformation stresses and moduli of elasticity were derived from the resulting stress–strain curves. Groups were compared using independent t-tests, ($p < 0.05$).

Results: No archwire fractures occurred in either group. The ultimate tensile strength was significantly lower in the test group (991.66 ± 18.37 MPa) than in the control group (1522.00 ± 43.70 MPa; $p < 0.001$). Cyclic testing showed that the test group exhibited significant reductions in austenitic and martensitic moduli of elasticity and the direct and reverse transformation stresses (σMs, σMf, σAs, σAf), along with a significantly greater hysteresis (Δε) ($p < 0.05$).

Conclusion: Exposure to S. mutans, S. salivarius, and S. parasanguinis significantly compromised the load–deflection behavior of NiTi archwires, indicating a reduced capacity to deliver the light continuous forces required for efficient tooth movement. No hydrogen-embrittlement fracture was observed, suggesting that the microbial environment altered archwire without causing failure.

INTRODUCTION

Clinical relevance: Colonization of NiTi archwires by oral streptococci may reduce and destabilize the forces delivered during the alignment phase, a factor clinician should consider when interpreting unexpectedly slow tooth movement and when emphasizing oral hygiene control during fixed-appliance therapy.

Contemporary orthodontic therapy corrects malocclusion and aligns the dentition using a combination of bands, brackets, archwires, and elastics. Among these components, nickel–titanium (NiTi) archwires are the most widely used for initial alignment because of their mechanical properties, including superelasticity, shape memory, wide elastic working range, low stiffness, and high flexibility (1). These properties allow NiTi archwires to deliver light, comparatively constant forces over a wide range of deflections (2).

The oral cavity harbors a complex and diverse microbiota comprising bacteria, viruses, and fungi. Oral bacteria readily adhere to tooth surfaces, brackets, and archwires, forming a structured biofilm (3). Biofilm formation and bacterial metabolism progressively alter the surface characteristics and mechanical behavior of orthodontic materials (4). Because of microbial colonization of the oral cavity, there is a continuing risk of reduced long-term performance of intraoral metal appliances. These processes can drive localized attacks on the alloy, a phenomenon described as microbiologically influenced corrosion (MIC) (5).

A study reported that oral streptococci, including Streptococcus mutans, Streptococcus sanguinis, and Streptococcus salivarius, create an initial biofilm covering the NiTi archwires. These bacterial species ferment dietary carbohydrates to produce lactic acid, lowering the local pH, organic acids, and metabolic by-products that accumulate within biofilms on archwires (4,6). Extracellular polymeric substances further promote bacterial attachment, biofilm maturation, and increase surface roughness (7,8).

Corrosion is an electrochemical process that alters the chemical composition of an alloy and reduces its mass (9). As metal corrosion increases, the durability and functional performance of the material change, with measurable effects on the stress–strain response, force, elastic modulus, and load–deflection behavior (10). These alterations can ultimately influence treatment outcomes and increase the risk of treatment failure (11). Therefore, it is clinically important to evaluate how exposure to streptococcal species affects the mechanical properties of NiTi archwires. Despite comprehensive investigations on the corrosion of orthodontic biomaterials. There is still a gap in the literature regarding how the three species of streptococcal biofilms directly cause the mechanical degradation and localized pitting of NiTi archwires. Therefore, the current in vitro investigation evaluated the tensile strength and modulus of elasticity of superelastic NiTi archwires after four weeks of exposure to three common oral streptococci. The null hypothesis was that exposure to S. mutans, S. salivarius, and S. parasanguinis would produce no statistically significant change in the mechanical properties of NiTi archwires or alter their force-delivery behavior compared with the control group.

MATERIALS AND METHODS

Study design and specimens: This in vitro study was conducted at the Faculty of Dentistry, University of Monastir in Tunisia. According to the G Power analysis, forty-eight superelastic NiTi archwires (3M Unitek shape-memory alloy; composition Ti 50.8–Ni 48.2; round cross-section, 0.36 mm² / 0.014 inch) were used. The specimens were equally allocated to a test group (n = 24), exposed to oral streptococci for four weeks, and a control group (n = 24), maintained in a sterile medium without bacterial exposure. The parameters were adjusted with a significance level (α) of 0.05 and a statistical power of 0.80, a medium effect size of Cohen's d = 0.828, and based on an independent t-test as statistical analysis using SPSS version 31 (IBM Corp., Armonk, NY, USA) for the comparison of mechanical parameters between the experimental and control groups.

Specimen mounting: Each archwire was ligated to a three-dimensionally printed resin model reproducing a Class I malocclusion with symmetrical tooth malposition. Twelve brackets (0.022-inch slot) were bonded to the model using orthodontic composite resin, and arch wires were secured with elastomeric ligatures (Figure 1A–D). All resin models were disinfected and sterilized by autoclaving according to an approved protocol to eliminate residual microorganisms (16).

Bacterial strains and immersion: Blood agar cultures were prepared following established protocols at the Department of Microbiology, Fattouma Bourguiba University Hospital, Monastir, Tunisia, as previously described. Incubation typically lasts 18–24 h at 37°C. Three streptococcal species (Streptococcus mutans, Streptococcus salivarius, and Streptococcus parasanguinis) were selected from culture collections (Figure 2). Streptococcal species identity was confirmed using the VITEK 2 Compact system (bioMérieux) and bacterial concentration was calculated by density of 0.5 McFarland turbidity standard is equivalent to the density of a bacterial suspension containing 1.5 x 10^8 colony-formation units (CFU/ml). The arch wires of the test group were immersed in containers of sterile medium inoculated with the bacterial suspension (experimental solution), whereas those of the control group were immersed in sterile medium alone (Figure 3).

Mechanical testing: After the exposure period, the mechanical properties of the archwires were assessed using a universal testing machine (Lloyd Instruments EZ20). Specimens of 30 mm length were mounted in the grips (Figure 4) and tested at 37 °C to simulate the intraoral temperature. Uniaxial tensile tests to failure were used to determine the ultimate tensile strength and stress at the onset of martensitic transformation. Uniaxial cyclic tensile tests (loading–unloading) were performed and analyzed at the 1st, 5th, and 10th cycles to characterize the superelastic transformation: the austenitic and martensitic moduli of elasticity (EA and EM, respectively), direct transformation stresses (martensite start σMs and finish σMf), reverse transformation stresses

(austenite start σAs and finish σAf), apparent transformation moduli (EA/M and EM/A), stress variation between transformation plateaux (Δσ), strain hysteresis (Δε), and residual strain (εr).

RESULTS

No specimen fractured, dented, or showed permanent deformation during any of the tests in either group. Both the ultimate tensile strength (σMax) and the martensitic transformation start stress (εMax) were significantly lower in the test group than in the control group ($p < 0.001$ for both) (Table 1).

Table 1. Mean ± standard deviation of the ultimate tensile strength and martensitic transformation start stress in the uniaxial tensile test to failure (MPa).

Group	Ultimate strength (σMax)	Martensite transformation start stress (εMax)
Control (C)	1522.00 ± 43.70	499.50 ± 6.20
Test (T)	991.66 ± 18.37	390.00 ± 8.60
P-value	0.000*	0.000*

* Statistically significant ($p < 0.05$).

The austenitic (EA) and martensitic (EM) moduli of elasticity were reduced in the test group compared to the control group, as were the martensite transformation start (σMs) and finish (σMf) stresses. These reductions were statistically significant for most cycles, with the difference in EA reaching significance from the 5th cycle onward (Table 2).

Table 2. Mean ± standard deviation of the moduli of elasticity (EA, EM) and direct martensitic transformation stresses (σMs, σMf) at the 1st, 5th, and 10th cycles (MPa).

Group	EA	EM	σMs	σMf
C1	178.25 ± 2.21	231.30 ± 17.44	457.33 ± 11.71	490.00 ± 7.24
T1	161.50 ± 42.17	186.96 ± 14.29	432.50 ± 8.22	447.50 ± 2.74
P-value	0.37	0.001*	0.002*	0.000*
C5	233.68 ± 26.59	225.34 ± 17.57	411.67 ± 17.47	455.00 ± 13.62
T5	194.12 ± 8.26	179.67 ± 3.01	387.50 ± 16.59	410.00 ± 8.27
P-value	0.006*	0.001*	0.030*	0.000*
C10	233.68 ± 26.50	229.66 ± 14.63	391.67 ± 23.05	435.67 ± 16.88
T10	190.00 ± 7.80	184.89 ± 7.15	380.00 ± 22.62	394.00 ± 20.64
P-value	0.003*	0.000*	0.390	0.003*

C = control; T = test; numerals denote the loading cycles (1, 5, and 10). * Statistically significant ($p < 0.05$).

The austenite transformation starts (σAs) and finish (σAf) stresses decreased in both groups, and the apparent direct (EA/M) and reverse (EM/A) transformation moduli differed significantly between groups at several cycles (Table 3).

Table 3. Mean ± standard deviation of the reverse austenitic transformation stresses (σAs, σAf) and apparent transformation moduli (EA/M, EM/A) at the 1st, 5th, and 10th cycles.

Group	σAs	σAf	EA/M	EM/A
C1	161.67 ± 11.50	109.00 ± 17.13	5.00 ± 1.76	9.11 ± 1.03
T1	124.00 ± 15.34	95.00 ± 21.91	13.75 ± 1.32	2.77 ± 0.00
P-value	0.001*	0.240	0.140	0.001*

C5	137.33 ± 13.37	90.00 ± 24.70	6.58 ± 1.34	7.92 ± 0.82
T5	100.00 ± 15.90	70.00 ± 12.55	3.18 ± 1.35	5.00 ± 0.35
P-value	0.001*	0.100	0.001*	0.001*
C10	127.67 ± 15.92	80.33 ± 18.08	9.06 ± 1.70	9.58 ± 0.82
T10	93.50 ± 20.25	67.50 ± 20.13	2.61 ± 0.63	3.12 ± 0.02
P-value	0.009*	0.270	0.001*	0.002*

C = control; T = test; numerals denote the loading cycles (1, 5, and 10). * Statistically significant ($p < 0.05$).

The stress variation between the transformation stages (Δσ) decreased with cycling. The strain hysteresis (Δε) was significantly greater in the test group than in the control group at all cycles, indicating altered energy dissipation during the loading–unloading cycle. Residual strain (εr) remained negligible at the 1st and 5th cycles and showed no significant between-group differences at the 10th cycle (Table 4).

Table 4. Mean ± standard deviation of the stress variation (Δσ), strain hysteresis (Δε), residual strain (εr), and modulus (E) at the 1st, 5th, and 10th cycles.

Group	Δσ	Δε	Er	E
C1	328.33 ± 8.91	5.77 ± 0.55	0 ± 0	0.70 ± 0.55
T1	322.50 ± 8.22	7.58 ± 0.63	0 ± 0	1.20 ± 1.31
P-value	0.260	0.000*	—	0.410
C5	310.00 ± 14.87	5.23 ± 0.57	0 ± 0	0 ± 0
T5	309.00 ± 1.79	6.83 ± 0.07	0 ± 0	0 ± 0
P-value	0.870	0.000*	—	—
C10	293.33 ± 3.98	5.15 ± 0.49	5.15 ± 0.49	0 ± 0
T10	302.50 ± 1.87	6.78 ± 1.17	1.20 ± 1.31	0 ± 0
P-value	0.000*	0.010*	0.420	—

C = control; T = test; numerals denote the loading cycles (1, 5, and 10). * Statistically significant ($p < 0.05$).

DISCUSSION

Orthodontic arch wires are the main components of fixed-appliance systems, and NiTi alloys are among the most frequently used materials (12). However, when NiTi archwires function intraorally, they are exposed to a hostile electrochemical environment in which ionic, thermal, microbiological, and enzymatic processes interact to biodegrade the metal; clinically observed wire fractures have been attributed principally to fatigue or corrosion (13). Therefore, it is important to evaluate the degree to which patients are exposed to corrosion products from the outset of treatment.

Fixed appliances act as reservoirs for microbial proliferation and colonization, facilitating bacterial adhesion (14). Regions of the metal surface exposed to lower oxygen tension behave as anodes and corrode preferentially, releasing metal ions into saliva; these ions react with bacterial by-products to form more corrosive compounds. As the biofilm thickens and matures, anaerobic conditions develop, further promoting corrosion (15). Only bacteria in direct contact with the metal appreciably influence the process (16), and the extent of corrosion is governed by the bacteria–metal contact area (17). Fermentation of dietary sugars releases acids and carbon dioxide (16), and the resulting low pH accelerates metal ion release (15). The initial biofilm on the NiTi archwire formed by streptococci species, especially Streptococcus mutans, Streptococcus sanguinis, and Streptococcus salivarius, results in a reduced pH in the area by fermenting dietary carbohydrates to produce organic acid and metabolic products (4,6). S. mutans, one of the most prevalent oral

species, exploits dietary sucrose to synthesize extracellular polysaccharides that enhance colonization and produce lactic acid associated with corrosion; reciprocally, the roughened, corroded surface upregulates streptococcal virulence-gene expression, reinforcing adhesion and degradation (18).

Hydrogen embrittlement is the most destructive form of hydrogen-induced degradation that affects NiTi orthodontic archwires (19). During the present testing, a complete absence of arch wire fracture was observed, indicating that hydrogen embrittlement did not occur under these conditions, including samples that completed more than 100 loading and unloading cycles. This outcome, far from being incidental, has significant implications for interpretation. The favorable behavior observed in the current study was the absence of NiTi archwire fracture, which can be explained by a combination of factors. The improved manufacturing quality of the 3M archwires studied, whose particular processing is designed to maximize resistance to corrosion. The strain rate settings experimental test protocol might have been in the range where hydrogen absorption declined below the threshold necessary to cause embrittlement-induced fracture. Therefore, rather than contradicting the previous literature on hydrogen embrittlement, the current findings outline the boundaries of clinical significance. The gradual degradation of mechanical properties without fracture, as demonstrated in this study, represents a more prevalent and possibly more clinically mild form of NiTi wire degradation caused by oral bacteria. Moreover, a cross-sectional study reported that the amount of absorbed hydrogen and sustained stress are important factors in determining whether embrittlement manifests as obvious fractures (20). However, the current results are more aligned with a fractographic study that concluded that masticatory overloading was the most probable cause of clinical failure and that hydrogen embrittlement did not represent the operative mechanism after assessment of clinically sampled NiTi wires that demonstrated a brittle fracture (21). The samples of the current study withstood repeated cyclic loading, indicating that streptococcal organic acid exposure alone, without simultaneous mechanical overloading, failed to produce sufficient oxide layer degradation to cause fracture during the experimental period. This is in agreement with a study showing that the fracture of metallic NiTi alloys under bacterial exposure necessitates the simultaneous interaction of repetitive cyclic fatigue and microbiologically induced corrosion, neither of which alone is sufficient to exceed the fracture threshold (22).

The maximum load that an arch wire can sustain corresponds to the ultimate stress before structural collapse, which is derived from the tensile-to-failure test. The test group showed a significant reduction in ultimate stress relative to the control group, and this difference is expected to widen with longer exposure. Bacteriological environments can promote the corrosion of NiTi alloys and encourage ion release, surface pitting, and passive oxide layer disintegration, thereby degrading mechanical properties, particularly the maximum stress. This outcome agrees with findings that have been widely reported. Pai et al. (2023) documented decreased ultimate tensile strength, reduced elasticity, and worsened performance as a result of structural and chemical changes. The breakdown of the protective titanium oxide layer intensifies the stress and hydrogen absorption processes (23). Yokoyama et al., (2007) who reported that diverse oral conditions affect the tensile strength of orthodontic archwire alloys and may reduce their therapeutic effectiveness (24). Furthermore, Taha et al. (2015) documented that at four and eight weeks of intraoral exposure, biofilm adherence and surface roughness considerably increased. The amount of change increased over time, which was in accordance with the decrease in maximum stress (σMax) revealed in this study (25).

The cyclic test reproduces repeated loadings and unloadings. The observed reduction in transformation stress can be explained by the accumulation of dislocations within the retained austenitic phase, which facilitates martensite nucleation and lowers the external force required for stress-induced martensitic transformation. While cycling is usually expected to increase transformation stresses by generating internal defects and microstructural reconfiguration that raise resistance to phase transformation (24), the present study observed a reduction in these stresses in the bacterially exposed group. The reversible martensitic transformation between the austenitic and martensitic phases, driven by mechanical loading and unloading, underlies the superelastic behavior that makes these wires effective in correcting dental malposition; therefore, its modification has direct clinical relevance.

The constraints of direct and reverse transformation (σMs, σMf, σAs, and σAf) values can be determined from cyclic tensile tests. The current study documented a significant reduction in this variation of constraints. This result appears to be opposite to the findings reported by Yokoyama K, et al., (2007), as exposure duration to hydrogen increases, the ability of hydrogen to prevent the process of transformation increase (24). In fact, it appears as an obstacle to the creation of martensite variations, which increases the significance of the mechanical force required.

The stress for the start of martensitic transformation (σMs) was significantly reduced in the test group. With cycling, the between-group difference in σMs increased, plausibly reflecting a combined effect of mechanical cycling and bacterial action that accelerates degradation; a significant change in the mean transformation stress was also evident. In agreement with this finding, Pastor F. et al. (2022) observed that Ni leakage at the archwire surface gradually raises the martensitic start as chemical exposure time increases, which indicates that one of the main causes of σMs reduction is compositional surface alterations (26). Pandis and Bourauel observed a significant decrease in the martensite transformation start stress after clinical use of superelastic NiTi arch wires (27).

The modulus of elasticity (rigidity) is a key determinant of treatment outcome, as it describes the force required to deflect an arch wire. Low-rigidity wires deliver lighter, more continuous forces during deactivation, optimizing the biological environment for tooth movement and reducing patient discomfort (28). In the present study, the modulus of elasticity differed significantly between

groups across the cyclic tests, with reductions in the exposed group potentially related to trapped interstitial hydrogen and electrochemical reaction between the wire surface and streptococcal organic acids occurred. This affects the resistance of NiTi structure to elastic deformation and destroys its crystallographic regularity. Yokoyama et al. (2007) attributed modulus changes to hydrogen absorption that occurs when the wire is loaded beyond its elastic limit. Comparable losses in superelastic force-delivery capacity have also been reported after clinical use and intraoral ageing (24). The result was in the consistent with Sawan and Gassem (2024) who reported that the hydrogen penetrates grain boundaries and interstitial spaces, resulting in progressive decreases in elastic modulus that were directly related to this hydrogen penetration process rather than only surface morphological changes (10).

A principal therapeutic advantage of superelastic hysteresis is that less force is transmitted to the periodontal structures during deactivation than during activation (29). The present study found significantly greater strain hysteresis in the test group, indicating that bacterial exposure altered the wire's energy-dissipation characteristics. Activation forces exceeded deactivation forces, with no significant between-group difference in residual strain. Sarul et al. reported a statistically significant reduction in deactivation force (30). This was in the agreement with Sapata et al., (2020) who concluded that the NiTi arch wires exposed to an oral environment for four weeks caused measurable decreases in deactivation force. Exposure to the biological oral condition changes the hysteretic mechanical properties of NiTi archwire (31). Gamaoun et al. (2014) examined the susceptibility of NiTi to hydrogen embrittlement in terms of residual strain after a small number of cycles, demonstrating that embrittlement occurs preferentially at low strain rates, attributable to the interaction between diffused hydrogen and the martensitic transformation (32).

LIMITATIONS

In vitro models offer controlled conditions for isolating specific variables but cannot fully reproduce the biological and mechanical complexity of the oral cavity. Static immersion does not replicate the ion-exchange and washing actions of saliva, nor the continuous loading associated with tooth movement. Furthermore, the arch wires tested were new and smooth, whereas intraorally, they are additionally subjected to debris, wear, and plaque. The four-week exposure and fixed bacterial concentration may also underestimate effects that accumulate over a longer clinical treatment period.

CONCLUSION

Within the limitations of this in vitro study, exposure to S. mutans, S. salivarius, and S. parasanguinis significantly compromised the elasticity and load–deflection behavior of NiTi arch wires. No fracture was observed during any test, indicating the absence of hydrogen-embrittlement failure despite cycling. The ultimate tensile strength was significantly lower in the bacterially exposed group, and cyclic testing showed consistent reductions across loading cycles relative to controls. Collectively, these results suggest that the oral microbial environment can degrade the functional performance of NiTi arch wires without causing catastrophic failure.

CLINICAL RECOMMENDATION

Although these findings are clinically relevant, further in vivo research is needed to corroborate the findings. Because the metallic components of orthodontic appliances are inherently susceptible to corrosion, manufacturers, clinicians, and patients should collaborate to minimize it, for example, through rigorous oral hygiene and, where appropriate, the use of corrosion-resistant, antibacterial arch-wire surface treatments now under development.

DECLARATION

Conflict of interest: The authors declare that they have no competing interests.

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ETHICAL APPROVAL: This study was conducted in accordance with the principles of the Declaration of Helsinki. The study protocol was approved by the Research Ethics Committee of the Faculty of Dentistry at the University of Monastir.

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Figures Legends

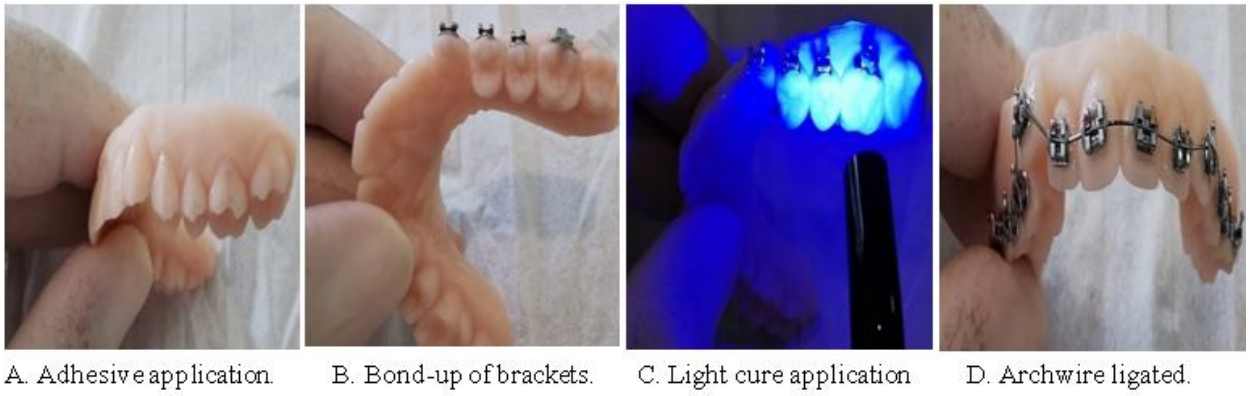

Fig.1. The step of resin model fabrication with ligated arch wires.

Figure 1. Steps of resin-model fabrication and archwire ligation. (A) Application of the adhesive agent; (B) bonding of brackets; (C) light-curing; (D) ligated archwire.

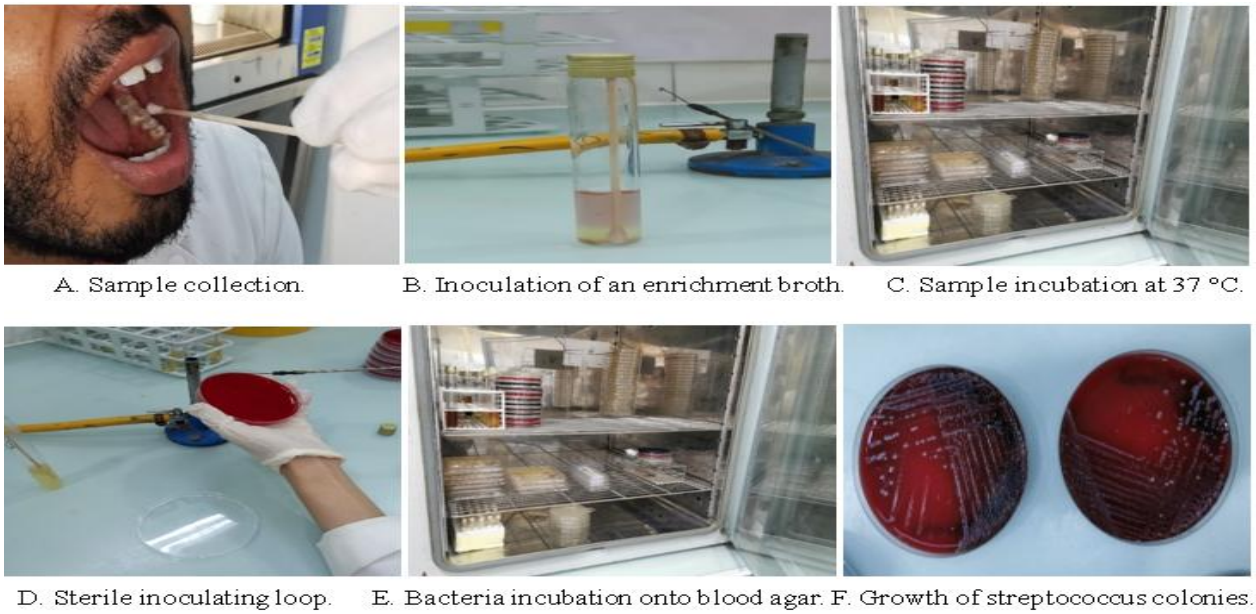

Fig.2. the steps for obtaining streptococcus from an oral cavity swab to a culture within the microbiology lab

Figure 2. Sequence for obtaining streptococci, from an oral-cavity swab to culture within the microbiology laboratory.

A. The VITEK® 2 Compact system. B. Density of 0.5 McFarland. C. bacterial suspension in a tube. D. Heart brain media with a density of 0.5 McFarland.

E. Two sterile containers. F. Containers with media. G. Containers with resin models. H. Resin models in media only as a control group and resin models in media with bacteria as a test group.

Fig. 3. Illustrated the method of identification and analysis of strain of streptococcus, as well as arch wires were immersed in containers with experimental solution as test group and in a container with sterile media only as control group.

Figure 3. Identification and analysis of the streptococcal strains, and immersion of the arch wires in the experimental solution (test group) and in medium only (control group).

Fig. 4. A. Tensile machine (Lloyd Instrument EZ 20). B. Fit the arch wire into the grips. C. Adjustment of the test setting. D. Stress-strain curve of the NiTi arch wire.

Figure 4. Mechanical testing. (A) Universal testing machine (Lloyd Instruments EZ20); (B) the arch wire fitted into the grips; (C) adjustment of the test settings; (D) representative stress–strain curve of the NiTi arch wire.