# Surgical Site Infections

# 115

A. Walker and M. Wilson

**Learning Goals**

- Appreciate the high burden of morbidity caused by SSI and the need for effective auditing practices.
- Know the likely causative organisms for SSI and how they interfere with normal wound healing.
- Recognise patients at high risk for developing SSIs, and consider perioperative and surgical strategies to minimise their occurrence.

## 115.1 Introduction

Surgical site infection (SSI) is one of the most common postoperative complications in both elective and emergency surgery, occurring in at least 5% of all patients and significantly higher following emergency surgery. An estimated 110,800 SSI occurred in the USA in 2015 [1], with each SSI estimated to increase hospital inpatient stay by 6 days on average and be a major contributor to patient morbidity and mortality [2, 3]. With the annual volume of major surgical procedures estimated to exceed 234.2 million cases worldwide [4], SSI are likely to represent an underestimated burden for both patients and healthcare providers. SSIs are also a major risk factor for the development of wound dehiscence and incisional hernias (IH), resulting in further increased morbidity to patients and cost to healthcare resources [5].

In contrast to the increased burden of wound complications and SSI associated with emergency surgery, there is a relative paucity of evidence available compared to the elective setting. Pragmatic hurdles, including difficulties in patient recruitment and a lack of follow-up for these patients, contribute to this problem. Here we offer guidance based on the existing body of emergency surgery research, with additional guidance and opinion derived from the elective laparotomy setting.

### 115.1.1 Epidemiology

The reported incidence of SSI after surgery varies widely, due to not only geographic and health-economic factors but also variation in SSI surveillance practices and case definitions [6]. In the USA, an estimated 110,800 SSI occurred in 2015; however, the true incidence is most likely

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significantly higher as most infections may be diagnosed after discharge from the hospital [7, 8] and hence escape standard auditing practices.

In England in 2019–2020, the national SSI Surveillance Service (SSISS) collecting data on open surgical procedures from 17 surgical categories identified hip and knee replacement surgery as carrying the lowest SSI risk (0.5% for both), whereas bile duct, liver, or pancreatic surgery as well as large bowel surgery carried the highest risk of SSI (9.1% and 8.3%, respectively), with the median time to infection varying from 6 days for cholecystectomy to 22 days for knee replacement [9]. The breakdown of SSI by anatomical plane reveals large differences between procedures; 75% of SSI for bile duct, liver, and pancreatic surgery were organ/space SSIs, whereas these accounted for 32% of SSI in large bowel surgery and 22% in hip replacement.

Rates of SSI vary by geographical location and, in particular, by the country's Human Development Index (HDI). This is partly explained by low- and middle-HDI countries having correspondingly higher rates of emergency surgery than high-HDI countries, as well as increased proportions of contaminated and dirty surgery. In addition, low-HDI countries carry a higher risk of SSI independent of the acuity of care and degree of contamination [8].

The additional cost burden of each SSI is estimated to lie between $10,443 and $25,546 US dollars per infection in the USA [10]. The presence of multi-drug-resistant organisms can increase the cost further, with MRSA SSI adding a mean weighted $13,901 cost to a hospital stay in the USA [11] and up to 12-fold increased rates of mortality.

### 115.1.2 Aetiology and Risk Factors

Surgical wound class is a major risk factor for SSI, as the bacterial inoculum both in the affected tissues and in the incision will inevitably be high. The risk of SSI increases with each class of wound contamination (Table 115.1).

Risk factors for SSI are categorised into patient- and procedure-related risk factors (Table 115.2). Although the scope for modifying or optimising risk factors is often limited in the context of emergency surgery, it is nonetheless important to identify these in order to inform the peri- and intra-operative decision-making, obtain informed consent, and implement risk-reduction strategies for SSI.

Although the exact mechanism of action for these risk factors is often unclear, most patient-related risk factors are likely to be related to the impairment of innate host defences, acting both on the clearance of infectious organisms and impairing the wound healing process.

**Table 115.2** Risk factors for surgical site infection

| Patient factors | Surgical factors |
|---|---|
| • Higher ASA[a]<br>• Age<br>• Diabetes<br>• Obesity (BMI $\geq$ 30 kg/m$^2$)<br>• Poor nutritional status<br>• Smoking history<br>• Alcohol excess<br>• High-dose or long-term steroid treatment | • Emergency surgery<br>• Wound class<br>• Site of surgery (gastroduodenal, small- and large-bowel)<br>• Prolonged operation time<br>• Need for intraoperative blood transfusion<br>• Break in sterile technique |

[a] *ASA* American Society of Anaesthesiologists physical status classification system

**Table 115.1** Wound classification by degree of contamination

| Clean | Clean-contaminated | Contaminated | Dirty |
|---|---|---|---|
| • Elective<br>• No trauma<br>• Hollow viscera not entered<br>• E.g. hernia repair, lipoma cision | • Urgent/emergency care that is otherwise clean<br>• Elective opening of hollow viscera, minimal spillage<br>• Not encountered infected bile or urine<br>• Minor break in sterile technique | • Non-purulent inflammation<br>• Gross spillage from GI tract<br>• Encounter with infected bile or urine<br>• Major break in sterile technique<br>• Penetrating trauma <4 h<br>• Chronic open wound to be grafted or covered | • Purulent inflammation<br>• Pre-operative perforation of hollow viscera<br>• Penetrating trauma >4 h |

### 115.1.3 Classification

SSIs can be categorised by the anatomical plane affected: skin and subcutaneous tissue (superficial incisional SSI), musculofascial planes (deep incisional SSI), and organs or deep space, including the peritoneum and pleura (organ or space SSI) (Fig. 115.1). For incisional SSI, the anatomical plane refers to the deepest layer affected. Intraperitoneal organ/deep space SSI will tend to occur in dependent areas: the pelvis, hepatorenal recess (Morrison's pouch), and splenorenal space.

### 115.1.4 Pathophysiology

#### 115.1.4.1 Physiology of Wound Healing

The process of surgical wound healing begins at the moment the tissue is incised and is characterised by four overlapping and interconnected phases: *haemostasis*, *inflammation*, *proliferation*, and *remodelling*.

From the time of incision, blood components initiate *haemostasis* at the wound edge. This includes platelet aggregation and the release of products such as platelet-derived growth factor (PDGF) and tissue growth factor beta (TGF-β).

Within 48 h of incision, polymorphonuclear neutrophils (PMNs) infiltrate the wound and scavenge necrotic tissue and debris during the initial phase of *inflammation*. PMNs are short-lived and soon die by apoptosis, a process driven in part by infiltrating wound macrophages which characterise the late period of tissue *inflammation* [12]. These macrophages continue to debride the wound by phagocytosis of cellular debris and apoptotic bodies whilst also releasing cytokines including tissue necrosis factor alpha (TNF-α) and TGF-β, eventually leading to the recruitment and activation of fibroblasts [13].

*Proliferation* is driven by both macrophages and fibroblasts starting between days 2 and 5 after tissue incision; the local release of chemokines drives angiogenesis, and fibroblasts secrete fibrillar material into the wound, providing an initial disorganised extracellular matrix (ECM).

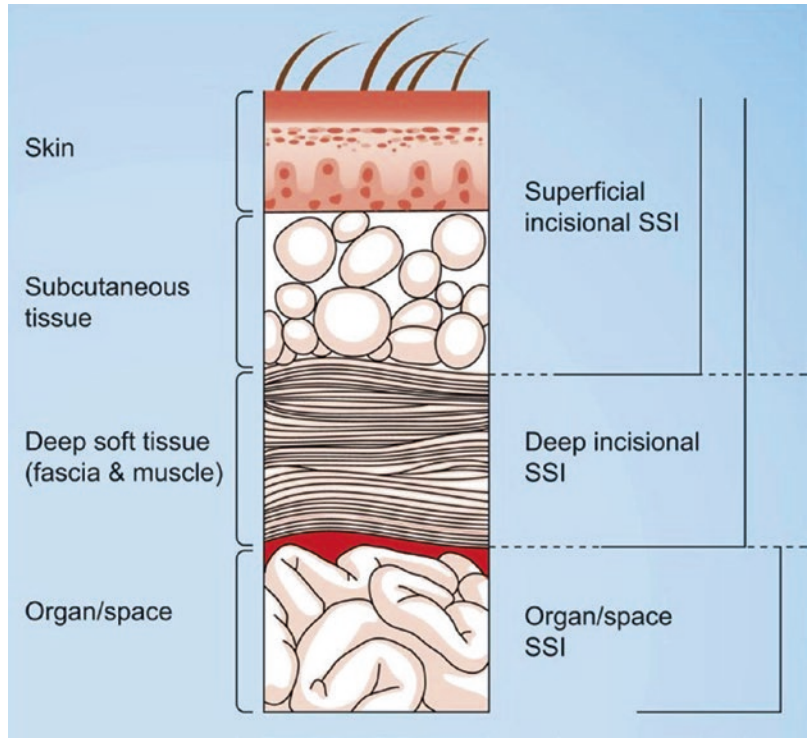


**Fig. 115.1** Classification of surgical site infections by anatomical plane. (Reproduced from [52])

Fibroblasts also take on a contractile phenotype to become myofibroblasts, which contract the ECM and bring together the wound edges, narrowing the superficial gap for re-epithelialisation.

The final phase of wound healing is *remodelling*, whereby a balance of ECM resorption and deposition occurs and replaces the disorganised and weak early ECM with mature scar tissue. Collagen content increases during this stage with a relative decrease in type III collagen [14]. The process can take up to one year to complete, and the tensile strength can reach up to 80% of the original uninjured tissue strength.

Bacteria disrupt the normal wound healing process through a variety of mechanisms. Through direct effects as well as the secretion of endotoxins, they disturb and prolong the inflammatory phase of healing, increase the level of tissue matrix metalloproteinases, and reduce protease inhibitors, leading to poor wound healing or tissue degradation. Bacteria can also move beyond local tissue infection to invasive infection in adjacent tissue.

#### 115.1.4.2 Pathogens in SSI

Causative organisms for SSIs vary depending on the type of surgery and the infected anatomical plane. Organisms usually originate from the patient's endogenous flora; however, they can also be from exogenous sources where a break in sterile conditions occurs. Infections are frequently polymicrobial. Enterobacterales (enteric Gram-negative bacilli), a third of which are *E. coli*, are the most common causative organisms, accounting for up to 29.6% of superficial SSIs and 26.2% of deep incisional or organ/space SSIs. This proportion increases in colonic surgery, where Enterobacterales account for 48.5% of superficial SSIs and 55.7% of deep or organ/space SSIs. *Staphylococcus aureus* is the second most prevalent causative organism for SSIs and can include both methicillin-sensitive and resistant strains depending on regional prevalence, with coagulase-negative staphylococci (CoNS) and *Pseudomonas* spp. being the next most common organisms (Table 115.3).

Drug-resistant organisms are a significant and worsening global concern. A recent prospective study in Saudi Arabia reported that up to 27.7% of Gram-positive and 16.1% of Gram-negative SSI are caused by resistant organisms, and the GlobalSurg collaborative has also identified a high proportion of antimicrobial resistance in SSI, particularly in low-HDI countries (35.9% resistant strains compared with 16.6% in high-HDI countries) [8]. MRSA and vancomycin-resistant *Enterococcus* (VRE) are the most common resistant pathogens encountered worldwide; however, carbapenem and cephalosporin resistance in Enterobacterales organisms and multi-drug-resistant (MDR) organisms, including *E. coli*, are also encountered and present an established threat to modern-day medicine.

**Table 115.3** Most common causative organisms for SSI

| | |
|---|---|
| Enterobacterales | *Escherichia coli* |
| | *Klebsiella* spp. |
| | *Enterobacter* spp. |
| | *Proteus* spp. |
| *Gram-positive cocci* | *Staphyloccus aureus* |
| | *Enterococcus* spp. |
| | Coagulase-negative staphylococci |
| | *Streptococcus* spp. |
| Non-fermenting gram-negative bacteria | *Pseudomonas* spp. |
| | *Acinetobacter* spp. |
| Fungi | *Candida* spp. |
| Anaerobic bacilli | *Bacteroides* spp. |
| Unknown/no identified pathogen | |

## 115.2 Diagnosis

### 115.2.1 Risk Prediction

Multiple tools have been devised to predict the risk of SSI for patients, with the intention of targeting risk-reduction strategies for high-risk patients and for monitoring SSI rates across healthcare networks. An inherent difficulty for SSI risk prediction tools is the need to balance a simple tool based on routinely collected data with the wide variety of SSI incidence for different categories of surgical procedures and across multiple risk factors. Initial attempts at devising a

universal SSI risk prediction tool have now been superseded by procedure-specific algorithms; however, predictive ability overall remains poor [15, 16].

An example of an SSI risk prediction model is the CDC National Healthcare Safety Network (NHSN) risk index (Table 115.4), which uses three variables accounting for patient- and procedure-specific risk factors. An NHSN risk index of 2–3 has been shown to significantly increase the risk of SSI across a number of surgical procedures, and high-risk patients are up to four times more likely to develop an SSI [9].

### 115.2.2 Diagnostic Criteria

Surgical site infections (SSIs) occur at the operative site within 30 days of surgery, or within 90 days of surgery where foreign bodies, including prostheses and mesh, are used. A diagnosis of SSI usually requires purulent discharge from the wound or evidence of a collection or an inflammatory process (*rubor, calor, dolor, tumor*) with breakdown in tissue apposition. The most commonly adopted definition and classification for SSI is that by the Centers for Disease Control and Prevention (CDC) (Table 115.5) [1].

**Table 115.4** NHSN risk index. Low risk = 0–1, high risk = 2–3

| NHSN criteria | Points |
|---|---|
| ASA score ≥ 3 | 1 |
| Operation duration ≥ 'T-time'[a] | 1 |
| Contaminated or dirty wound | 1 |

[a] T-time defined as ≥75th percentile for that operation

## 115.3 Prevention and Treatment

### 115.3.1 SSI Prevention

Prevention is better than cure, and in the case of SSI, a range of peri- and intra-operative strategies have been shown to reduce the incidence of infection [17]. These strategies can broadly be divided into peri-operative care and surgical strategies.

#### 115.3.1.1 Peri-operative Care

##### Pre-operative Antibiotics

Antibiotics should be given when there is a confirmed or high suspicion of infection. Antibiotics

**Table 115.5** Criteria for SSI, adapted from the Centre for Disease Control and Prevention

| | Superficial incisional SSI[a] | Deep incisional SSI | Organ/space SSI |
|---|---|---|---|
| Time frame | Within 30 days | Within 30 days or 90 days if implants used | Within 30 days or 90 days if implants used |
| Anatomical plane | Skin and subcutaneous tissue | Deep soft tissues (e.g. musculofascial layer) | Deeper to the relevant musculofascial plane, in a recognised site of organ/space SSI |
| Infection criteria | • Purulent discharge from wound<br>• Positive microbiology from a suspected SSI<br>• Localised pain or tenderness, swelling, erythema, or heat<br>AND<br>Superficial incision is deliberately opened[b]<br>• Clinical diagnosis by an experienced practitioner | • Purulent discharge from the deep wound<br>• Localised pain, tenderness or fever (>38 °C)<br>AND<br>Deep layer spontaneously dehisces or is deliberately opened or aspirated<br>AND<br>Positive microbiology<br>• Abscess or evidence of deep incisional infection detected on gross anatomical or histopathological examination, or imaging | • Purulent discharge from a drain placed into the organ/space (during or after the procedure)<br>• Positive microbiology from a suspected SSI<br>• Organ/space abscess or evidence of infection detected on gross anatomical or histopathological examination, or imaging |

[a] Includes laparoscopic port sites
[b] Cellulitis does not meet criteria for SSI diagnosis

should be administered through the parenteral route, and the timing of administration, where practicable, should aim to achieve the highest target site concentration at the time of incision. This is broadly accepted to be within 30–60 min of incision for most antibiotics or 2 h for vancomycin and fluoroquinolones [18]. Antibiotic choice should be dictated by the type of surgery involved and the most likely pathogens encountered during that surgery whilst also taking into account local hospital guidelines and antimicrobial resistance patterns..

Intra-operative re-dosing during surgery should be considered for extended surgery or for estimated blood losses of over 1500 mL. The specific dose frequency of antibiotics will depend on the antibiotic's particular pharmacokinetics. For example, beta-lactam agents exhibit antimicrobial activity when their target tissue concentration remains above the bacterial minimal inhibition concentration (MIC), hence making them suitable for higher frequency dosage or continuous infusion. In contrast, peak serum concentration is more closely associated with efficacy for aminoglycosides, and re-dosing is therefore not necessary during surgery [17, 19].

#### Peri-operative Normothermia

Hypothermia is an independent risk factor for mortality in major trauma patients and forms part of the physiological "lethal triad" along with acidosis and coagulopathy [20]. Core body temperature is normally kept within a narrow homeostatic range. However, in the emergency setting, particularly in the context of major trauma, patients are prone to hypothermia due to a combination of environmental exposure, heat loss from open wounds and surgical incisions, and homeostatic derangements caused by anaesthesia.

The benefit of normothermia during surgery also applies to SSI; during major elective and emergency surgery, maintaining normothermia has been shown to decrease the rate of SSI. In a series of 200 patients undergoing elective colorectal surgery, Kurz et al. [21] reported a decrease in SSI from 19% to 6% for patients where normothermia was maintained through a combination of forced air and fluid warming.

#### Glycaemic Control

Diabetes is a recognised risk factor for poor wound healing and SSI [8]. Moreover, acute hyperglycaemia has also been shown to have deleterious effects on the physiological wound healing process and to contribute to increased rates of SSI. The immune system, neutrophils, in particular, shows an impaired inflammatory response in the presence of hyperglycaemia, characterised by delayed migration/chemotaxis and defective phagocytosis. Keratinocyte migration and proliferation are hampered by the hyperglycaemic environment, in turn preventing re-epithelialisation from taking place [22]. Hyperglycaemia also enhances the deleterious effects of hypoxia on wound healing through a variety of mechanisms.

Retrospective cohort studies of surgical patients have shown pre- and postoperative hyperglycaemia (above 10.0 mmol/L) to be significant predictors of SSI. The intensity of glycaemic control should be cautious; the NICE-SUGAR trial collaboration has shown that "tight" glycaemic control (4.5–6.0 mmol/L) is overall deleterious with increased incidence of severe hypoglycaemia. A conventional glucose target of 10.0 mmol/L or less is an adequate target perioperatively and in particular for postoperative ICU patients [23].

#### Supplemental Oxygen

Increased oxygen delivery to tissues was thought to reduce the rate of SSI by improving tissue healing. Although early studies suggested a benefit for supplemental oxygen (usually 80%), more recent large RCTs and meta-analyses have shown no clear benefit for supplemental oxygen and have raised the possibility of long-term harm [24, 25]. Definitive evidence is still lacking, in particular data for emergency surgery.

### 115.3.1.2 Surgical Strategies

#### Skin Preparation and Optimal Incision

The midline incision remains the most common method of laparotomy in emergency surgery. It offers quick and adequate access to the abdominal cavity and avoids the need to enter musculofascial planes such as the rectus sheath, which may

be relevant for contaminated or dirty wounds. Although it leads to higher incisional hernia rates than the paramedian incision, the latter is more time-consuming to perform, which may be disadvantageous in emergency surgery [26]. There is no difference in the rate of SSI between different types of access [27], although caution should be used when wound contamination is anticipated.

Hair at the surgical site has not been shown to increase the risk of SSI; it should not be removed unless necessary, in which case clippers should be used rather than shaving [28]. The debate over skin scrubbing solution is ongoing; however, evidence favours alcohol-based solutions over aqueous agents, and chlorhexidine may be superior to povidone-iodine [29].

### Wound Protectors

Wound protector devices, or "wound retractors", consist of a semi-flexible ring connected to an impervious membrane, used to protect wound edges and improve surgical exposure during laparotomy. The semi-flexible ring is introduced intraperitoneally with the membrane forming a drape over the wound edge. The membrane either hangs freely outside the abdominal cavity ("single ring") or is connected to a second rigid ring providing a frame for support and retracting the wound edge (the "double-ring" design, Fig. 115.2). Wound protectors are thought to reduce the SSI rate by acting as a physical barrier and preventing the incursion of pathogens into the skin and subcutaneous tissue protected by the device. They also provide haemostasis to the wound edge, and by controlling humidification and preventing environmental exposure to the wound edge, they may contribute to local temperature control.

Evidence from several meta-analyses supports the use of wound protector devices, in particular double-ring designs. There is a pooled risk reduction of 30% and up to 70% with the use of dual-ring wound protector designs for overall SSI and superficial SSI. The benefit of reducing SSI applies both to clean-contaminated and contaminated wounds, with an unclear benefit for dirty wounds [30, 31].

It should be noted, however, that the largest randomised controlled trial investigating the role of wound protectors (the ROSSINI Trial, $n$ = 760), which was conducted across elective and emergency settings and included in both meta-analyses, did not show a benefit of wound protector devices. In this study, the overall postoperative SSI rate was 25.0%, with no significant difference between wound protector and control groups [32].

### Fascial Closure

#### Interrupted or Continuous Sutures

The evidence base for fascial closure technique is heterogenous, in part due to the wide variety of interrupted closure methods reported in the lit-


**Fig. 115.2** Double-ring wound protectors reduce the incidence of SSI

erature. However a systematic review and meta-analysis by van't Riet et al. concluded there is no significant difference between interrupted and continuous sutures in terms of SSI or incisional hernias. The continuous fascial closure technique is significantly faster, which makes it more appropriate in the context of emergency surgery [33]. A suture length-to-wound length ratio (SL/WL) of 4/1 or higher reduces the incisional hernia rate [34], and animal models have shown that this ratio provides a high rate of collagen deposition as long as excessive pulling along the suture line with consequent tissue ischaemia is avoided [14].

### Absorbable or Non-absorbable Sutures

As discussed above, the early stages of wound healing are characterised by an inflammatory response with progressive deposition and strengthening of the extracellular matrix, which can take up to a year to reach its maximal tensile strength. This long remodelling phase and the overall reduction in tensile strength mean incisional hernias can take between months and years to develop [35], leading some surgeons to advocate closure with non-absorbable sutures. However, the use of non-absorbable sutures such as polypropylene is associated with increased postoperative pain and the development of stitch sinus infections in the longer term.

### Coated Sutures

The potential for suture material to act as a foreign body and contribute to SSIs is well known, and monofilament sutures such as polydioxanone or polypropylene have a lower bacterial burden than multifilament sutures such as polyglactin 910. The use of antiseptic suture coating ("Plus sutures") further reduces bacterial counts and has the added benefits of localised action, low acute toxicity and altered resistance patterns. Triclosan is the most commonly used coating; however, other coatings including chlorhexidine-based coatings are also available [36, 37].

In a meta-analysis by Daoud [38] including 4800 patients from 15 randomised controlled trials, triclosan-coated polyglactin 910 braided sutures were shown to reduce the risk of SSI by up to 30% versus uncoated polyglactin 910. This included clean, clean-contaminated, and contaminated wounds. A cost analysis by Leaper et al. [39] suggests an overall cost saving of between £63 and £470 per surgical procedure.

Evidence for the use of coated sutures for dirty wounds is still relatively sparse; although its clinical effectiveness for polyglactin 910 is clear [40], in contrast there is conflicting evidence for coated polydioxanone sutures. A well-conducted trial by Justinger et al. [41] including clean-contaminated and contaminated wounds has shown a reduction in SSI using loop-coated polydioxanone suture closure across all categories of wound contamination; however, the result from a meta-analysis including mostly elective surgeries remains equivocal [42]. The FALCON trial, run by the GlobalSurg collaborative, is due to report results and may shed further evidence on the effectiveness of antiseptic-coated sutures in both elective and emergency settings.

## Skin Closure

### Wound Washout and Topical Antibiotics

Skin and subcutaneous tissue washout using saline, povidone-iodine, or antibiotic solution is widely practised and assumed to reduce the rate of SSI. It is thought to work by dilution of local pathogens and physical removal of devitalised tissue, with advocates suggesting it should be vigorous enough to remove debris but careful to avoid further trauma to the wound edge.

A Cochrane meta-analysis of surgical wound washout found that studies were highly heterogenous but suggested a possible benefit for both povidone-iodine wound treatment and wound washout with pressurised saline. Both of these findings were of low certainty given the heterogenous studies and risks of publication bias [43].

### Staples or Sutures

The most common methods of skin closure after emergency surgery are disposable skin staples, interrupted non-dissolvable sutures, and continuous subcuticular dissolvable sutures. Although there is a paucity of evidence from emergency general surgery to favour one strategy over

another, current evidence suggests that continuous subcuticular suturing decreases the rate of superficial wound infection as opposed to interrupted sutures [44]. Similarly, skin staples have been shown to increase superficial wound infection in both orthopaedic and obstetric surgery [45, 46].

Evidence from emergency abdominal surgery is lacking to support either strategy of skin closure; however, studies of skin closure during elective abdominal surgery show no difference in the rate of superficial skin dehiscence or SSI between subcuticular and stapled skin closure [47, 48]. It should be noted, however, that continuous subcuticular skin closure can add up to 30 min of operative time. Although stapled skin closure is not recommended as routine for the closure of emergency surgery, in some circumstances including prolonged operative time or ahead of a planned re-look laparotomy, closure with staples is a reasonable recourse.

#### Negative Pressure Wound Therapy (NPWT)

NPWT uses a suction pump to provide continuous or intermittent negative pressure to the wound (Fig. 115.3). This promotes blood flow and oxygenation to the wound, as well as controlling wound exudate to help promote granulation and wound healing. NPWT has been used for over 25 years and applied to various wound types, in particular diabetic, vascular, and pressure ulcers, burns, and donor sites.

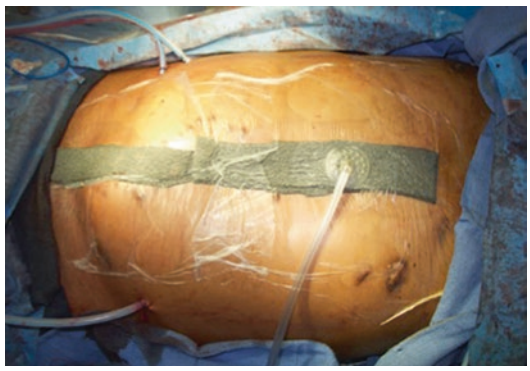

**Fig. 115.3** NPWT should be considered for wounds at high risk of SSI. (Reproduced from [53])

The use of NPWT in traumatic wounds remains contentious: although a Cochrane review including a heterogenous population of surgical patients concluded NPWT can reduce SSI [49], a separate review assessing its use in open traumatic fracture wound closures suggests with moderate certainty that NPWT does not improve wound healing and does not provide a cost-effective treatment modality [50].

The use of NPWT has more recently been used for prophylactic surgical wound management. A meta-analysis by Hyldig et al. [51] showed that amongst a mixed group of scheduled and unscheduled operations, incisional NPWT had a lower incidence of SSI and seroma formation (relative risks of 0.54 and 0.48, respectively), with a non-significant reduction in wound dehiscence. A number of trials are assessing the use of NPWT in obese women undergoing planned or emergency C-sections, and it is possible that the amount of subcutaneous fat and patient BMI are factors that may allow surgeons to target the use of NPWT to high-risk patients.

### 115.3.2 Treatment of SSI

Although a large proportion of SSIs present in the early postoperative period, they may take between weeks and months to present, including after discharge from hospital. It is therefore advisable to have an established institutional SSI surveillance mechanism in place, particularly for high-risk patients.

The broad principles of SSI management involve drainage of infection, debridement of tissue, and the early institution of antibiotics.

#### 115.3.2.1 Incisional SSI

Most incisional SSI will be associated with fluid or purulent collections, and it is advisable to drain this fluid or pus to prevent worsening local or invasive infection and promote wound healing. For superficial incisional SSI, a partial opening

may be made to drain the wound, with localised removal of involved clips or sutures. Where an infection is extensive or there is concern about deep incisional SSI or wound dehiscence, the wound and fascial layer should be probed as a defect in the musculofascial layer will usually require formal operative reintervention and repair. Early treatment with antibiotics is usually required for incisional SSI, using empirical broad-spectrum antibiotics or guided by wound sampling. However, for small and limited superficial incisional SSI, drainage of pus and the use of antiseptics with appropriate interactive moist dressings may be sufficient to control the infection.

Where early measures have failed to control the infection, the portion of skin over the infected wound segment should be opened for wound debridement, at which point it may be allowed to heal by secondary intention or with the use of NPWT, or in very selected cases by delayed primary closure.

#### 115.3.2.2 Deep-Space SSI

Deep-space SSI may present with a failure to progress or acute deterioration and a new onset of sepsis in the postoperative phase. A high index of suspicion is required, and radiological imaging will usually confirm the diagnosis. Deep-space SSI may lead to severe and life-threatening infection, and the initial response to resuscitation and broad-spectrum antibiotics will guide the decision to proceed with further radiologically guided drainage or operative management.

Empirical broad-spectrum antibiotics should be used in the first instance, guided by sensitivities from peripheral blood cultures or aspirates when these become available. The use of percutaneous drainage of abscesses greater than 4 cm in diameter should be considered and will often depend on the precise abscess location and local availability of interventional radiology.

In the unusual circumstance where the above measures fail to adequately resolve the deep-space SSI, surgical management should be considered.

**Dos and Don'ts**

- DO have a range of closure methods available to decrease the risk of SSI.
- DO remove dressings and inspect the postoperative wound when patients are at high risk of SSI.
- DO obtain microbiology samples from SSIs to guide antibiotic choice.
- DON'T leave the closure of high-risk surgical wounds to inexperienced surgeons.
- DON'T continue prophylactic antibiotics unless infection is suspected or confirmed.
- DON'T assume your SSI outcomes are low unless you can show this through a rigorous audit.

**Take-Home Messages**

- Surgical site infection carries significant morbidity and mortality.
- Targeted therapies and bundles should be initiated for patients at high risk of SSI.
- Treatment of SSI relies on the drainage of infection, debridement of tissues, and appropriate antibiotic stewardship.
- Institutions should have audit structures in place to monitor the incidence of SSI.

**Questions**

1. Three days after an emergency Hartmann's resection, a patient develops pain, redness, and swelling around the caudal aspect of their laparotomy wound. The skin remains epithelialized; however, a fluctuant mass is felt. Which of the following is true:
   A. This does not constitute an SSI as there is no break in the skin.

B. **Skin incision should be performed to drain the collection.**
C. There is no value in obtaining microbiology samples as skin flora are the only possible pathogens.
D. If an SSI is suspected, the entire length of the wound needs to be opened.

2. With regards to tissue healing:
A. Wound hypothermia improves wound healing by decreasing metabolic demand on inflammatory cells.
B. Bacterial pathogens cause an increase in protease inhibitors in the infected wound.
C. After the tissue remodelling phase, injured tissue reaches a tensile strength equal or superior to that of uninjured tissue.
D. **Cellular wound debridement by macrophages occurs during the inflammatory phase of healing.**

3. An emergency right hemicolectomy is performed on a 76-year-old man with an obstructing colorectal tumour. They suffer from insulin-controlled type 2 diabetes and mild chronic kidney disease, and their ASA score is 3. There is no perforation or inflammation present during surgery; however, the operation is prolonged due to bleeding and the patient requires an intraoperative blood transfusion. With regards to their risk of SSI.
A. Their CDC NHSN risk score is 1 and they have a low risk of SSI.
B. Their CDC NHSN risk score is 1 and they have a high risk of SSI.
C. Their CDC NHSN risk score is 2 and they have a low risk of SSI.
D. **Their CDC NHSN risk score is 2 and they have a high risk of SSI.**

4. A haemodynamically unstable patient with blunt abdominal injury undergoes an exploratory laparotomy. Which perioperative strategies should NOT be implemented to reduce SSI risk:
A. Maintain normothermia through a combination of forced air and warmed intravenous fluids and blood products.
B. **Maintaining tight glycaemic control to between 4.5 and 6.0 mmol/L.**
C. Consider re-dosing of antibiotics if blood loss over 1.5 L is confirmed during surgery.
D. Aggressive fluid and blood resuscitation to maintain tissue perfusion.

5. With regards to antiseptic-coated (Plus) sutures:
A. Triclosan is a novel suture coating with a poorly understood safety profile.
B. **The use of triclosan-coated polyglycatin 910 reduces the rate of SSI as compared to uncoated polyclycatin 910.**
C. Triclosan only has a narrow spectrum of activity against Gram positive bacteria derived from skin flora.
D. Cost-benefit studies have failed to show a cost savings effect for the use of Plus sutures.

6. Which of the following is NOT a recognised surgical strategy for reducing SSI:
A. **Interrupted fascia sutures rather than continuous suturing.**
B. Use of single-ring wound protectors.
C. Use of clippers where hair removal is required.
D. Fascia closure with Plus sutures.

7. Surgical site infections.
A. Are an uncommon complication of emergency surgery.
B. Always present within 14 days of surgery.

C. Occur most commonly in clean-contaminated wounds.
D. **Are a risk factor for incisional hernias.**

8. Regarding pathogens in SSI.
   A. Positive microbiology is mandatory for a diagnosis of SSI.
   B. **Infections are frequently polymicrobial.**
   C. Antimicrobial resistance is uncommon in SSIs.
   D. Fungal infections do not cause SSIs.
9. Negative pressure wound therapy.
   A. Should not be applied in patient with excessive subcutaneous tissue.
   B. Is only used to treat established SSIs.
   C. **Promotes blood flow and oxygenation to the wound.**
   D. Has a clear evidence base supporting its use in traumatic wounds.
10. After emergency laparotomy, a decision is made to close the fascia with loop polydioxanone. Each suture length is 150 cm. If the laparotomy wound measures 40 cm, what is the minimum number of sutures required for adequate wound closure?
    A. 1
    B. **2**
    C. 3
    D. 4

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