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Practice Points

# A hypothesis for association between electrical surgical incision techniques and surgical site infection

C. Clancy [a], S.S. Dunne [a], C. Baban [a,b], S. Tormey [a,b], A. Merrigan [b], N.H. O'Connell [a,c,d], J.C. Coffey [a,b,d], C.P. Dunne [a,d,*]

[a] School of Medicine, University of Limerick, Limerick, Ireland
[b] Department of Surgery, University Hospital Limerick, Limerick, Ireland
[c] Department of Clinical Microbiology, University of Limerick Hospitals Group, Limerick, Ireland
[d] Centre for Interventions in Infection, Inflammation & Immunity (4i), University of Limerick, Limerick, Ireland

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The use of electrosurgical methods may create a microenvironment (in impacted tissues) similar to that of a burn; we hypothesize that this microenvironment may enable growth of bacterial species in the same way that burn wounds do. There may be value in considering this proposed aspect of electrosurgery when choosing appropriate surgical strategies.

Surgical site infections (SSIs) are defined by the US Centres for Disease Control and Prevention (CDC) as infections occurring at the site of surgery within 30 days of an operation or within 90 days if involving an implant [1]. They are among the most frequently occurring surgical complications and are the leading cause of healthcare-associated infection in European hospitals [2]. Associated with considerable morbidity and mortality, SSIs contribute to longer hospital stays, increased healthcare costs (especially where health insurers decline to reimburse hospitals for SSI-related care) and treatment burden [1,2].

Electrical incision techniques, such as electrocautery and electrosurgery, are common in modern surgery. Electrocautery involves channelling a direct current through an instrument that is applied to the tissues directly [3]. Electrosurgery, by contrast, uses an alternating current, which is passed through the patient's tissues [3]. In both electrosurgical and electrocautery techniques, electrical energy is converted to thermal energy, causing denaturation and coagulation, slow vaporization of the tissue water content (desiccation), and rapid vaporization leading to incision (electrosection) [3].

Electrical surgical instruments have been shown to cause tissue damage [3,4]. Madden *et al.* demonstrated delayed healing, increased inflammation and greater susceptibility to infection in tissues exposed to electrosurgical incision compared with sharp incision [5]. Indeed, there are histological similarities between burn wounds and tissues that have undergone thermal damage from such electrical instruments, specifically collagen denaturation, vascular damage, fibrin accumulation, and necrosis [6,7]. Burn-like injuries may create foci of slough and necrotic tissue, which have been proposed to serve as media for chronic infection with consequent impact on health outcomes and costs [8]. Various Gram-negative bacilli, anaerobes, staphylococci, and pseudomonads have been shown to thrive in damaged or burned tissue, causing chronic infection [9,10]. For example, SSIs frequently involve *Pseudomonas aeruginosa*, a species often implicated in both acute and chronic burn wound infection [9,11,12].

In this context, chronic and delayed SSIs have been reported [13,14]. However, systematic or meta-analysis of SSI relating to electrical surgical instruments has been constrained by methodological inconsistencies. Disparities in SSI definition, wound surveillance periods, and inconsistent case-finding and follow-up may limit comparison between studies [15,16]. Cruse and Foord, in a study of more than 23,000 patients, showed that

* Corresponding author. Address: School of Medicine, University of Limerick, Limerick V94 T9PX, Ireland. Tel.: +353-(0)86-0430739.
*E-mail address:* colum.dunne@ul.ie (C.P. Dunne).

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wound infections within 28 days of operation almost doubled when using electrocautery versus sharp incision [17]. By contrast, systematic review data have indicated that no statistically significant increased risk of infection is associated with electrical incision techniques [18]. The methodological inconsistencies within and between such studies may explain why the reported incidence rates of SSI vary widely [1,2,15].

We propose that the thermal damage caused by electrical surgical instruments contributes to subsequent surgical site infection. Surgeons often excise the edges of openings where electrical instruments, or physical retractors, may have caused damage and a possible hypoxic microenvironment. However, the potential impact on patient outcomes and health costs, the plausible rationale for infection detailed above, and discrepancies in the literature regarding potential relationships between electrical surgical incision techniques and SSI warrant further exploration. More precisely, we hypothesize that the tissue damage caused by these tools creates an environment favourable for infection by specific microbes, e.g. *P. aeruginosa*, and one that is strikingly similar to general burn injuries.

As Beriat *et al*. described, burn damage causes interruption of immunological defence at the burn site along with compromised blood flow, creating environments with available nutrients in which opportunistic bacteria may thrive, resulting in infection [6]. Necrotic tissue and slough in surgical wounds have also been suggested to promote biofilm formation, posing challenges with respect to diagnosis of postoperative complications and management of antimicrobial therapies [8,19].

The impact of electrical surgical devices on SSI may be obscured in the literature due to variation in surveillance periods. For example, the 30-day limit advised by the CDC to define SSI would not encompass delayed wound healing, nor would it capture delayed onset of wound infection in chemotherapy patients. For instance, with particular regard to breast surgery patients, Olsen *et al*. noted that over half of SSIs were diagnosed after 30 days and 16.7% were diagnosed between 91 and 180 days post mastectomy [13]. Longer surveillance periods would more likely reflect incidence of SSI in practice. Further, where sharp scalpel is used for parts of the operation, electrical surgical instruments are now almost always used for haemostasis, creating a nidus of necrotic tissue that may confound a fully discrete analysis. Therefore, clear conclusions regarding the potential contribution of electrical surgical instruments to SSI is not possible based on current data. We propose that these methodological issues curtail any complete analysis of SSI, especially chronic infection, and further research is needed to examine the broader impact of electrical surgical instruments on SSI, and potential contribution to such infections.

The histological evidence from animal models combined with experimental evidence of the behaviour of *P. aeruginosa*, both in acute burn wounds and in chronic wounds, provides a possible explanation for the prevalence of *Pseudomonas* spp. in SSI following surgery. Given the difficulties of eradicating *Pseudomonas* in burn wounds once a biofilm is established, and the considerable morbidity and economic costs of chronic infection, a full exploration of incision approach as a potential risk factor seems warranted [8,9]. Such analysis could inform a surgeon's choice of incision technique, in particular for patients at heightened risk of SSI such as patients with a significant comorbidity such as peripheral vascular disease or cancer, or older patients, or those with a significant smoking history [20]. An evaluation of electrical incision instruments as a potential risk factor for SSI may improve understanding of wound infections overall, may inform new treatment protocols for 'burn-like' infections, and help to reduce infection rates and enhance surgical patient safety and outcomes.

In summary, this article has discussed how widely used electrical surgical devices may create a microenvironment similar to that of burns and so facilitate growth of bacterial species in the same way that burn wounds do. We suggest that the proposal is compelling but not surprising; however, it *is* surprising how little is present in the literature regarding the concept. A full and thorough review of the literature, which assesses the quality of available studies, is needed and well-designed prospective studies may provide further clarity.

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