Local Anesthetics as Antimicrobial Agents: A Review

SVENA M. JOHNSON, BARBARA E. SAINT JOHN, and ALAN P. DINE

ABSTRACT

Background: Since the introduction of cocaine in 1884, local anesthetics have been used as a mainstay of pain management. However, numerous studies over the past several decades have elucidated the supplemental role of local anesthetics as antimicrobial agents. In addition to their anesthetic properties, medications such as bupivacaine and lidocaine have been shown to exhibit bacteriostatic, bactericidal, fungistatic, and fungicidal properties against a wide spectrum of microorganisms.

Methods: A comprehensive literature search was conducted using MEDLINE 1950—present for in vitro and in vivo studies pertaining to the antimicrobial activity of various local anesthetics on a broad range of bacterial and fungal pathogens. Studies testing the effect on microbial growth inhibition of local anesthetics alone and in combination with other agents, such as preservatives and other medications, as well as the effect of conditions such as concentration and temperature, were included for review. Outcome measures included colony counts, area-under-the-curve and time-kill curve calculations, minimum inhibitory concentrations, and post-antibiotic effect.

Results: Evidence suggests that local anesthetics as a class possess inherent antimicrobial properties against a wide spectrum of human pathogens. Multiple local anesthetics at concentrations typically used in the clinical setting (e.g., bupivacaine 0.125%–0.75%; lidocaine 1%–3%) inhibit the growth of numerous bacteria and fungi under various conditions. Different local anesthetics showed various degrees of antimicrobial capacity; bupivacaine and lidocaine, for example, inhibit growth to a significantly greater extent than does ropivacaine. Greater concentrations, longer exposure, and higher temperature each correlate with a proportional increase in microbial growth inhibition. Addition of other agents to the anesthetic solutions, such as preservatives, opioids, or intravenous anesthetics such as propofol, modify the antimicrobial activity via either synergistic or antagonistic action. Limited studies attribute the mechanism of action of antimicrobial activity of local anesthetics to a disruption of microbial cell membrane permeability, leading to leakage of cellular components and subsequent cell lysis.

Conclusions: Local anesthetics not only serve as agents for pain control, but possess antimicrobial activity as well. In such a capacity, local anesthetics can be considered as an adjunct to traditional antimicrobial use in the clinical or laboratory setting. Additionally, caution should be exercised when administering local anesthetics prior to diagnostic procedures in which culture specimens are to be obtained, as the antimicrobial activity of the local anesthetic could lead to false-negative results or suboptimal culture yields.
Many factors influence the development of postoperative surgical site infections, such as anesthetic regimens, antimicrobial use, and perioperative fluid management. The connection between the relief of pain and infections of surgical incisions remains unclear. Unrelieved pain increases vasoconstriction in the periphery, leading to a reduction of perfusion and oxygenation of the tissue surrounding the incision. This decrease in tissue oxygenation may increase the risk of surgical site infections.

Tissue perfusion delivers oxygen, inflammatory cells, growth factors, cytokines, and nutritional components to injured tissues. Hypoperfused regions become hypoxic, with tissue oxygen tensions that do not support adequate oxidative killing or scar formation. In a hypoxic environment, wound healing is arrested by decreased fibroblast proliferation, collagen production, and capillary angiogenesis. Hypoxia also facilitates growth of anaerobic organisms, further complicating wound healing and increasing the risk of infection. A significant increase in tissue oxygenation of the hypoperfused infected wound influences the rate of collagen deposition, angiogenesis, and bacterial clearance in wounds [1].

In response to tissue trauma, neutrophils, lymphocytes, macrophages, and fibroblasts migrate to the site of injury [2]. Hypoxia, which is present to some degree in all wounds, impairs the function of these cells. These conditions may interfere with host defenses and collagen deposition, particularly fibroblast function. Many studies, both in vitro and in vivo, show that collagen deposition is proportional to the partial pressure of oxygen over the range observed in wounds and that the rate of surgical site infection is linked closely to oxygen tension [3,4]. Furthermore, in vitro studies by Hohn et al. [5] and Mandell [6] demonstrated that hypoxia suppresses the killing of Staphylococcus aureus by wound leukocytes. Consequently, higher infection rates in surgical incisions may result from the impaired killing of bacterial contaminants by leukocytes in hypoxic or ischemic tissue. The combination of decreased vascular supply and increased cellularity results in a hypoxic environment within the incision.

The role of local anesthetics in local tissue oxygenation and perfusion is in part attributable to the inherent vasodilatory properties of the agents [7]. One of the properties of local anesthetics that determines their level of analgesic activity is their effect on blood vessels in areas where they are injected. Local anesthetics, with the exception of cocaine and ropivacaine, cause peripheral vasodilation by direct relaxation of vascular smooth muscle, which serves not only to enhance vascular absorption of the local anesthetic, but also to ensure delivery of oxygen and other nutritional components to the tissues. The blood concentrations, duration of action, and the proportion of vessel dilation associated with each agent modulate the systemic effects.

LOCAL ANESTHETICS AS ANTIMICROBIAL AGENTS: A REVIEW

The results of a multitude of in vitro and in vivo studies over the past several decades have substantiated a supplemental role of local anesthetics in the potential prevention and treatment of surgical site infections. In 1976, James et al. [8] examined the effect of bupivacaine on bacterial growth, in addition to the incidence of contamination of catheters and syringes used during epidural analgesia. Syringes in 5/101 cases were found to be contaminated with skin commensal organisms (i.e., Staphylococcus epidermidis), likely originating from the personnel administering the epidurals; catheter tips were not contaminated. In this study, bupivacaine (0.25%) proved bactericidal to both S. epidermidis and Corynebacterium spp. at 37°C, but not at room temperature (Table 1).

Further evidence of the antimicrobial effect of local anesthetics was presented by Rosenberg et al. [9]. Those authors reported that high clinical concentrations (≥ 0.25%) of the local anesthetic bupivacaine inhibited the growth of multiple bacterial and fungal organisms, namely Escherichia coli, S. aureus, S. epidermidis, Streptococcus pneumoniae, S. pyogenes, Enterococcus faecalis, Bacillus cereus, and Candida albicans. With an agar dilution method, bupivacaine was found to have antimicrobial activity against nine of the ten microbial strains tested, suggesting a protective effect against bacterial
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MRSA = methicillin resistant *Staphylococcus aureus*; MSSA = methicillin susceptible *S. aureus*. 
and fungal infections; only *Pseudomonas aeruginosa* showed no inhibition of growth at bupivacaine concentrations as high as 5 mg/mL (0.5%). Morphine 0.2 and 2 mg/mL failed to inhibit the growth of any of the ten strains.

Hodson et al. [10] compared the antibacterial activity of the isomers bupivacaine and levobupivacaine against *S. epidermidis*, *S. aureus*, and *E. faecalis*, and found the minimum bactericidal concentration of bupivacaine to be lower than that of levobupivacaine (0.25% vs. 0.5%, respectively). Racemic bupivacaine therefore appears to have more potent antimicrobial activity than its isomer levobupivacaine.

Noda et al. [11] performed quantitative analysis of the antibacterial activity of local anesthetics by calculating their minimum inhibitory concentration (MIC), killing curves, and post-antibiotic effect (PAE). Colonies of *S. aureus*, *S. epidermidis*, and *P. aeruginosa* were used in the study. At standard clinical concentrations, both bupivacaine and lidocaine had bactericidal activity against the aforementioned species. A comparison of MIC values indicated that bupivacaine has greater antibacterial activity than lidocaine. At equal concentrations, even greater antibacterial activity was found when preservatives were added to the anesthetics, as is common in commercial solutions. The preservatives alone, however, were only weakly bacteriostatic and not bactericidal, merely enhancing the bactericidal activity of the pure anesthetic solutions. Similarly, Grimmond and Brownridge [12] showed increasing microbial inhibition with increasing concentrations of bupivacaine and pethidine (meperidine) using an agar dilution method. At clinical concentrations, bupivacaine inhibited eight of ten pathogens tested, and pethidine inhibited six, confirming the antimicrobial potential of local anesthetics.

In addition to examining the antimicrobial capacity of particular local anesthetics, Sakuragi et al. [13] analyzed the rate of onset of bacterial growth inhibition. Bupivacaine (0.125%, 0.25%, and 0.5%), mepivacaine (2.0%), lidocaine (2.0%), and lidocaine (2.0%) with preservatives were each tested with two strains of methicillin-resistant *S. aureus* (MRSA) for 1, 3, 6, 12, and 24 h at room temperature and cultured subsequently on agar. The authors found that the greater the exposure time, the greater the growth inhibition, corresponding to lower colony counts. Bupivacaine (0.5%) showed the greatest antimicrobial activity, likely bactericidal, inhibiting growth by more than 99% at 24 h, 70% at 6 h, and 60% at 3 h. Colony counts were highest using 0.125% bupivacaine and 2.0% mepivacaine.

In a follow-up study, Sakuragi et al. [14] examined the bactericidal activity of preservative-free bupivacaine (0.125%, 0.25%, 0.5%, and 0.75%) for two strains of MRSA, two strains of methicillin-susceptible *S. aureus* (MSSA), *S. epidermidis*, and *E. coli*. The pathogens were exposed to the bupivacaine for 1, 3, 6, 12, and 24 h at 37°C and room temperature. The results showed both temperature- and concentration-dependent bactericidal activity. Increasing concentrations of bupivacaine correlated with lower colony counts. Likewise, increasing temperatures from room temperature to 37°C increased the growth inhibition of the *S. aureus* strains from 81% to 96% at 24 h with 0.5% bupivacaine, and 22% to 34% at 1 h. No *E. coli* or *S. epidermidis* growth occurred at all after 24 h at 37°C; in fact, *E. coli* growth was inhibited at 12 h. Thus, *S. epidermidis* and *E. coli* proved more sensitive than *S. aureus* to the bactericidal activity of bupivacaine.

In an earlier complementary study in 1997, Sakuragi et al. [15] used the same parameters, yet examined the antimicrobial effect of preservatives (methyl para-oxybenzoate and propyl para-aminobenzoate) alone and when added to 0.5% bupivacaine. Preservatives alone showed significantly lower bactericidal activity than when combined with bupivacaine. As in the previous study, increasing the temperature from room temperature to body temperature increased the growth inhibition of *S. aureus* from 89.6% to 99.8% at 12 h and from 24% to 74% at 1 h using 0.5% bupivacaine with preservatives. Again, *S. aureus* was found to be more resistant to the bactericidal activity of bupivacaine than *S. epidermidis* and *E. coli*.

Aydin et al. [16] examined the antimicrobial activity of the local anesthetics ropivacaine, bupivacaine, lidocaine, and prilocaine on various pathogens, namely *E. coli*, *S. aureus*, *P. aeruginosa*, and *C. albicans*. Of the four drugs tested, lidocaine and prilocaine had the most potent antimicrobial activity, both inhibiting all growth of all pathogens tested at anesthetic concentrations.
concentrations of 2%; at a concentration of 1%, prilocaine inhibited the growth of *E. coli*, *S. aureus*, and *P. aeruginosa*, whereas 1% lidocaine inhibited only *P. aeruginosa*. Bupivacaine was found to inhibit only *P. aeruginosa* at ≥ 0.25% concentrations, whereas ropivacaine failed to inhibit the growth of any pathogens. Pere et al. [17] also found ropivacaine to have less antibacterial effect than bupivacaine, as did Rodrigues et al. [18], who conducted a study using *C. albicans*. In the case of *C. albicans*, Rodrigues et al. suggested that the local anesthetics inhibited fungal germ tube formation secondary to a blockade of ionic channels. Batai et al. [19] found that ropivacaine 2 mg/mL supported the growth of *E. coli*, whereas a higher concentration (10 mg/mL) killed both *E. coli* and *S. aureus*.

Pina-Vaz et al. [20] evaluated the antifungal activity of benzylamine, lidocaine, and bupivacaine against 20 *Candida* strains, including *C. albicans*. The activity of the three drugs was analyzed by viability counts under epifluorescence microscopy. The antifungal activity progressed from fungistatic at lower concentrations, secondary to yeast metabolic impairment, to fungicidal at higher concentrations, secondary to cytoplasmic membrane damage, as evidenced by staining.

Sporicidal activity of local anesthetics and their preservatives was tested by Abdelaziz and el-Nakeeb [21]. The local anesthetics procaine, lignocaine (lidocaine), amylocaine, cincochaine, and amethocaine, all at a 1% concentration, as well as the preservatives cetrimide, chlorocresol, chlorhexidine, phenoxyethanol, and phenylmercuric nitrate were tested alone and in binary combinations to assess their effects on the growth of *Bacillus subtilis* and *Aspergillus niger* spores at various temperatures. Inhibition of growth proved to be temperature-dependent for all agents. Amethocaine was sporicidal (99% death) against *A. niger* at the lowest temperature (30°C), followed by amylocaine and cincochaine (45°C), lignocaine (48°C), and procaine (50°C), compared with 58°C for the saline control. Higher temperatures were required to elicit sporicidal activity against *B. subtilis*. Cincochaine proved sporicidal at the lowest temperature (60°C), followed by amylocaine and amethocaine (84°C and 90°C, respectively). Procaine, lignocaine, and the saline control required temperatures > 100°C to kill 99% of the *B. subtilis* spores. Among the preservatives, chlorocresol/local anesthetic combinations exhibited the highest sporicidal activity.

**INTERACTIONS OF LOCAL ANESTHETICS WITH PROPOFOL AND OPIOIDS**

Propofol, an agent commonly used during operative anesthesia in an emulsion formulation, promotes the rapid growth of microorganisms and has been implicated as a source of postoperative sepsis and surgical site infection. Local anesthetics, particularly lidocaine, often are added to the solution to minimize pain on intravenous injection. Several authors have investigated whether this addition of local anesthetic confers microbial growth inhibition. Gajrag et al. [22] examined the antimicrobial effect of lidocaine in the presence of propofol on cultures of *E. coli* and other pathogens. The investigators found that lidocaine–propofol mixtures inhibited bacterial growth significantly, whereas propofol alone increased the growth rate. Increasing concentrations of lidocaine led to a proportional increase of bacterial growth inhibition. Such results suggest that lidocaine may help to prevent surgical infection even in cases where extrinsic propofol contamination has occurred. Likewise, Sakuragi et al. [23] found colony counts of *E. coli* to be significantly lower after exposure to either lidocaine (1%, 2%, or 4%) or lidocaine (0.25%-4%)–propofol mixtures, leading to the conclusion that lidocaine confers bacteriostatic activity when added to extrinsically contaminated solutions of propofol.

Wachowski et al. [24] arrived at the opposite conclusion. Those authors used parameters similar to those in the previous studies, comparing the growth of four microorganisms (*E. coli*, *P. aeruginosa*, *S. aureus*, and *C. albicans*) in solutions of propofol, lidocaine, and propofol + lidocaine at 20°C. However, the concentrations of lidocaine used in this study were considerably lower than those tested by Sakuragi et al. [23]. Wachowski et al. [24] found that the addition of 0.2% and 0.5% lidocaine to propofol failed to inhibit the growth of the
aforementioned pathogens and concluded that clinically relevant concentrations of lidocaine did not exhibit antimicrobial properties when added to contaminated propofol. In a letter to the editor, Driver [25] described such a claim as misleading. Driver argued that the conditions maintained in Wachowski’s study, such as temperature, pH, and drug concentration, were either suboptimal or unspecified. In their own study, Driver et al. [26] did in fact achieve results that supported bacterial growth inhibition using a mixture of propofol/lidocaine. Aliquots of *S. aureus* diluted to a 1:10⁸ ratio were incubated at 37°C and transferred to solutions containing either lidocaine, propofol, or a mixture of the two. Colony counts were lowest in the mixture and highest in the propofol solution alone. Such results suggest a synergistic antimicrobial action achieved with the combination of lidocaine and propofol that exceeds that of either of the two agents alone. Driver proposed activation of the lidocaine driven by the higher pH when the two agents are combined.

Another local anesthetic, lignocaine, was tested by Ozer et al. [27] to assess its effect on bacterial growth in contaminated propofol emulsions. Cultures of *E. coli*, *S. aureus*, *S. epidermidis*, and *P. aeruginosa* were incubated at 37°C and added to either propofol alone or a propofol/lignocaine mixture (0.1%–2.0%). A significant decrease in colony-forming units (CFU) numbers was seen with *E. coli* in mixtures of 1% and 2% lignocaine. With the three other pathogens, only 2% lignocaine significantly suppressed colony counts. As the recommended clinical doses of lignocaine are reported to be 0.05%–0.1%, the authors concluded that this particular local anesthetic exhibits inadequate antimicrobial activity to prevent infection in a clinical setting.

The addition of other agents, namely opioids, to local anesthetic solutions was tested by investigators including Feldman et al. [28]. Various bacteria were cultured in agar media preparations containing clinical concentrations of lidocaine, bupivacaine, fentanyl, or sufentanil and in mixtures of bupivacaine with each of the two opioids. Reinforcing the findings of Rosenberg et al. [9], both lidocaine and bupivacaine were found to inhibit bacterial growth significantly, whereas the opioids failed to inhibit growth. The degree of growth inhibition was directly proportional to the concentration of local anesthetic; decreasing concentrations of the local anesthetic yielded a significant reduction in bacterial growth inhibition, particularly for certain species such as *S. aureus*.

In 2003, Kampe et al. [29] studied the effect of ropivacaine (0.1%) when mixed with sufentanil (1 mcg/mL) on the growth of the pathogens *S. aureus* and *P. aeruginosa* at room temperature. The combination of the local anesthetic and the opioid inhibited growth of *P. aeruginosa* significantly; multiplication of *S. aureus* was slowed as well.

Tamanai-Shacoori et al. [30] extended previous study to the local anesthetics ropivacaine and bupivacaine in 2004. The effect of ropivacaine (1.2 mg/mL), bupivacaine (0.77 mg/mL), sufentanil (0.38 and 0.5 mcg/mL), and combinations of sufentanil and the two local anesthetics on the growth of *E. coli*, *S. aureus*, and *E. faecalis* at 37°C was investigated. Both bupivacaine and ropivacaine alone were found to inhibit growth of *E. coli* and *S. aureus*, yet both were ineffective against *E. faecalis*. The addition of sufentanil to each of the two local anesthetics had opposing effects, modifying the antimicrobial activity of each drug. When combined with bupivacaine, sufentanil exhibited a synergistic effect, increasing the inhibitory effect on the growth of all three pathogens. When added to ropivacaine, however, the antibacterial activity of the mixture was lower than that of ropivacaine alone, thereby exerting an antagonistic effect.

**EFFECTS OF LOCAL ANESTHETICS ON THE YIELD OF BACTERIAL CULTURES**

Because of this antimicrobial activity, several studies have focused on the potential of local anesthetics to interfere with clinical diagnostic cultures and lead to false-negative results. With the sensitivity of bronchoalveolar fluid (BAL) cultures as low as 50%–60% for the diagnosis of pneumonia, Anding et al. [31] investigated the antimicrobial activity of local anesthetics used in the procedure as a possible explanation for the low sensitivity found with bronchos-
copy. The bactericidal potential of various concentrations (0.01%–1%) of the local anesthetic oxybuprocaine was tested against 10^4/mL inocula of *S. pneumoniae*, *Haemophilus influenzae*, *P. aeruginosa*, and *E. coli*. Time–kill curves revealed significant bactericidal activity against *S. pneumoniae* and *H. influenzae* with even the lowest concentration of oxybuprocaine (0.01%). Oxybuprocaine 1% inhibited the growth of *E. coli* and *P. aeruginosa*. If local anesthetics such as oxybuprocaine are used prior to obtaining material for culture, false-negative results may ensue.

Olsen et al. [32] investigated the effect of adding lidocaine to suspensions of BAL fluid contaminated with clinical respiratory isolates. There was significant inhibition of the growth of two of the four *S. pneumoniae* isolates in the presence of lidocaine compared with saline controls, suggesting that *S. pneumoniae* may be underestimated as a pathogen with the use of the local anesthetic lidocaine.

A recent study in 2005 by Chandan et al. [33] examined whether lignocaine (1% and 2%), another anesthetic agent commonly used prior to bronchoscopy and BAL procedures, inhibited growth of respiratory tract flora, particularly *S. pneumoniae*, *Moraxella catarrhalis*, *H. influenzae*, *P. aeruginosa*, and *C. albicans*. With a microbroth dilution method, lignocaine 2% exhibited bactericidal activity against *S. pneumoniae*, *M. catarrhalis*, and *H. influenzae*; however, no inhibition of growth of *P. aeruginosa* or *C. albicans* was observed. Lignocaine 1% partially inhibited the growth of *S. pneumoniae*. Because of such antimicrobial activity, the authors advise using the lowest concentration possible of local anesthetic prior to bronchoscopy and BAL procedures in order to maximize recovery of pathogens on culture.

Aldous et al. [34] also investigated the potential for false-negative results with culture specimens when using local anesthetics. The antimicrobial activity of 4% lidocaine with phenylephrine and 4% cocaine in nasal procedures was examined. Both agents exhibited antimicrobial activity against the following pathogens: *S. aureus*, *S. pneumoniae*, *K. pneumoniae*, *H. influenzae*, *M. catarrhalis*, and *Enterobacter* spp., with cocaine exhibiting greater inhibition than lidocaine. The authors recommended using a low concentration of anesthetic to decrease the possibility of obtaining false-negative cultures.

Topical anesthetics are used routinely prior to obtaining bacterial cultures for ophthalmic diagnoses such as bacterial keratitis as well. Mullin and Rubinsfeld [35] examined the bacteriostatic and bactericidal effects of three preserved anesthetic agents, proparacaine, tetracaine, and cocaine, on *P. aeruginosa* and *S. aureus*. Proparacaine exhibited the strongest antimicrobial activity, inhibiting the growth of *S. aureus* at even the lowest concentration of 0.125%, whereas *P. aeruginosa* was inhibited at 0.25% and 0.5%. Tetracaine inhibited growth of *S. aureus* at 0.5% and *P. aeruginosa* at 0.25% and 0.5% concentrations. Cocaine exhibited only mild inhibition of growth of *P. aeruginosa* at a 4% concentration. Because culture yields are reportedly suboptimal in diagnosing clinical ulcerative keratitis, the authors proposed that this growth inhibition by local anesthetics is a likely reason, and recommended that clinicians use a low concentration of the minimally inhibitory cocaine in place of the standard commercial anesthetics in order to optimize culture yields.

**ANTIBACTERIAL MECHANISM OF ACTION OF LOCAL ANESTHETICS**

An early study by Leung and Rawal in 1977 [36] reported on a mechanism of action by which tetracaine exerts its bactericidal action on the bacterial cell. The authors found that tetracaine damaged the cell membrane of *P. aeruginosa* through lysis, leakage of intracellular components, dehydrogenase activity, and increased cell wall permeability.

**LOCAL ANESTHETICS AND PROPHYLAXIS OF SURGICAL SITE INFECTION**

Parr et al. [37] analyzed the antibacterial activity of clinical doses of lidocaine with and without epinephrine on isolates of a spectrum of bacterial pathogens common in surgical site infections, namely *E. faecalis*, *E. coli*, *P. aeruginosa*, *S. aureus*, MRSA, and vancomycin-resis-
tant enterococci (VRE). Addition of epinephrine to the local anesthetic solution decreased the rate of vascular absorption, thereby improving the depth and prolonging the duration of local action. Lidocaine inhibited the growth of all pathogens tested independent of the presence or absence of epinephrine in a dose-dependent fashion. The local anesthetic had the greatest effect on E. coli and P. aeruginosa, the gram-negative organisms, and the least effect on S. aureus. Given the results of this study, the authors made the assertion that “wider application of the use of local anesthetics should be mandated” in the treatment of surgical wound infections.

Using an in vivo approach in a guinea pig model, Stratford et al. [38] evaluated the effects of lidocaine with and without epinephrine on bacterial colonization of surgical wounds. Two wounds on each animal were compared for bacteria counts, one of which was infiltrated with lidocaine (2%) and the other left untreated prior to inoculation with S. aureus. The results showed a > 70% decrease in colony counts in the wounds treated with plain lidocaine compared with the controls. However, when epinephrine was added, a 20-fold increase in colony counts compared with controls was found, suggesting that the hypoxia resulting from vasoconstriction directly increases the risk of surgical site infection. This study supports the potential role of local anesthetics in the prophylaxis of surgical site infection, provided their vasodilating properties are not inhibited by vasoconstrictors such as epinephrine.

The results of these investigations are provocative in that they reveal that these agents have the potential to reduce to below an invasive threshold the colony counts of bacteria and fungi normally found in infected wounds. These studies were performed with local anesthetic concentrations typically used in the clinical setting (i.e., bupivacaine 0.125%, 0.25%, and 0.5% and lidocaine 0.5%, 1.0%, and 2.0%).

CONCLUSIONS

As evidenced by numerous studies, local anesthetics serve not only as agents for pain control, but potentially as antimicrobial agents as well. In that capacity, local anesthetics may be considered an adjunct or alternative to traditional antimicrobial means in the clinical or laboratory setting. As Parr et al. claimed [37], “wider application of the use of local anesthetics should be mandated [in the treatment of surgical site infection].” Additionally, caution should be taken when administering local anesthetics prior to diagnostic procedures in which culture specimens are to be obtained, as the antimicrobial activity of the local anesthetic could lead to false-negative results or suboptimal culture yields. In such cases, it is recommended by various authors that if use of a local anesthetic cannot be avoided, the lowest concentration possible of a mildly antimicrobial agent, such as cocaine or ropivacaine, should be used in order to optimize culture yields. The indirect effect of increased perfusion resulting from peripheral vasodilation by the local anesthetic, as well as the direct effect of the local anesthetic’s ability to disrupt microbial cell membrane permeability and lead to cell lysis, all appear to play a role in the antimicrobial capacity of local anesthetics.

REFERENCES


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