POSITION
DOCUMENT

Identifying criteria for **wound infection**

Understanding wound infection

Clinical identification of wound infection: a Delphi approach

Identifying criteria for pressure ulcer infection

Identifying surgical site infection in wounds healing by primary intention

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POSITION DOCUMENT

Identifying criteria for wound infection

CJ Moffatt

Intense media interest and close public scrutiny have forced the subject of wound infection into the limelight. There is, in particular, interest in the rising prevalence of resistant bacterial strains with their associated morbidity and mortality, and criticism of the indiscriminate use of antibiotics, which has been a crucial contributory factor in the rise of these resistant organisms. There is also an increasing awareness of the cost burden of wound infection. It is clear that clinicians have a professional responsibility to promptly and accurately recognise episodes of infection and to treat them appropriately. This position document on 'Identifying criteria for wound infection' is therefore both pertinent and timely.

If treatment is to be effective, the complexity of the mechanisms involved and the pathophysiology of wound infection must not be underestimated. Cooper, in the first paper of this document, stresses the need for a greater understanding of the complex interactions that precede the development of overt wound infection and clearer definitions of terms such as 'critical colonisation'. Infection is the end result of a complex interaction between the host, organism, wound environment and therapeutic interventions, which is further complicated by bacterial cooperation and virulence. Recognition of subtle clinical changes in the inflammatory response will be necessary if the early signs of infection are to be identified.

Access to more precise and sophisticated clinical assessment tools will increase the possibility for prompt diagnosis and help reduce patient morbidity. The second paper by Cutting, White, Mahoney and Harding discusses recent work using the Delphi process to identify clinical signs of wound infection in six different wound types. In this study an international, multidisciplinary group of 54 wound care experts generated criteria for infection in each wound type. A key consideration is the fact that, despite some common criteria, each wound type may present with different clinical signs of infection. These are sometimes of a subtle nature and will only be detected by consistent and repeated observation, but may provide vital clues to the early identification of infection.

The two final papers in this document offer a detailed critical evaluation of the criteria generated by the Delphi study in two wound types: pressure ulcers and acute surgical wounds. Both papers emphasise that to be clinically useful, each criterion identified in the Delphi study must be evaluated and validated with a clarification of the definitions used. In the absence of any other existing guidance, this work does raise significant issues and provides a stimulus for further debate and the development of tools to help in the early identification of infection.

The importance of early diagnosis and treatment in patients with Grade 3 or 4 pressure ulcers is emphasised by Sanada, Nakagami and Romanelli. Recognising criteria of infection in these wounds is problematic because the signs of chronic inflammation are so similar to those for overt infection. The focus should be on close observation of the wound over time so that subtle changes can be identified.

In the final paper, Melling, Hollander and Gottrup demonstrate how different the picture is for identifying infection in acute surgical wounds. A number of validated tools exist for diagnosing and classifying surgical site infection. These are designed predominantly for auditing, classification and surveillance. Early surgical discharge and reduced follow-up have implications for data collection and the recognition of the early signs of infection. The paper emphasises the need for the consistent application of recording tools if comparable data is to be collected.

Not all wounds will become infected and the level of suspicion will vary according to the host status, susceptibility to infection and the consequences of any infection. The challenge is to use the criteria generated by the Delphi expert panel as a platform for further work to provide clearer guidance for patients, carers and clinicians. The benefits are clear – improved standards of patient care, faster intervention, reduced patient mortality and lower financial costs to health services worldwide.

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POSITION DOCUMENT

Understanding wound infection

RA Cooper

INTRODUCTION

Infection is the outcome of the dynamic interactions that take place between a host, a potential pathogen and the environment. It occurs when host defence strategies are successfully evaded by micro-organisms and results in deleterious changes in the host. Complex interactions that are not yet fully understood precede the development of an infection.

NORMAL IMMUNE FUNCTION OF SKIN

The human body is not sterile. Its outer surface, as well as canals and cavities that open to the exterior, provide a range of different environmental niches that become inhabited by relatively stable but diverse, mixed communities of micro-organisms that constitute its normal flora. Total numbers of microbial cells are estimated to exceed human cells by a factor of at least ten, yet these commensals do not usually breach natural barriers unless the host becomes immuno-compromised or is wounded. Human host and micro-organisms normally exist in a balanced relationship. Indeed the normal flora can confer advantages to its host in terms of protection from invasion by more aggressive species.

When immuno-competent individuals are wounded an acute inflammatory response is immediately initiated that leads to the ingress of blood proteins and phagocytic cells whose function is to remove tissue debris and micro-organisms. Arrival of these components causes the development of the cardinal signs of Celsus (redness, elevated local temperature, swelling and pain). Coagulation of blood and the formation of a fibrin clot help to establish an immediate plug to stem the movement of substances. Ingress of microbial cells into the epidermis or dermis provides an opportunity for infection, but rapidly mobilised immune responses help to limit this possibility.

Until relatively recently the skin has been viewed simply as a passive barrier to infection, but the presence of both innate and adaptive immune surveillance systems in skin indicates a more sophisticated role in protection against infection[1]. Within the epidermis and dermis reside sentinel cells such as keratinocytes, Langerhans cells, mast cells, dendritic cells and macrophages, which possess surface receptors capable of recognising antigens characteristically associated with pathogenic species. Binding of any of these pathogen-associated molecules to these sentinel cells can cause them to release stored and inducible alarm signals such as antimicrobial peptides, chemotactic proteins and cytokines. These products in turn influence the behaviour of local cells as well as attracting additional cells to the site; they also help to coordinate the adaptive immune response that relies on T and B lymphocytes.

Host issues

Patients at increased risk of developing a wound infection are those in whom immune responses do not occur optimally[2]. Age is considered an important factor, with neonates and the elderly at particular risk of infection. Both infection and wound healing are adversely influenced by poorly controlled diabetes mellitus[3], and dietary imbalances that give rise to either emaciation or obesity; each can affect infection rates. Lifestyle can also impinge on immuno-competency especially stress, alcohol and drug abuse, smoking and lack of exercise or sleep. Tissue oxygen levels influence infection rates[4]; perioperative supplementation of oxygen[5] and patient warming prior to surgery[6] can reduce

KEY POINTS

1. The development of a wound infection is dependent on the pathogenicity and virulence of the micro-organism and the immuno-competency of the host.
2. The host-pathogen interaction does not always lead to disease and additional terms and definitions are required.
3. Microbiological assessment alone is not a reliable method for diagnosing wound infection and a full, holistic assessment of the patient is also required.

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postoperative infection rates. Therapies that affect immuno-competency significantly influence infection rates; steroids can elicit multiple adverse effects and the use of immunosuppressive agents in recipients of transplanted organs cause increased susceptibility to infection and retarded inflammatory responses. The impact of deficiencies in cell-mediated immunity on infection has been reviewed[2].

MICROBIAL PATHOGENICITY

The ability of a micro-organism to cause disease is described by its **pathogenicity**, and this is determined by its success in finding a susceptible host, gaining access to suitable target tissue and circumventing host defence mechanisms[7]. The capacity of a micro-organism to cause deleterious effects on a host is known as **virulence**. Multiple factors contribute to microbial pathogenicity, and these can be affected by genetic and environmental influences. In bacteria capable of causing wound infections, structural features, enzyme production and metabolic products contribute to virulence and pathogenicity. The possession of capsules (eg *Pseudomonas aeruginosa* and *Klebsiella pneumoniae*) protect bacteria against phagocyte-mediated killing or complement activation. Fine surface appendages (pili) that extend from many bacteria (eg *Pseudomonas aeruginosa* and *Escherichia coli*) allow attachment to target host cells, which is often the first step in the infection process. Polysaccharide components of the cell walls (eg *Staphylococcus* and *Streptococcus*) facilitate adherence to extracellular matrix components in target tissue, like fibronectin or collagen.

In wounds extracellular infection is more common than intracellular infection and many pathogens rely on the production of extracellular enzymes to invade deep into host tissue.

Host damage also results from the production of microbial toxins. Exotoxins are released from viable bacteria, while endotoxins are integral cell wall components that are released only on microbial cell death and lysis. The effects of both types of toxin are dose dependent and may cause either local or systemic effects. Exotoxins usually demonstrate higher toxicity than endotoxins and affect specific target cells.

The versatility of micro-organisms depends on their ability to rapidly detect and respond to environmental changes. Similarly they can reflect host challenges during the infection process by regulating the expression of genes that code for virulence determinants[7]. Some of these adaptations are cell-density dependent, so that at low numbers virulence genes are not expressed, but when numbers exceed a threshold limit certain genes are expressed and the organism exhibits greater virulence. This phenomenon is known as quorum sensing[8-11].

BIOFILMS

Biofilms are communities of microbial cells, attached to surfaces and encased in a slime. This offers protection against phagocytosis, antibiotics and antimicrobial agents.

Quorum sensing was thought to be restricted to chemical signals passed between cells of the same species, but evidence suggests that a dialogue between different species may exist and that natural flora may have a greater influence than expected[12]. The dynamics of such interactions are not yet fully understood. A further complication is the possibility that polymicrobial communities in wounds might form biofilms. These have been demonstrated in animal wound models[13], and because biofilms have previously been linked to persistent human infections[14] their presence in chronic wounds may be linked to failure to heal.

HOST-PATHOGEN INTERACTIONS AND OUTCOMES

Distribution patterns of micro-organisms are always subject to a combination of chemical, physical and biological factors and every microbial species has specific demands that must be satisfied for its continued survival in any given place.

Wounds do not all provide identical conditions and therefore different wounds support different communities of micro-organisms[15]. Acquisition of microbial species by wounds can lead to three clearly defined outcomes:

- contamination
- colonisation[16]
- infection.

Outcomes of host-pathogen interactions	
Contamination	All wounds may acquire micro-organisms. If suitable nutritive and physical conditions are not available for each microbial species, or they are not able to successfully evade host defences, they will not multiply or persist; their presence is therefore only transient and wound healing is not delayed.
Colonisation	Microbial species successfully grow and divide, but do not cause damage to the host or initiate wound infection.
Infection	Microbial growth, multiplication and invasion into host tissue leads to cellular injury and overt host immunological reactions. Wound healing is interrupted. Local factors can increase the risk of infection.

The critical colonisation debate

One further situation has been described as 'critical colonisation'[17]. The difficulty in distinguishing between colonisation and infection is apparent in this study: two patients with non-healing (not overtly infected) venous leg ulcers responded to antimicrobial intervention. An inference from this study is that an intermediate stage between benign colonisation and overt infection had existed in these wounds. Since the publication of this study a spectrum or continuum of states between wound colonisation and infection has been suggested[18]. Recently further evidence has been reported that topical antimicrobials exhibited a beneficial effect on leg ulcers when healing was impaired by critical colonisation[19,20].

These varying definitions reflect the complex and often unpredictable nature of the interactions that develop between potential hosts, potential pathogens and the environment. Both microbial virulence and host predisposition to infection are subject to change. Definitions of microbial pathogenicity and virulence were originally established when pathogens were invariably regarded as the causative agents of disease without due reference to host responses. However host-pathogen interaction does not always lead to disease, and additional terms and modified definitions have been developed to describe intermediate conditions, which have caused some ambiguity.

Following the perception that the contributions of both pathogen and host must be recognised, the concept of microbial pathogenesis has recently been revised to reflect host damage as the most important outcome of host-pathogen interactions[21]. New definitions and a classification of pathogens based on their ability to cause disease as a function of host immune response were proposed[21]. Against this new framework of host damage, outcomes of host-pathogen interactions were re-examined and re-defined[22]. Infection was defined as the acquisition of a microbe by a host, to discriminate it from disease, which is the clinical manifestation of damage that results from the host-pathogen interaction. Colonisation was defined as the presence of a microbe in a host for an undefined period, with a continuum of host damage ranging from none to significant, depending on the microbe. Failure to remove the microbe would result in persistence, and progressive host damage could result in disease or death. The relevance of these new approaches to wound infection has not yet been accepted or applied, but may explain why some microbes are pathogens in some patients, but not in others.

CRITICAL COLONISATION

- The distinction between colonisation and wound infection is made by evaluating clinical criteria
- Critical colonisation is a term that is in common usage, but the concept needs to be definitively characterised.

In the studies published to date, critical colonisation does not seem to represent a consistent outcome of the host-pathogen interaction. Failure to heal indicates host damage and resolution of healing following antimicrobial interventions indicates microbial involvement[15,17]. Delayed healing and increasing pain suggest possible progression towards overt infection[16]. Critical colonisation has yet to be definitively characterised. Ultimately detailed longitudinal studies will demonstrate whether critical colonisation represents the transition from colonisation to overt infection, or the transition to persistence and perhaps chronic inflammation.

DIAGNOSING WOUND INFECTION

Prompt recognition of wound infection allows suitable antimicrobial interventions to be applied; since infection always interrupts the normal healing process, efficient diagnosis and treatment of infection is required. Monitoring wound infection rates has also contributed to a lower level of infection. Surveillance of surgical infection began in the US during the 1960s with the classification of wounds into four categories (clean, clean-contaminated,

contaminated and dirty or infected) and surveillance reports by Cruse and Foord[23]. Later the Centers for Disease Control and Prevention (CDC) developed definitions for the range of nosocomial infections[24], that were modified in 1992 and surgical wound infections became known as surgical site infections (SSI)[25]. Subjective definitions of wound infection led to the development of two wound scoring systems: ASEPSIS[26] and the Southampton Wound Assessment Scale[27]. For open skin wounds a variety of assessment tools have been developed that employ varying combinations of indicators of infection[28]. In the UK surveillance of surgical site infection in orthopaedics became mandatory on 1st April 2004, and other specialties will soon be included. The need to use a consistent system of diagnosis of wound infection is becoming increasingly imperative, but inconsistencies between tools are apparent[29] (see page 14–17 for further discussion of SSI).

Microbiological criteria

Since the late nineteenth century it has been recognised that the principal pathogens associated with wound infections are *Staphylococcus aureus*, *Streptococcus* species, anaerobes and *Pseudomonas aeruginosa*. In the UK standard operating conditions for the investigation of skin and superficial wound swabs (BSOP 11), and the investigation of abscesses, postoperative wounds and deep-seated infections (BSOP 14) are specified by the Health Protection Agency[30]. Pus, if available, is the preferred specimen, although wound or pus swabs are suitable for processing in laboratories. The regimens are designed to characterise organisms considered to be clinically significant, but many isolates are not identified to species level and numbers are not evaluated. The information provided to healthcare practitioners is, therefore, not normally sufficiently detailed to derive a diagnosis of wound infection without reference to clinical signs and symptoms. Given the incompletely defined nature of inter-microbial interactions, as well as the complicated variety of host-pathogen interactions, holistic assessment of the patient (with its current limitations) is a more reliable way of diagnosing wound infection than microbial assessment alone.

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POSITION DOCUMENT

Clinical identification of wound infection: a Delphi approach

KF Cutting[1], RJ White[2], P Mahoney[3], KG Harding[4]

INTRODUCTION

There is clearly a need for further development of the criteria for early recognition of wound infection. Access to more precise and sophisticated assessment tools will increase the possibility for prompt diagnosis and assist with the obvious benefit of reducing patient morbidity. This article presents and discusses the results of a Delphi study to obtain consensus opinion on criteria for wound infection in six wound types.

HISTORICAL ANALYSIS

Criteria for wound infection

Traditional criteria

- Abscess
- Cellulitis
- Discharge (serous exudate with inflammation; seropurulent; haemopurulent; pus)

Suggested additional criteria

- Delayed healing (compared with normal rate for site/condition)
- Discolouration
- Friable granulation tissue that bleeds easily
- Unexpected pain/tenderness
- Pocketing at base of wound
- Bridging of the epithelium or soft tissue
- Abnormal smell
- Wound breakdown

Adapted from Cutting and Harding, 1994[1]

Wound infection and associated delayed healing present considerable challenges for clinicians, particularly with respect to identifying clinical infection and choosing appropriate treatment options. The development in 1994 of a set of criteria to facilitate the identification of wound infection emphasised the value of additional 'subtle' signs (see Box)[1], which had up to that time been largely unrecognised. The merit of this work has since been confirmed in two subsequent validation studies[2,3]. However, shortcomings in the 1994 criteria became evident when it was recognised that different wound types exhibited their own individual sets of criteria to indicate infection[4].

Although infection is acknowledged as an impediment to healing and prompt intervention is vital[5], few texts concentrate on identifying infection in specific wound types. A notable exception to this is in the field of diabetic foot wounds[6,7] and in surgical wounds[8-11], where formal criteria have been generated.

However, even with these initiatives difficulties remain. For example, identifying infection in diabetic foot ulcers is complicated by the fact that at least 50% of patients 'with a limb-threatening infection do not manifest systemic signs or symptoms'[12]. The answer may lie in identifying 'new' signs of infection, for example, signs that have hitherto been unrecognised or not validated in the literature, but nonetheless are important indicators of infection that can be used in clinical practice.

Refining and defining the clinical signs of wound infection will amplify precision in the identification of wound infection and assist clinicians in recognising the more subtle features for what they are – clinical signs of infection. This confers the obvious benefit of reducing patient morbidity and will have a positive impact on the associated socio-economic burden[13].

METHODS

The Delphi approach

The Delphi process, first developed in the 1950s, is a practical method for developing consensus based on a group response[14]. This involves a number of stages or rounds in which participants are provided with a set of issues on which to comment or rank their views. The group's responses are collated and analysed by an independent researcher and reported back to the group. Participants can compare their own responses with those of the group and decide whether to re-rank their views. The process is repeated until a group consensus is obtained.

The Delphi approach has previously been used in the context of both acute and chronic wound management[15,16] and is a valuable method where inconsistencies or paucity of data exist[17]. In this study, a Delphi approach was used to facilitate the identification of the clinical signs of wound infection in six wound types.

The Delphi group

An international, multidisciplinary Delphi group of 54 members was recruited. Individual members were selected on the basis of possessing recognised expertise in their field, demonstrated through clinical reputation and publication profile. The multidisciplinary group included doctors (physicians and surgeons), nurses, podiatrists and clinical scientists who have a close involvement with clinical practice.

Members of the Delphi group were allocated to one of six panels related to their individual area of expertise. There were 8–10 members in each panel. These panels were set the task of generating criteria for infection in one of the six wound types: acute wounds (primary and secondary); arterial ulcers; burns (partial and full-thickness); diabetic foot ulcers; pressure ulcers and venous leg ulcers.

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Table 1 | **The Delphi process**

Round	
1	Panel members were asked to list the clinical indicators of infection relevant to one wound type group.
2	Criteria from round 1 were collated by the researcher. A new list was returned to panel members with instructions to score each criterion according to importance (0=not important; 9=highly important).
3	Mean, median and standard deviation values were generated from collated responses. Clinically similar criteria and those that demonstrated a correlation coefficient ≥0.7 were merged. Criteria scoring <4 were deleted as they were considered to be of little or no significance by virtue of their low score. Reduced lists were returned to panellists with an invitation to review their own score in light of the group position.
Final	Where scores had been revised in round 3, data was amended and new means, medians and standard deviations generated. Criteria were grouped into three bands according to their scores: 4-5 (important), 6-7 (very important), 8-9 (diagnostic). The structure of these bandings was driven by the data.

To retain the integrity of the Delphi approach, individual panel members were not aware of the identity of other members of the panel. All communication was conducted via email or mail. To help clarify the process and introduce some background to the study, the panel members were provided with copies of four papers[1-3, 14]. The Delphi process followed in this study can be seen in Table 1.

RESULTS

Criteria generated

The results of the study are presented overleaf. These indicate that 'cellulitis', 'malodour', 'pain', 'delayed healing' or 'deterioration in the wound'/'wound breakdown' (although individual descriptions differ) are criteria that are common to all wound types.

An 'increase in exudate volume' was identified as an infection criterion in all wound types except for acute wounds healing by primary intention and burns (full-thickness). This is consistent with clinical observation as full-thickness burns tend naturally to generate large volumes of exudate[18] and acute wounds healing by primary intention do not provide an observable wound bed unless they break down.

Bridging of the epithelium or soft tissue did not feature in any of the panel responses. This is an unexpected finding, particularly in acute wounds healing through secondary intention as it is featured in the literature[19,20]. This is however consistent with the Clinical Signs and Symptoms Checklist (CSSC) developed in 2001[3].

Ranking order

It is important to note that this study did not attempt to categorise criteria by producing subsets of early/late or superficial/deep signs of infection, but to list the clinical indicators of infection and to rank them according to importance. Criteria consistently ranked as 8–9 (mean score) were considered to be diagnostic of infection. Criteria achieving lower mean scores (6–7 or 4–5) were perceived by the panel to be more subtle clinical indicators or signposts of infection. It may be interesting to look at these in relation to the point in time where the change from colonisation to either overt infection or chronic inflammation begins. In addition, it will be important to look at the role of the criteria when used in combinations or clusters.

Clarifying terminology

Clarifying definitions of the terms used will be central to the process of developing the criteria into more useful clinical tools. Some of the terms used lack robust definition or may differ between wound types. A good example of this is the term 'delayed healing', first identified as a criterion for infection in 1994[1].

In this study, delayed healing featured as a sign of infection in the acute wounds group together with diabetic foot, pressure and venous leg ulcers. However, in these latter three, delayed healing is qualified when it occurs despite appropriate intervention (eg offloading and debridement, relevant measures and appropriate compression therapy).

ACUTE WOUNDS – PRIMARY

- Cellulitis
- Pus/abscess

- Delayed healing
- Erythema ± induration
- Haemopurulent exudate
- Malodour
- Seropurulent exudate
- Wound breakdown/enlargement

- Increase in local skin temperature
- Oedema
- Serous exudate with erythema
- Swelling with increase in exudate volume
- Unexpected pain/tenderness

ACUTE WOUNDS – SECONDARY

- Cellulitis
- Pus/abscess

- Delayed healing
- Erythema ± induration
- Haemopurulent exudate
- Increase in exudate volume
- Malodour
- Pocketing
- Seropurulent exudate
- Wound breakdown/enlargement

- Discolouration
- Friable granulation tissue that bleeds easily
- Increase in local skin temperature
- Oedema
- Unexpected pain/tenderness

DIABETIC FOOT ULCERS

- Cellulitis
- Lymphangitis
- Phlegmon
- Purulent exudate
- Pus/abscess

- Crepitus in the joint
- Erythema
- Fluctuation
- Increase in exudate volume
- Induration
- Localised pain in a normally asensate foot
- Malodour
- Probes to bone
- Unexpected pain/tenderness

- Blue-black discolouration and haemorrhage (halo)
- Bone or tendon becomes exposed at base of ulcer
- Delayed/arrested wound healing despite offloading and debridement
- Deterioration of the wound
- Friable granulation tissue that bleeds easily
- Local oedema
- Sinuses develop in an ulcer
- Spreading necrosis/gangrene
- Ulcer base changes from healthy pink to yellow or grey

ARTERIAL LEG ULCERS

- Cellulitis
- Pus/abscess

- Change in colour/viscosity of exudate
- Change in wound bed colour*
- Crepitus
- Deterioration of wound
- Dry necrosis turning wet
- Increase in local skin temperature
- Lymphangitis
- Malodour
- Necrosis – new or spreading

- Erythema
- Erythema in peri-ulcer tissue – persists with leg elevation
- Fluctuation
- Increase in exudate volume
- Increase in size in a previously healing ulcer
- Increased pain
- Ulcer breakdown

** black for aerobes, bright red for Streptococcus, green for Pseudomonas*

VENOUS LEG ULCERS

- Cellulitis

- Delayed healing despite appropriate compression therapy
- Increase in local skin temperature
- Increase in ulcer pain/change in nature of pain
- Newly formed ulcers within inflamed margins of pre-existing ulcers
- Wound bed extension within inflamed margins

- Discolouration eg dull, dark brick red
- Friable granulation tissue that bleeds easily
- Increase in exudate viscosity
- Increase in exudate volume
- Malodour
- New onset dusky wound hue
- Sudden appearance/increase in amount of slough
- Sudden appearance of necrotic black spots
- Ulcer enlargement

PRESSURE ULCER

- Cellulitis

- Change in nature of pain
- Crepitus
- Increase in exudate volume
- Pus
- Serous exudate with inflammation
- Spreading erythema
- Viable tissues become sloughy
- Warmth in surrounding tissues
- Wound stops healing despite relevant measures

- Enlarging wound despite pressure relief
- Erythema
- Friable granulation tissue that bleeds easily
- Malodour
- Oedema

BURNS – PARTIAL-THICKNESS

- Cellulitis
- Ecthyma gangrenosum

- Black/dark brown focal areas of discolouration in burn
- Erythema
- Haemorrhagic lesions in subcutaneous tissue of burn wound or surrounding skin
- Malodour
- Spreading peri-burn erythema (purplish discolouration or oedema)
- Unexpected increase in wound breadth
- Unexpected increase in wound depth

- Discolouration
- Friable granulation tissue that bleeds easily
- Sub-eschar pus/abscess formation
- Increased fragility of skin graft
- Increase in exudate volume
- Increase in local skin temperature
- Loss of graft
- Oedema
- Onset of pain in previously pain-free burn
- Opaque exudate
- Rejection/loosening of temporary skin substitutes
- Secondary loss of keratinised areas

BURNS – FULL-THICKNESS

- Black/dark brown focal areas of discolouration in burn
- Cellulitis
- Ecthyma gangrenosum
- Erythema
- Haemorrhagic lesions in subcutaneous tissue of burn wound or surrounding skin
- Increased fragility of skin graft
- Loss of graft
- Onset of pain in previously pain-free burn
- Spreading peri-burn erythema (purplish discolouration or oedema)
- Sub-eschar pus/abscess formation
- Unexpected increase in wound breadth

- Discolouration
- Friable granulation tissue that bleeds easily
- Malodour
- Oedema
- Opaque exudate
- Rapid eschar separation
- Rejection/loosening of temporary skin substitutes
- Secondary loss of keratinised areas

KEY

HIGH	Mean score 8 or 9
MEDIUM	Mean score 6 or 7
LOW	Mean score 4 or 5

Results of the Delphi process identifying criteria in six different wound types

KEY POINTS

1. A Delphi approach was used to generate criteria for six different wound types.
2. Cellulitis, malodour, pain, delayed healing or deterioration of the wound/ wound breakdown are criteria common to all wound types.
3. Criteria ranked 8-9 were perceived as important diagnostic criteria.
4. Criteria that were ranked lower may be considered as signposts of infection and may be important in the early recognition of infection.

Defining delayed healing is difficult. A rigorous approach is therefore required to explore what constitutes delayed healing in the six different wound types. The subtlety of definitions is further illustrated in the different descriptions of exudate. For example, exudate is described as opaque in burns, while in arterial and venous ulcers an increase in the viscosity is described. Although the dynamic nature of exudate content is known to be related to the infection status of the wound[21], it remains to be seen if variations in exudate features can be related to specific wound types when they become infected.

Identification of new criteria

The advantage of using a Delphi approach can be seen in the generation of some new and interesting criteria. Ecthyma gangrenosum[22] is usually regarded as a rare complication of burns[23]; interestingly, the panel ranked this feature highly in both partial and full-thickness wounds. Alteration in colour in partial-thickness burns was also considered to be pathological of wound infection by the burns panel.

'Crepitus' and 'phlegmon' achieved high mean scores in the diabetic foot ulcer panel in this study, although these features have not been reported previously[7].

Limitations of methodology

Limitations of the research methodology lie in the ambiguity of definitions used and of the term 'importance' in relation to ranking and generation of criteria. In addition, reasons other than infection should be eliminated when assessing the relevance of these clinical signs. For example, a delay in healing could be due to several factors such as poor nutrition, lack of concordance, inappropriate treatment or allergy.

CONCLUSION

The Delphi technique is well established in other areas of clinical practice but its use to generate criteria for infection is novel and challenging. This work provides a stimulus for further debate on how to correlate clinical features with patient outcome and microbiological results in an area, where to-date, most clinicians are unsure of what is happening and often use microbiological results in isolation to diagnose infection. Expansion of this work to ensure international and multidisciplinary acceptance is required as is work on validation.

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POSITION DOCUMENT

Identifying criteria for pressure ulcer infection

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INTRODUCTION

The early diagnosis of infection is difficult in pressure ulcers and requires a high level of clinical suspicion. When infection is present, the potential for further complications such as osteomyelitis and bacteraemia is increased. This paper reviews the existing criteria and the criteria generated by a recent Delphi study[1] to offer clarity in the clinical recognition of infection in Grade 3 or 4 pressure ulcers.

CLASSIFICATION

Pressure ulcers are classified into four grades according to the guidelines of the European Pressure Ulcer Advisory Panel[2]. Infection rarely occurs in Grade 1 or Grade 2 (partial-thickness) pressure ulcers, but is more common in Grade 3 or 4 (full-thickness) pressure ulcers[3], which heal by granulation, epithelial cell migration from the wound edge and wound contraction induced by myofibroblast function[4]. The focus of this article is on recognising criteria for the early diagnosis of infection in Grade 3 or 4 pressure ulcers.

RISK FACTORS

Host issues

The majority of Grade 3 or 4 pressure ulcers occur in elderly people and as a result many of these patients will have impaired immune systems related to advanced age, malnutrition or co-morbidities[5]. This increases their risk of infection and also of 'silent infection'. The latter occurs when several classic clinical markers often associated with infection are absent[3]. This is because many patients with pressure ulcers are less able to activate immune responses to the microbiological burden. It is also important to recognise that if there is a deterioration in the general condition of these patients, their susceptibility to infection increases.

Wound issues

Grade 3 or 4 pressure ulcers are chronically open wounds, which may involve other structures such as muscle, bone or joints. This increases the potential for pathogenic invasion. In addition, pressure ulcers are often in the pelvic region and are at increased risk of contamination from faeces or urine. Faecal materials contain high concentrations of bacteria[6], which can result in a heavy bacterial burden in the wound bed or surrounding skin[7]. Urine is sterile and rarely contaminates wounds unless a urinary tract infection is present. However, incontinence of urine can have an adverse effect on the surrounding skin[8].

Many Grade 3 or 4 pressure ulcers contain necrotic tissue within the wound bed. It has been shown that necrotic ulcers contain high levels of both aerobes and anaerobes, and the density of all organisms is greater than in non-necrotic ulcers[9,10].

Tissue ischaemia is usually related to an inadequate blood flow and is closely linked to pressure ulcer development. The relationship between transcutaneous oxygen pressure ($TcPO_2$) levels, which indirectly indicate the level of tissue oxygen density, and chronic wound infection has been demonstrated[11,12]. Compared with non-infected wounds, infected wounds show a significantly lower $TcPO_2$.

The skin of elderly pressure ulcer patients has a decreased density of Langerhans cells. This also results in decreased responsiveness and reduced ability to combat pathogen invasion[13].

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KEY POINTS

1. Host issues should be taken into account when assessing a patient's susceptibility to infection.
2. There is a need to develop a validated tool to facilitate recognition of infection in Grade 3 or 4 pressure ulcers and to establish how such a tool can be used effectively in practice.
3. The key to early identification of overt infection is recognising subtle changes in the patient and the chronically inflamed wound.
4. The criteria generated recently by the Delphi expert panel offer detailed descriptive criteria for recognising infection in pressure ulcers. These could be used as a platform for further investigation.

DIAGNOSIS

The complexity of diagnosis and the differences in patient populations have led to a lack of accurate data on the prevalence and associated mortality rate of pressure ulcer infection. Delayed diagnosis can increase the risk of complications such as osteomyelitis, transient bacteraemia and septicaemia[14], which in turn can lead to multi-organ failure and sometimes death[15,16].

Methods

Quantitative

The bacterial burden of pressure ulcers is typically heavy and since the wound bed is often grossly contaminated, diagnosis using microbiological techniques is not ideal. With pressure ulcers superficial swab cultures generally reflect bacterial colonisation rather than overt infection. Needle aspirations also give limited detail as the material taken is liquid[17]. The results of bone culture or culture of other deep-tissue biopsy specimens should not be used as the sole criterion for infection without supporting clinical or histopathological evidence[18,19].

Qualitative

The development of clinical criteria for pressure ulcer infection, with the exception of classical signs and symptoms, is limited. Several tools such as DESIGN[20], the Pressure Sore Status Tool (PSST)[21], Pressure Ulcer Scale for Healing (PUSH)[22] and the Sussman Wound Healing Tool[23] are available for assessing pressure ulcer wound status (wound size, depth, granulation tissue condition and infection). However assessment of infection is based on the classic signs only (erythema, oedema, elevated temperature and pain). These indicators are often present in the absence of infection as these wounds are in a state of chronic inflammation. It is important, therefore, to establish whether a change in these indicators is predictive of wound infection.

The 2004 Delphi study presented in this document is the first attempt to generate criteria specific to pressure ulcer infection (Fig 1)[1]. Cellulitis is by definition diagnostic of wound infection[11] and this concurs with its high ranking by the Delphi pressure ulcer panel. The Delphi panel also identified the classic signs of erythema, oedema and pain, but perhaps more usefully have described some of them in more detail (ie 'spreading erythema' and a 'change in nature of pain'). The term 'spreading erythema' helps to distinguish between chronic inflammation when erythema is present and a change in condition where the erythema is spreading. The presence of pus was not ranked as diagnostic of infection. This is important as accurately determining whether pus is present is difficult in these wounds. For example, the effect of certain dressings can give exudate a pus-like appearance.

Criterion	Mean score
Cellulitis	8 or 9
Change in nature of pain Crepitus Increase in exudate volume Pus Serous exudate with inflammation Spreading erythema Viable tissues become sloughy Warmth in surrounding tissues Wound stops healing despite relevant measures	6 or 7
Enlarging wound despite pressure relief Erythema Friable granulation tissue that bleeds easily Malodour Oedema	4 or 5

Figure 1| Criteria identified by the Delphi panel for pressure ulcers[1]

EVALUATION OF EXISTING CRITERIA

Validated criteria

The validity of each of the criterion generated by the Delphi pressure ulcer panel has yet to be demonstrated. A study by Gardner and colleagues previously investigated the validity of the clinical signs and symptoms of chronic wound infection proposed by Cutting and Harding in 1994[11,24]. Pressure ulcers accounted for 53% of the 36 subject wounds, and 27% of these were diagnosed as being infected according to quantitative bacteriology. As a result, 'increasing pain' and 'wound breakdown' were shown to be sufficient indicators of infection with a specificity of 100%. 'Foul odour' and 'friable granulation tissue' also showed some evidence of validity (although not 100%)[11]. These criteria are identified in the Delphi study, but are usefully described in more detail:

- *Increasing pain/change in nature of pain* Pressure ulcers can cause localised pain, and when infected, the pain often increases. It is likely that if a wound is infected, the nature of the pain will also change with the immunological response[25].
- *Wound breakdown/wound stops healing despite relevant measures/enlarging wound despite pressure relief* Infection can interrupt the normal wound healing process. This is due to competitive metabolism, destructive toxins, intracellular replication or antigen-antibody responses[3].

VALIDATED CRITERIA

- Increasing pain
- Wound breakdown

Validated by Gardner SE et al, *Wound Repair Regen* 2001[11]

Figure 2 | Suggested recommendations for early recognition of infection in Grade 3 or 4 pressure ulcers based on the work of the recent Delphi study[1]

The key is recognising subtle changes in the patient and the wound. It is important to:

- Provide accurate and regular documentation
- Document wound appearance (eg size, level of exudate, type of tissue)
- Document appearance of surrounding skin (eg level of erythema)
- Ensure regular pain assessment
- Be alert to subtle deterioration in the patient's general condition
- Be alert to subtle changes in the patient's behaviour (eg loss of appetite, confusion)

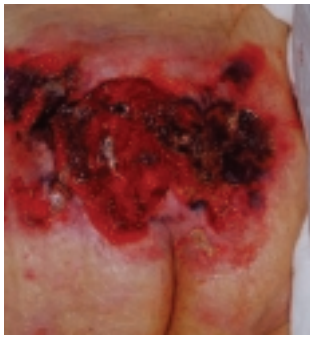
The chronically inflammed wound may have the following signs:

- Erythema
- Exudate
- Serous exudate with inflammation
- Enlarging wound despite pressure relief

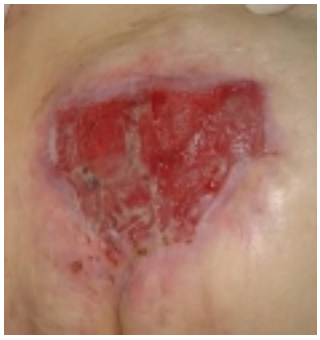
Subtle changes in the wound suggesting infection include:

- Increase in pain severity/ change in nature of pain
- Erythema becomes spreading
- Level of exudate increases
- Odour becomes apparent or foul
- Tissues become friable and bleed easily
- Previously viable tissues become sloughy
- Wound stops healing despite relevant measures

The presence of cellulitis is indicative of overt infection



Spreading erythema and an increasingly painful wound indicate overt infection.



Erythema has resolved and the pain has reduced. The wound is no longer infected.

- *Foul odour/malodour* 'Malodour' was not ranked highly by the Delphi pressure ulcer panel. This may be related to the fact that odour can occur in the absence of infection, although a definite odour is associated with protein degradation from specific bacteria[10].
- *Friable granulation tissue* Although granulation tissues becomes friable when the wound is infected, recognising this in practice is clinically very difficult because of the lack of granulation tissue and the presence of hypergranulation caused by shear and friction.

'Serous exudate with (concurrent) inflammation' and 'warmth of surrounding tissue (heat)' were indicators that did not reach statistical significance in the study by Gardner and colleagues as predictors of wound infection[11,26].

Longitudinal observation

Reviewing these criteria raises a number of practical issues that need to be addressed to ensure their clinical relevance. An interesting aspect is that many of the criteria require close monitoring of the wound over time. An 'increase in exudate volume' is a good example of this. Although this criteria was not previously validated, a high exudate level is often observed in infected pressure ulcers[27]. Assessing volume of exudate is, however, complicated because some absorbent dressings (ie hydrocolloids, hydropolymers or polyurethane foams), when applied to a wound, may reduce the level of visible exudate. Criteria such as 'change in nature of pain', 'wound stops healing' or 'enlarges', 'viable tissues become sloughy' and 'spreading erythema' also require close monitoring. Observing such subtle changes in a chronically inflamed wound is difficult and will demand a high level of vigilance and commitment from clinicians (see Fig 2). The problem is exacerbated for those assessing the wound for the first time and will depend on access to accurate and exemplary documentation.

Criteria in combination

Most of the criteria listed by the Delphi panel, when viewed in isolation, may be due to causes other than wound infection. Healing, for example, can be interrupted by other factors such as external force, malnutrition, co-morbidities including chest or urinary tract infection, and medication. When more than one or two of the criteria are observed, the level of suspicion is raised – the clinician may note that erythema starts to spread into the surrounding tissues and, on probing, the wound is painful to touch and bleeds easily. It is important that these criteria are referred to within an holistic assessment of the patient. For example, changes in the patient's behaviour such as a loss of appetite, patient withdraws socially or becomes confused, may be additional indicators of infection.

The importance of using criteria in combination to achieve accurate diagnosis has been debated in other wound types[28]. However, further investigation is clearly required to establish which combinations of criteria, including criteria unrelated to the wound, have the greatest impact on facilitating early identification of infection in pressure ulcers.

New criteria

'Viable tissues becomes sloughy' and 'crepitus' were identified by the Delphi pressure ulcer panel as indicators of infection, although these have not previously been described in the literature. Crepitation in surrounding tissue may indicate the presence of gas in the subcutaneous tissue. Although there are few reports documenting crepitation in relation to wound infection, this item is regarded as a clinical sign of gas gangrene. Bates-Jensen used crepitation as a sign of severe oedema to assess wound status in the PSST[21].

Further investigation is required to evaluate the importance of these new criteria.

CONCLUSION

Early diagnosis of infection in patients with Grade 3 or 4 pressure ulcers can reduce the risk of complications and lead to improved patient outcomes. At present the methods used to diagnose pressure ulcer infection are limited due to the complexity of these wounds. The results of bacterial tests, for example, do not always correlate with the clinical signs and symptoms, which may be absent or altered in the chronically inflamed wound. The 2004 Delphi study suggests some subtle criteria that may be useful in the early recognition of infection[1], although evaluation is needed to scientifically validate these criteria and to identify which combinations of criteria, including holistic criteria, are clinically useful. The need for sequential observation and accurate documentation of both the wound and the patient's status is necessary if an increasing bacterial load is to be recognised and for effective treatment to begin without delay.

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POSITION DOCUMENT

Identifying surgical site infection in wounds healing by primary intention

A Melling[1], DA Hollander[2], F Gottrup[3]

INTRODUCTION

Most sutured surgical wounds heal normally. In these patients it is simple to identify that no infection has occurred. However, for a significant number of patients wound healing is affected by a variety of problems including haematoma, seroma (sterile collection of serous fluid below the wound surface) and infection. The key to identifying infection is recognising the difference between a complication of healing, such as a haematoma, and a surgical wound that has become infected. This paper uses existing tools and the results of a recent Delphi study[1] to discuss the early identification of surgical site infection (SSI) in wounds healing by primary intention.

IDENTIFYING SURGICAL SITE INFECTION

SSIs are largely preventable and are one of the most common healthcare associated infections (HAIs) to affect surgical patients. There are multiple factors that influence surgical wound healing and determine the potential for, and the incidence of, infection[2,3]. The median time for a wound infection to present is nine days[4]. The increase in day-case procedures and shortened hospital stay has meant that many postoperative infections occur after discharge. Patients therefore require careful follow-up in the community post surgery to enable early identification of infection and appropriate instigation of treatment.

Definitions of SSI

There are many definitions of infection that can aid the process of accurate diagnosis. One simple definition is that infection presents as a purulent discharge or a painful erythema, indicative of cellulitis[5]. However, all simple definitions of infection contain an aspect of subjectivity; for instance, it may even be difficult to obtain agreement on the presence of pus in a wound between two healthcare workers, as pus can present in several different colours and consistencies. This is why most definitions now try to aid the user with additional criteria and symptoms.

The most widely recognised definition of SSI is that devised by Horan and colleagues and adopted by the Centers for Disease Control and Prevention (CDC)[6]. This definition is now used throughout the US and in Europe. It splits SSI into three groups: superficial, deep and organ space, depending on the site and the extent of the infection. A summary of the definition of superficial SSI is presented below. Controversially, the CDC definition states that a wound infection can be diagnosed by an attending physician or surgeon without apparently meeting the definition criteria[6].

Wound scoring systems

Several wound scoring systems exist; two of the most widely recognised are ASEPSIS[7] and the Southampton Wound Assessment Scale[8]. These enable surgical wound healing to be graded according to specific criteria, usually giving a numerical value, thereby providing a more objective assessment of the wound[7,8]. The ASEPSIS scoring system was devised to assess wounds following cardiothoracic surgery and can be used to categorise the severity of infection. Wounds are given a score depending on the extent of any wound healing

CDC definition of superficial surgical site infection (SSSI)[6]

- Infection occurs within 30 days of procedure
- Involves only skin or subcutaneous tissue around the incision

And at least **one** of the following:

- Purulent drainage from the superficial incision
- Organisms isolated from an aseptically obtained culture of fluid or tissue from the superficial incision
- At least one of the following signs or symptoms of infection: pain or tenderness, localised swelling, redness, or heat *and* superficial incision is deliberately opened by surgeon, *unless* culture of incision is negative
- Diagnosis of superficial incisional SSI by the surgeon or attending physician

The following are not reported as superficial SSI: (1) stitch abscess (minimal inflammation and discharge confined to the points of suture penetration), (2) infection of an episiotomy or neonate's circumcision site, (3) infected burn wound and (4) incisional SSI that extends into the facial and muscle layers (see deep SSI)

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complications such as serous exudate, erythema, purulent discharge and separation of deep tissues. In addition, points are awarded for specific criteria such as positive swab results and prescription of antibiotics. Scoring is meant to take place in five of the first seven days postoperatively, and then the additional scores can be added over the subsequent six weeks[7].

The Southampton scoring system was designed for use in the postoperative assessment of hernia wounds. It is much simpler than the ASEPSIS system with wounds being categorised depending on any complications and their extent[8].

These scoring systems require thorough patient follow-up, which is often time-consuming and expensive. For this reason, they have not been widely implemented, although this situation may change with the trend towards mandatory postoperative surveillance. Two studies have examined and used the ASEPSIS system and highlight its benefits in providing less subjective detailed information on wound healing[9,10]. Another study has successfully used the Southampton system for routine infection surveillance and audit[11].

VALIDITY OF EXISTING TOOLS

One recent paper has compared several definitions of infection in the same group of patients and found a large variation in reported rates (6.8–19.2%)[12]. For this reason, one definition should be used consistently when changes in the incidence of SSI are being evaluated over time in a single institution. However, it is still premature to use wound infection rates as a performance indicator for comparing different centres or countries, as a slight adaptation of the CDC definition was found to reduce the rate of infection by 4.6%[12]. The same paper has also shown that the effectiveness of the ASEPSIS scoring system may be reduced when patients are discharged before the minimum seven days as the scoring system only identified 6.8% of patients with infection when 12.3% of patients were classified as infected due to the presence of pus alone[12]. The ASEPSIS and Southampton scoring systems can help grade wound healing and identify infection; however both systems have been specifically designed for use after either cardiovascular surgery or hernia surgery. The recent publication by Wilson and colleagues[12] shows that ASEPSIS may be less valid when used on patients with a short length of postoperative stay and these concerns are reflected by other authors[9,10].

DISCUSSION

Clinical signs and symptons

Criterion	Mean score
Cellulits Pus/abscess	8 or 9
Delayed healing Erythema ± induration Haemopurulent exudate Malodour Seropurulent exudate Wound breakdown/enlargement	6 or 7
Increase in local skin temperature Oedema Serous exudate with erythema Swelling with increase in exudate volume Unexpected pain/tenderness	4 or 5

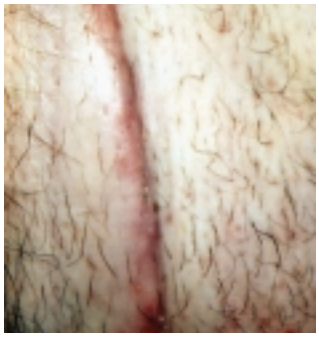
Figure 1| Criteria identified by the Delphi panel for acute wounds healing by primary intention[1]

Even with experience and knowledge, early identification of infection in a surgical wound is difficult as the wound itself may not be open to observation. Interpretations must be made of what is observed in relation to what is happening under the skin. By the time a purulent discharge is observed or cellulitis clearly apparent, infection is established. The presence of accompanying fever and leucocytosis as systemic indicators of infection varies[3]. Wound infection occurring below muscle or fascial layers or below thick, uninfected subcutaneous tissue (in obese patients) may have a delayed presentation or lack many of the local signs mentioned above.

There is currently no validated, universal system that is designed specifically to aid in the early identification of SSI and help instigate the correct treatment when infection occurs. However, a recent Delphi study[1] generated a list of criteria that were selected by the acute wounds panel as important indicators of SSI in wounds healing by primary intention (Fig 1). The type of surgery was not specified and the assumption is that the criteria are applicable to all types of surgical wounds. In examining the results of the Delphi study, the following discussion raises some important issues related to the early recognition of SSI.

Cellulitis

'Cellulitis' and 'pus/abscess' were identified by the Delphi study as the most important criteria (ranked 8–9) in this wound type and may be considered as diagnostic of infection. Cellulitis is defined as a 'spreading infection of the skin and subcutaneous tissues, characterised by local pain, tenderness, oedema and erythema'. This is a controversial



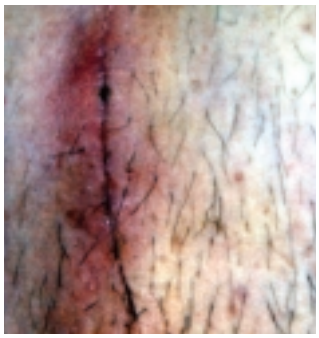
Mild erythema around the suture sites and along the scar. There are no other signs of infection and this wound went on to heal normally.

indicator, as redness and swelling may often appear around the wound for other reasons, perhaps due to the normal inflammation of healing, removal of a dressing, allergy to a dressing, tight-fitting clothes, seroma or haematoma. This ambiguity may be why it does not appear in the CDC definition.

Erythema

Severe erythema can be defined as a painful spreading redness around a wound[5]. The distinction between cellulitis and severe erythema is minor and most definitions of SSI refer to 'erythema' rather than 'cellulitis' as an indicator of infection, providing it is accompanied by other criteria such as a raised temperature or pain[5,7,8].

The inclusion of 'erythema' in a definition of infection has been shown to increase the reported incidence of SSI. In a study of prophylactic antibiotic use in hernia surgery, the reported incidence of infection was 9%. However, if infection had been defined purely as a 'purulent discharge' and/or 'wound breakdown/abscess', then infection rates would have only been 4%[13]. A review of the literature by Reilly[11] has shown that in many studies, if the definition is limited to a 'purulent discharge' alone then infection rates were found to be between 1% and 5%. However, in those where 'erythema' or 'cellulitis' is included in the definition infection rates were 6–17%.



More extensive erythema in conjunction with some swelling. The surrounding skin is hot and painful to touch. This wound eventually broke down with a purulent discharge.

Purulent discharge

It is universally agreed that the presence of pus and/or abscess or a purulent discharge indicates the presence of infection[5-8].

It is interesting to note that the Delphi acute wounds panel[1] identified 'seropurulent exudate' and 'haemopurulent exudate' as important indicators of infection (mean score 6 or 7). However, haemopurulent and seropurulent discharge could simply be classified as 'pus' or a 'purulent discharge' and the inclusion of these as additional indicators reinforces the need for clarity in relation to defining the terms used[8]. Discharge due to infection most commonly presents around 5–10 days post surgery, although any discharge from the closed surgical wound after 48 hours of closure is of concern and warrants investigation.

It is not clear how important 'malodour' is in the identification of SSI and it is not included in any of the validated definitions or wound scoring systems. However, a discharge that becomes foul smelling is a clearer indication of infection.

Early signs of infection

Crucially, the Delphi study attempts to identify other, more subtle, early indicators of infection. These include 'serous exudate with erythema', 'swelling with increase in exudate volume', 'oedema', 'increase in local skin temperature' and 'unexpected pain/tenderness'. Most of these are also used by other definitions as collaborative signs of infection[5-7].

The focus needs to be on translating these criteria so they have clinical value to the non-expert. For example, of more concern than 'unexpected pain', is pain that begins or increases around the wound area in conjunction with other signs of inflammation several days after surgery. The inflamed skin around the wound will usually be warmer than the surrounding area and also painful to touch. A summary of these issues is illustrated in Figure 2.

Using criteria in combination

From the literature, it is clear that accurate diagnosis depends on looking at a number of criteria in combination to exclude causes other than infection for the clinical signs and symptoms observed. A delay in healing, induration and/or wound breakdown standing alone may be related to other factors – for example, wound breakdown/enlargement may be due to poor suturing, suturing under high tension or inadequate coagulation.

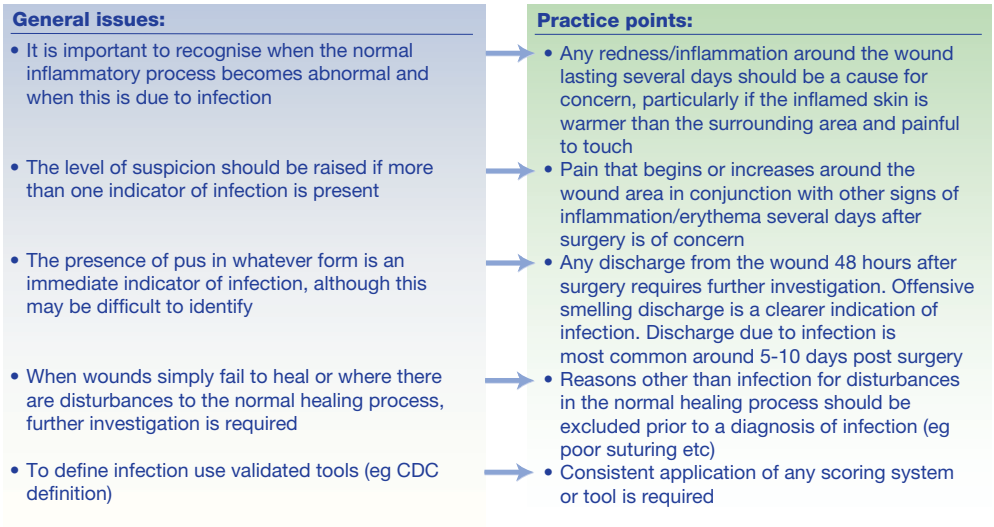


Figure 2 | Some basic recommendations for the early recognition of SSI

CONCLUSION

It is clear that there are already definitions and scoring systems that aid in the assessment of surgical wound healing and the diagnosis and classification of SSI. The most commonly used, the CDC definition, uses stringent criteria to classify infection. This allows audit of practice and surveillance of SSI. However, these stringent criteria may place a reduced emphasis on the more subjective, subtle signs of infection such as erythema. The Delphi study[1] has identified a number of these subtle indicators of infection that should not be ignored clinically. Clarity and guidance is required for both the patient and clinician to recognise when the normal inflammatory process becomes abnormal and when the cause of this is likely to be due to infection. The focus needs to be on establishing whether infection will be potentially severe or devastating and will require treatment with antibiotics, or whether the wound can be managed with less intervention and avoid unnecessary antibiotic treatment and risk of resistance.

KEY POINTS

1. There are well established definitions and scoring systems for defining, classifying and grading the severity of infection.
2. The early recognition of SSI depends on identifying a number of criteria in combination.
3. Discussions around the criteria developed by a recent Delphi study have been used to develop basic recommendations in the early recognition of SSI.

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