pH of pus collected from periapical abscesses

M. H. Nekoofar1,2,3, M. S. Namazikhah3,4, M. S. Sheykhr-ezae1,3, M. M. Mohammadi3, A. Kazemi3, Z. Aseeley2 & P. M. H. Dummer2

1Department of Endodontics, Faculty of Dentistry, Tehran University of Medical Sciences, Tehran, Iran; 2Endodontology Research Group, School of Dentistry, Cardiff University, Cardiff, UK; 3Dental Research Center, Tehran University of Medical Sciences, Tehran, Iran; and 4Private Practice, Beverly Hills, CA, USA

Abstract


Aim To determine the pH of pus collected from periapical abscesses.

Methodology Forty patients (Male = 17/Female = 23) between the ages 17 and 37 years, each with a periapical abscess and with no relevant medical history, were recruited. All the participants had moderate-to-severe pain on percussion accompanied by localized or generalized swelling. At least 1 mL of pus was aspirated from each participant using a No 20 gauge needle. A pH meter was used to define the pH of the pus immediately following aspiration.

Result The mean pH of pus from the periapical abscesses of patients was 6.68 ± 0.324 with a range between 6.0 and 7.3. There was no statistically significant difference in pH by gender or age.

Conclusion The mean pH of pus from periapical abscesses was generally acidic, but some samples (two female and three male) were neutral and some samples (four female and one male) were alkaline.

Keywords: periapical abscess, pH.

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Introduction

The concept of pH was first introduced by Sörenson (1909) and defined as the hydrogen ion concentration in a solution. Theoretically, it is defined by a logarithmic relationship: pH = –log [H+]. The logarithmic scale reduces the extremely wide variations in hydrogen ion concentrations to a narrow range of pH from 0 to 14, which led to the establishment of the pH scale. According to the definition of pH, a small change in pH leads to an immense change in hydrogen concentration. Thus, a decrease in one unit of pH is equivalent to a 10-fold increase in the hydrogen ion concentration (Hampel et al. 1991). The pH of blood plasma and interstitial fluid is 7.4 (Moi et al. 1990, Cicha et al. 2003), whilst saliva has a pH ranging from 6.4 to 7 (Mandel 1974). Living organisms can tolerate only minor alterations in pH (Wray 1988). In humans, the normal pH in different organs varies, but a change in blood pH below 6.8 or above 7.4 can be fatal (Kellum 2000).

During acute inflammation, arterial dilation results in an increased blood flow to the site of injury. This process leads to increased vascular permeability and collection of protein-rich, extracellular fluid. This infiltrate contains leucocytes, predominantly neutrophils, which migrate along with phagocytic agents to the injured tissue (Henson & Johnston 1987). Jensen & Bainton (1973) reported that the pH of neutrophils and monocyte phagosomes dropped to 3.5–4.5 within 7–15 min of ingestion of microorganisms. This phenomenon initiates acid hydrolysis and the degradation of dead microorganisms (Geisow 1981, Mayer et al. 1989). At the same time, polymorphonuclear leucocytes (PMN) that exhibit signs of cell injury and/or die release their acidic contents into the extracellular environment. Dead microorganisms and particles along with dead neutrophils may subsequently form pus that can be defined
as a thick opaque, usually yellowish to green liquid that is formed by exudate, leucocytes, various bacterial species, dead cells and tissue debris (Dorland 1994). It also contains various bacterial species, most of which are anaerobic (Siqueira et al. 2001).

The hydrogen ion concentration in blood plasma and various body solutions is amongst the most intensively regulated variables in human physiology. At the intercellular membrane level, the regulation of pH is essential to control the metabolism and fluxes of the ionized forms of weak acids and bases (Schwiening & Willoughby 2000). The long-term stability of blood pH is achieved by removing the acid as rapidly as possible (Clancy & McVicar 2007a).

Within clinical dentistry, the pH of tissues has a number of implications. The pH of local anaesthetic solution and the tissues into which it is injected has an effect on its nerve blocking action (Malmed 2004). Acidification of the tissues as a result of inflammatory products is believed to decrease the effectiveness of local anaesthetics (Tsuchiya et al. 2007). The increased hydrogen ion concentration results in a greater proportion of the anaesthetic agent existing in its cationic form, rather than in its basic form. This shift in equilibrium means that the anaesthetic may be less able to diffuse through infected tissues than through normal tissues, leading to delayed onset and lowered intensity of anaesthesia (Ondrias et al. 1987, Malmed 2004).

Furthermore, a low pH environment may affect dental materials (Roy et al. 2001, Francisconi et al. 2008). For example, Silva et al. (2007) reported in a laboratory study that an acidic environment altered the surface characteristics and microhardness of glass ionomer cements. Torabinejad et al. (1995) suggested that an acidic pH may impede the setting process of Mineral Trioxide Aggregate (MTA). Thus, variations in the pH of host tissues, because of pre-existing disease at the time of MTA placement, could affect its physical and chemical properties (Lee et al. 2004, Nameakhhah et al. 2008).

Various investigations have suggested that the pH of an infected tissue and of pus is likely to be lower than that of healthy tissue (Malmed 2004, Tsuchiya et al. 2007). However, surprisingly, there is limited clinical information on the pH of pus. The aim of this study was to determine the pH of pus drained from periapical abscesses in humans.

**Materials and methods**

Patients were recruited from the Oral and Maxillofacial Surgery clinic at the Shariati hospital in Tehran, Iran. All documentation and procedures were approved by the ethical board of the local research review committee, in the Faculty of Dentistry, Tehran University of Medical Sciences, Iran. Subjects who volunteered were given a description of the project and informed consent was obtained. Forty patients (17 male and 23 female) with an acute periapical abscess and associated swelling were included. All participants had moderate-to-severe pain on percussion accompanied by localized intraoral or generalized facial swelling. They were in otherwise good general health, which was assessed in a review of their medical history.

To minimize observer bias, only one clinician examined the patients. Once the diagnosis of an acute abscess was made, at least 1 mL of pus was aspirated using a sterile syringe with a No 20 gauge needle from each subject under strict infection control procedures. To eliminate any confounding effect, local anaesthetic solution was not used prior to aspiration. If the pus sample was <1 mL or was contaminated with blood, it was excluded from the study. An Ultra Basic Portable pH meter (Denver Instrument, Denver, CO, USA) was immediately used to measure the pH. The pH meter electrode was inserted into the container that carried the pus sample and a value for pH was obtained. After every measurement, the electrode was placed in the neutralizing solution supplied by the manufacturer to calibrate it to the pH of 7. Independent t-tests and Spearman’s rank correlation coefficients were employed to investigate the relationship between pH and age and gender.

**Results**

The results are summarized in Table 1. Overall, 40 participants (17 male and 23 female) were included. The mean pH measurement was 6.68 (SD = 0.324). The pH ranged from 6 to 7.3. The mean pH in male patients was 6.7 (SD 0.382), whilst in female patients.
it was 6.66 (SD = 0.358); gender did not have a significant effect on pH ($P = 0.750$). The age of participants ranged between 17 and 35 years; the mean age was 26.22 (SD = 4.26). There was no statistically significant correlation between age and pH.

**Discussion**

In 1893, during his studies on phagocytosis, Metchnikoff revealed that the pH inside phagocytes was acidic. He concluded that this acidic environment within the cell could result in the destruction of ingested organisms (Gourko et al. 2000). During phagocytosis, polymorphonuclear (PMN) leucocytes degranulate and release enzymes, which are activated in an acidic pH. In a laboratory study, Jensen & Bainton (1973) investigated pH changes during phagocytosis and demonstrated that the pH of a phagosome was reduced to approximately 6.5 within 3–4 min after initiation of phagocytosis. They reported that after 7–15 min, the pH dropped to 3.5–4.5 and remained within this range for up to 2 h. As the pH falls, leucocytes may exhibit cell injury and consequently die, a phenomenon that results in pus formation. Therefore, it is generally believed that inflamed and/or infected tissues have a low pH (Malmel 2004, Tsuchiya et al. 2007). Accordingly, the presence of certain acids such as isobutyric, butyric, isovaleric, valeric, isocaproic and caproic acid can be identified by gas chromatography (Tanaka et al. 1990) and act as indicators of anaerobic bacterial infection (Kalowski et al. 1992). In addition, the production of lactic and acetic acid, as a result of glucose metabolism by bacteria, may decrease pH levels (Hall et al. 2005). However, acids outside the phagosomes are quickly neutralized by the physiological regulatory mechanisms. In an interventional study, McCormick et al. (1983) introduced *Streptococcus faecalis* into 24 immature premolar teeth of six young beagle dogs. The initial pH range of the developing periapical lesions fell from 7.1–7.2 to 6.2–6.6 respectively. Furthermore, they reported that the pH of the periapical lesions changed with time back to 7.0–7.2 irrespective of the type of treatment provided. Wiese et al. (1999) studied the pathophysiology of odontogenic abscesses and identified the pH value of pus to be 6.164 ± 0.233, which is in accordance with that reported by McCormick et al. (1983) and the findings of the present study. Indeed, in the present study, the mean pH of pus aspirated from periapical abscesses was 6.68 ± 0.324 suggesting that the physiological regulatory mechanisms are active in tissues to normalize the blood and tissue pH in health and disease. These complex physiological regulatory mechanisms can be categorized as short-term, intermediate and long-term homeostasis (Clancy & McVicar 2007a,b). The intermediate and long-term stability of the pH of blood is achieved by removing the acid as rapidly as possible through the respiratory and renal systems respectively (Clancy & McVicar 2007a). The short-term regulation of acid seems to be more complex (Meeseter & Siesjo 1971).

When the internal body is exposed to a substantial amount of acid, several rapid mechanisms become active to uptake the hydrogen ions such as changes in carbon dioxide tension, relative electrolyte concentrations and total weak acid concentration. In addition to this, physicochemical buffering, cellular consumption of nonvolatile acids and transfer of acid or alkali between the cytosol and organelles act as the short-term regulatory mechanisms (Meeseter & Siesjo 1971, Kellum 2000, 2005, Clancy & McVicar 2007b).

Acidic pH may have adverse effects on providing adequate local anaesthesia (Wong & Jacobsen 1992, Malmel 2004). However, there are a number of other reasons that can explain the failure of local anaesthetic including anatomical, pharmacological, psychological and pathological factors. Acidic pH as a result of pathological nature may decrease the concentration of the unionized fraction of local anaesthetic, that is, the lipophilic proportion of the local anaesthetic that diffuses through the nerve sheaths (Meechan 1999). The efficacy of local anaesthetic solutions depends largely on the concentration of this lipophilic fraction (Ondrias et al. 1987). The potential acidic pH of periapical abscesses cannot always explain the failure of local anaesthesia (Wong & Jacobsen 1992, Meechan 1999). Indeed, the results of the present study revealed that the pH of aspirated pus from periapical abscesses was not always acidic, but had a range between 6 and 7.3 (mean = 6.68, SD = 0.3345). In addition, during a regional nerve block, local anaesthetic solutions may be deposited at a site distant from inflammation (Madan et al. 2002). In this situation, nerve hyperalgesia and/or an increased blood supply to the inflamed area may explain the anaesthetic failure (Meechan 1999). Utilizing a combination of regional nerve block and local infiltration injections and/or the administration of supplementary techniques such as intraosseous and intraligamentary injections may overcome the failure of local anaesthesia (Madan et al. 2002, Meechan 2002, Malmel 2004). The use of a higher
concentration of local anaesthetic is also suggested (Meechan 1999).

Acidic pH may also have an effect on the properties of dental materials, which are routinely placed in environments that may be inflamed or infected. In a laboratory study, Namazikhah et al. (2008) showed that detrimental effects on surface microhardness of Mineral Trioxide Aggregate occurred at pH 4.4. The mean Vickers surface microhardness value at this pH was 14.34 compared to 53.19 observed when the material was exposed to pH 7.4. At pH 6.4, the mean Vickers surface microhardness value was 40.73. On the basis of their findings and the results of the present study, the mean pH of pus (6.68) would pose minimal effects on surface microhardness of MTA.

Lee et al. (2004) compared the surface microhardness and hydration behaviour of MTA samples under various physiological environments. They found that samples hydrated at a pH of 5, had surface microhardness values that were significantly lower compared with distilled water, normal saline and a solution buffered at pH 7. In addition, they showed that the mean microhardness of the group hydrated at pH 7 was significantly greater than that hydrated in distilled water, suggesting the beneficial effect of this pH. Roy et al. (2001) evaluated the effect of acidic pH on the microleakage of various root-end filling materials including amalgam, Geristore, Super-EBA, MTA, Calcium Phosphate Cement (CPC) or MTA with CPC matrix. They reported that at pH 5, no statistically significant difference existed between the materials except for amalgam. Various concentration and types of acid may have dissimilar effects on the physical and chemical characteristics of Portland cements (Taylor 1997) and as MTA is a Portland cement-like material (Asgary et al. 2006), it might also be affected. Singh et al. (1986) revealed that lactic acid accelerated the hydration of Portland cement by increasing the crystalline character of calcium hydroxide resulting in advanced growth of the hydration products. In contrast, Rai et al. (2006) reported that in the presence of tartaric acid, the silicate hydration-phase of Portland cement was retarded strongly. In the present study, the pH of pus as an indicator of acid concentration was measured. Further analysis of pus and/or inflamed tissue to determine presence and concentrations of various acids and the evaluation of the effect of different types of acid on various dental materials is suggested.

Silva et al. (2007) evaluated in a laboratory study, the effect of acidic pH (6.2–4.3) on fluoride release and the surface microhardness of two types of polyacid-modified resin composites and three types of glass ionomer cements; surface characteristics were also observed. They reported that fluoride release was increased as a result of a lower acidic pH on both materials, but there was no statistically significant effect of an acidic pH on the surface microhardness of polyacid-modified resin composites. However, at low pH values, a significant change in the surface microhardness of glass ionomer cements was observed. According to the results of the present study that showed the pH range of aspirated pus from periapical abscesses to be between 6.00 and 7.3, it can be concluded that the pH of pus in periapical abscesses would have only minimal effects on polyacid-modified resin composites and glass ionomer cements.

Conclusion

The pH of pus aspirated from periapical abscesses was acidic (6.68 ± 0.324). There was no statistically significant association between pH and age or gender. Further analysis of pus to determine the presence and concentrations of various acids is suggested.

References


