

NATIONAL STANDARD METHOD

INVESTIGATION OF ABSCESSSES AND DEEP-SEATED WOUND INFECTIONS

BSOP 14

Issued by Standards Unit, Department for Evaluations, Standards and Training
Centre for Infections



Scottish Microbiology Forum

Association of Medical Microbiologists
Association of Medical Microbiologists
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INVESTIGATION OF ABSCESSSES AND DEEP-SEATED WOUND INFECTIONS

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AMENDMENT PROCEDURE

Controlled document reference	BSOP 14
Controlled document title	Investigation of abscesses and deep-seated wound infections

Each National Standard Method has an individual record of amendments. The current amendments are listed on this page. The amendment history is available from standards@hpa.org.uk.

On issue of revised or new pages each controlled document should be updated by the copyholder in the laboratory.

Amendment Number/ Date	Issue no. Discarded	Insert Issue no.	Page	Section(s) involved	Amendment
5/ 23.11.09	4	5	All	All	Department name changed from ESL to DEST
			All	All	PDF links inserted to cross-reference NSM documents
			All	All	The term "CE Marked leak proof container" replaces "sterile leak proof container"; endnote ^a added to clarify the change; and referenced to the IVD Directive 98/79/EC.
			1	Front Page	NIMAG, Scottish Forum and CMN logos added
			2	Status	Taxonomy sentence inserted
			12	Introduction	Gangrene section amended
			17	4.4.3	Updated and amended
			19	4.5.2	Links to reference laboratory user manuals inserted.
			22	Appendix	Flow chart inserted
23	References	Reviewed and updated			

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INVESTIGATION OF ABSCESSSES AND DEEP-SEATED WOUND INFECTIONS

Types of specimens: Abscess pus Post-operative wound swab
Abscess swab Deep-seated pus swab
Wound exudate

SCOPE OF DOCUMENT

This National Standard Method (NSM) describes the processing and bacteriological investigation of specimens from abscesses and from post-operative wound and deep-seated infections.

INTRODUCTION

Abscesses are accumulations of pus in the tissues and any organism isolated from them may be of significance. They occur in many parts of the body as superficial infections or as deep-seated infections associated with any internal organ. Many abscesses are caused by *Staphylococcus aureus* alone, but others are caused by mixed infections. Anaerobes are predominant isolates in intra-abdominal abscesses and abscesses in the oral and anal areas. Members of the "*Streptococcus anginosus*" group and Enterobacteriaceae are also frequently present in lesions at these sites.

Bartholin gland abscesses and tubo-ovarian abscesses are considered in [BSOP 28 – Investigation of genital tract and associated specimens](#). Processing of specimens for *Mycobacterium* species from, for example, subcutaneous cold abscesses is described in [BSOP 40 – Investigation of specimens for Mycobacterium species](#).

Brain abscess²

Brain abscesses are serious and life-threatening.

Sources of abscess formation include:

- Direct contiguous spread from chronic otitic or paranasal sinus infection
- Metastatic haematogenous spread either from general sepsis or secondary to chronic suppurative lung disease
- Penetrating wounds
- Surgery
- Cryptogenic (ie source unknown)

Treatment of brain abscesses involves the drainage of pus and appropriate antimicrobial therapy. Brain stem abscesses have a poor prognosis due to their critical anatomical location³.

Bacteria isolated from brain abscesses are usually mixtures of aerobes and obligate anaerobes, and the prevalent organism may vary depending upon geographical location, age and underlying medical conditions. The most commonly isolated organisms include⁴⁻⁸:

- Anaerobic streptococci
- Anaerobic Gram-negative bacilli
- "*Streptococcus anginosus*" group
- Enterobacteriaceae

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- *Streptococcus pneumoniae*
- β -haemolytic streptococci
- *S. aureus*

Organisms commonly isolated vary according to the part of the brain involved. Many other less common organisms, for example *Haemophilus* species, may be isolated^{2,6-13}. *Nocardia* species often exhibit metastatic spread to the brain from the lung. Any organism isolated from a brain abscess must be regarded as clinically significant.

Organisms causing brain abscesses following trauma may often be environmental in origin, such as *Clostridium* species¹⁴ or skin derived, such as staphylococci and *Propionibacterium* species.

Brain abscesses due to fungi are rare. Aspergillus brain abscess can occur in patients who are neutropenic. Zygomycosis is an uncommon opportunistic infection caused by *Rhizopus* and *Absidia* species and related fungi. *Scedosporium apiospermum* (*Pseudallescheria boydii*) enters the lungs and spreads haematogenously¹⁵.

Breast abscess

Breast abscesses occur in both lactating and non-lactating women. In the former infections are commonly caused by *S. aureus*, but may alternatively be polymicrobial, involving anaerobes and streptococci¹⁶⁻¹⁸. Signs include discharge from the nipple, swelling, oedema, firmness and erythema.

In non-lactating women a subareolar abscess forms often with an inverted or retracted nipple. Mixed growths of anaerobes are usually isolated¹⁹. Some patients require surgery involving complete duct excision¹⁹. Abscesses may also be caused by *Pseudomonas aeruginosa* and *Proteus* species²⁰.

Carbuncles, furuncles, cutaneous, soft tissue and other abscesses

Carbuncles are deep and extensive subcutaneous abscesses involving several hair follicles and sebaceous glands. Carbuncles are most often caused by *S. aureus*.

Furuncles are abscesses which begin in hair follicles as firm, tender, red nodules that become painful and fluctuant. Furuncles are caused by the same pathogens as carbuncles. Recurrent staphylococcal furunculosis is highly infectious and may be the first sign of an underlying disease such as diabetes mellitus.

Cutaneous abscesses are usually painful, tender, fluctuant erythematous nodules often with a pustule on top. In some cases they are associated with extensive cellulitis, lymphangitis, lymphadenitis and fever. They are caused by a variety of organisms. The location of an abscess often determines the flora likely to be isolated. Thus *S. aureus* is most often isolated from cutaneous abscesses of the axillae, the extremities and the trunk, whereas cutaneous abscesses involving the vulva and buttocks may yield faecal or urogenital mucosal flora.

Soft tissue abscesses involve one or more tissue planes underlying the epidermis, usually developing after trauma to the skin. They may arise from animal bites, in which case common isolates include *Pasteurella* and *Actinobacillus* species²¹ as well as other organisms of the HACEK group (*Haemophilus*, *Actinobacillus*, *Cardiobacterium*, *Eikenella* and *Kingella* species).

Burkholderia pseudomallei causes melioidosis, but is rare in the UK. The disease may present in a variety of forms with skin lesions and/or cellulitis. Diagnosis is made by blood culture, serology or culture of pus (refer to [BSOP 37 – Investigation of blood culture \(for organisms other than mycobacterium species\)](#)).

Pyomyositis is a purulent infection of skeletal muscle in which solitary or multiple muscle abscesses form. It most often occurs in tropical areas, and in HIV-infected or other patients who are immunocompromised. The main causative organism is *S. aureus*^{22,23}.

Abscesses in intravenous drug users

Cutaneous abscesses frequently occur as a complication of injecting drug use. They commonly result

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from the use of non-sterile solutions in which the drug is dissolved or from lubrication of the needle using saliva.

Common bacterial isolates include²⁴:

- Oral streptococci
- *Streptococcus anginosus* group
- *Fusobacterium nucleatum*
- *Prevotella* species
- *Porphyromonas* species
- *Staphylococcus aureus*
- *Clostridium* species

Dental abscess

Dental abscesses involve microorganisms colonising the teeth that may become responsible for oral and dental infections, leading to dentoalveolar abscesses and associated diseases. They may also occur as a direct result of trauma or surgery.

Periodontal disease involves the gingiva and underlying connective tissue²⁵, and infection may result in gingivitis or periodontitis.

Organisms most commonly isolated in acute dentoalveolar abscesses are facultative or strict anaerobes. The most frequently isolated organisms are anaerobic Gram-negative rods, however other organisms have also been isolated. Examples include^{23,25-28}:

- α -haemolytic streptococci
- Anaerobic Gram-negative bacilli
- Anaerobic streptococci
- "S. *anginosus*" group
- *Actinobacillus actinomycetemcomitans*
- Spirochaetes
- *Actinomyces* species

Aspiration of dental abscesses is necessary to obtain samples containing the likely causative organisms. Swabs are likely to be contaminated with superficial commensal flora.

Liver abscess

Liver abscesses can be amoebic or bacterial (so-called pyogenic) in origin or, more rarely, a combination of the two.

Pyogenic liver abscesses usually present as multiple abscesses and are potentially life-threatening. They require prompt diagnosis and therapy by draining and/or aspirating purulent material, although it is possible to treat liver abscesses with antibiotics alone. They occur in older patients than those with amoebic liver abscesses, and are often secondary to a source of sepsis in the portal venous distribution.

Examples of the sources of pyogenic liver abscess include²⁷:

- Biliary tract disease
- Extrahepatic foci of metastatic infection

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- Surgery
- Trauma

Many different bacteria may be isolated from pyogenic liver abscesses. The most common include²⁹⁻³²:

- Enterobacteriaceae
- *Bacteroides* species
- *Clostridium* species
- Anaerobic streptococci
- "S. anginosus" group
- Enterococci
- *P. aeruginosa*
- *B. pseudomallei* (in endemic areas)

Other causes include *Candida* species.

Amoebic liver abscesses arise as a result of the spread of *Entamoeba histolytica* via the portal vein from the large bowel which is the primary site of infection (investigation of amoebae is described in [BSOP 31 – Investigation of specimens other than blood for parasites](#)).

Hydatid cysts may also occur as fluid-filled lesions in the liver. However, the clinical presentation is usually different from that of liver abscesses (see [BSOP 31 – Investigation of specimens other than blood for parasites](#)). Cysts may become super-infected with gut flora and progress to abscess formation.

Lung abscess³³

Lung abscesses involve the destruction of lung parenchyma and present on chest radiographs as large cavities often exhibiting air-fluid levels. This may be secondary to aspiration pneumonia, in which case the right middle zone is most frequently affected. Other organisms may give rise to multifocal abscess formation and the presence of widespread consolidation containing multiple small abscesses (<2 cm diameter) is sometimes referred to as necrotising pneumonia. Pneumonia caused by *S. aureus* and *Klebsiella pneumoniae* may show this picture. (See [BSOP 57 – Investigation of bronchoalveolar lavage, sputum and associated specimens](#)).

Lung abscesses most often follow aspiration of gastric or nasopharyngeal contents as a consequence of loss of consciousness, resulting for example from alcohol excess, cerebrovascular accident, drug overdose, general anaesthesia, seizure, diabetic coma, or shock. Other predisposing factors include oesophageal or neurological disease, tonsillectomy and tooth extraction.

Lung abscesses may arise from endogenous sources of infection. The bacteria involved in these cases are generally from the upper respiratory tract and anaerobes are often implicated, secondarily infecting consolidated lung after aspiration from the upper respiratory tract. Nosocomial infections involving *S. aureus*, *S. pneumoniae*, *Klebsiella* species and other organisms may also occur.

B. pseudomallei may cause lung abscesses or necrotising pneumonia in those who have visited endemic areas (mainly south east Asia and northern Australia)³⁴ especially in diabetics.

Nocardia infection is most often seen in the lung where it may produce an acute, often necrotising, pneumonia³⁵. This is commonly associated with cavitation. It may also produce a slowly enlarging pulmonary nodule with pneumonia, associated with empyema. Nocardiosis, almost always occurring in a setting of immunosuppression, may present as pulmonary abscesses.

Abscesses as a result of blood borne spread of infection from a distant focus may occur in conditions

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such as infective endocarditis.

Lemierre's syndrome (or necrobacillosis) originates as an acute oropharyngeal infection usually in a young adult. Infective thrombophlebitis of the internal jugular vein leads to septic embolisation and metastatic infection. The lung is most frequently involved but multifocal abscesses may develop. *Fusobacterium necrophorum* is the most common pathogen isolated from blood cultures in patients with this syndrome³³.

Aspergillus species have been isolated from lung abscesses in patients who are immunocompromised.

Pancreatic abscess

Pancreatic abscesses are potential complications of acute pancreatitis. Infections are often polymicrobial and common isolates include *Escherichia coli*, other Enterobacteriaceae, enterococci and anaerobes: longer-standing collections, especially after prolonged antibiotic therapy, are often infected with coagulase-negative staphylococci and *Candida* species.

Perirectal abscess

Perirectal abscesses are encountered in patients with predisposing factors. These include³⁶:

- Immunodeficiency
- Malignancy
- Rectal surgery
- Ulcerative colitis

They are often caused by³⁷:

- Anaerobes
- Enterobacteriaceae
- Streptococci
- *S. aureus*

Pilonidal abscess

Pilonidal abscesses are common in children and result from infection of a pilonidal sinus. Anaerobes and Enterobacteriaceae are usually isolated³⁸, but they may be caused by *S. aureus* and β -haemolytic streptococci.

Prostatic abscess

Prostatic abscesses may be caused by, or associated with³⁹:

- Diabetes Mellitus
- Acute and chronic prostatitis
- Instrumentation of the urethra and bladder
- Lower urinary tract obstruction
- Haematogenous spread of infection

Organisms that may cause prostatic abscesses include⁴⁰:

- *E. coli* and other Enterobacteriaceae
- Anaerobes

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- *Neisseria gonorrhoeae*
- *S. aureus*

Prostatic abscesses can act as reservoirs for *Cryptococcus neoformans* resulting in relapses of infection with this organism⁴¹.

Psoas abscess

Psoas abscesses may be seen as secondary infections to⁴²:

- Appendicitis
- Diverticulitis
- Osteomyelitis of the spine
- Infection of a disc space
- Bacteraemia
- Perinephric abscess

Pus tracks under the sheath of the psoas muscle. Infection often occurs in drug abusers after injection into the ipsilateral femoral vein.

Psoas abscesses are predominantly caused by⁴³⁻⁴⁵:

- Enterobacteriaceae
- *Bacteroides* species
- *S. aureus*
- Streptococci
- *Mycobacterium tuberculosis*

Renal abscess

Renal abscesses are typically caused by Gram-negative bacilli and result from ascending urinary tract infection, pyelonephritis, renal calculi or septicaemia⁴⁶. Diabetes mellitus can also occur in patients who are immunocompromised. *S. aureus* has been replaced by *E. coli* (from urinary tract infections) as the most prevalent organism found in these abscesses.

Perinephric abscesses are relatively uncommon, but serious, extensions of renal abscesses. Infection spreads beyond the cortex and capsule into the perinephric fat. Causative organisms are the same as those for renal abscesses.

Scalp abscess

Scalp abscesses are a recognised complication of electronic monitoring with fetal scalp electrodes during labour. A localised collection of pus surrounded by inflamed tissue forms where the electrodes are inserted. Anaerobes are most commonly isolated, probably as a result of contamination with vaginal organisms during delivery.

Polymicrobial infections also occur, involving⁴⁷:

- Anaerobes
- β -haemolytic streptococci
- *S. aureus*
- Enterobacteriaceae

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- Enterococci
- Coagulase-negative staphylococci

Kerion is a pustular folliculitis of adjacent hair follicles, creating dense inflamed areas of the scalp, and is caused by dermatophytes (see [BSOP 39 – Investigation of dermatological specimens for superficial mycoses](#)). Secondary bacterial infection may occur.

Spinal epidural abscess

Spinal epidural abscesses may occur in patients with:

- Predisposing disease (such as diabetes)
- Prior infection elsewhere in the body which may serve as a source for haematogenous spread
- Abnormality of, or trauma to, the spinal column (often involving invasive medical procedures such as epidural catheterisation)

The most common isolate is *S. aureus*⁴⁸. *Staphylococcus epidermidis* may be isolated in patients following invasive spinal manipulation. Streptococci (α -haemolytic, β -haemolytic and *S. pneumoniae*), Enterobacteriaceae and pseudomonads may also be isolated^{48,49}.

Subphrenic abscess

Subphrenic abscesses occur immediately below the diaphragm, often as a result of⁵⁰:

- Gastric, duodenal or colonic perforation
- Acute cholecystitis
- Procedures on the liver and upper part of the gastrointestinal tract
- Ruptured appendix
- Trauma

Subphrenic abscesses are caused by mixed infections from the normal gastrointestinal flora⁵⁰.

Unusual cases of abscess formation

Unusual cases of abscess formation can occur in patients with many underlying conditions and may be caused by a vast range of organisms⁵¹⁻⁵⁸. Any organism isolated from abscess pus is potentially significant.

Actinomycosis is a chronic suppurative infection characterised by chronic abscess formation with surrounding fibrosis. It is rare and usually follows perforation of a viscus, trauma or surgery. It is caused by *Actinomyces israelii*⁵⁹, usually in mixed culture with other bacteria.

Abscess formation is most often associated with the gastrointestinal tract, the jaw and the pelvis. Other areas of the body may be involved and the formation of abdominal abscesses may occur. Thoracic involvement occurs in 15% of cases of actinomycosis. Pulmonary actinomycosis can be difficult to diagnose prior to cutaneous involvement, which results in direct extension through the chest wall. The disease progresses to form a chronic indurated mass with draining fistulae. Material should be drained from abscesses and biopsies taken. Skin biopsies may reveal the presence of organisms (see [BSOP 17 – Investigation of tissues and biopsies](#)).

"Sulphur granules" are sought in the pus specimen⁶⁰. These are discharged from actinomycosis abscesses. Sulphur granules are colonies of organisms forming a filamentous inner mass which is surrounded by host reaction. They are formed only *in vivo*. They are hard, buff to yellow in colour, and have a clubbed surface.

Post-operative wound infections

Post-operative wound infections arise when microorganisms contaminate surgical wounds during an

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operation or immediately afterwards. Colonised body sites are frequent sources of pathogens, although they may be transmitted via medical and nursing staff or via inanimate objects from other patients or elsewhere in the hospital environment.

Organisms most commonly isolated include:

- *S. aureus* including MRSA
- *Bacteroides* species
- *Clostridium* species
- Enterobacteriaceae
- Pseudomonads
- β -haemolytic streptococci
- Enterococci
- *Peptostreptococcus* species

Coagulase-negative staphylococci and coryneforms isolated from post-operative sites overlying implants or prostheses may indicate infection. This is particularly true in the presence of a sinus tract in direct communication with the joint. However, with the exception of *S. aureus*, superficial flora do not necessarily represent the flora deep inside a wound and cultures should be interpreted with care.

The following, though uncommon, are important clinical conditions and all require surgical debridement as a vital component of therapy:

Soft Tissue and other abscesses

Gangrene

There are four main types:

- 1) **Meleney's progressive synergistic gangrene** - presents as a burrowing lesion or chronic gangrene of the skin following abdominal operations, and results from mixed infections by organisms such as *S. aureus*, streptococci, Enterobacteriaceae, pseudomonads and anaerobic Gram-negative bacilli^{61,62}.
- 2) **Gas Gangrene** - is a necrotising process associated with systemic signs of toxæmia and gas is present in the tissues. It often follows traumatic injuries such as penetrating wounds or crush injuries. Gas gangrene is caused by clostridia, in particular *Clostridium perfringens*. However, these organisms may colonise a wound without causing disease. Alternatively, they may cause a spreading cellulitis, or extend into the muscle causing myonecrosis⁶³. Classical gas gangrene is associated with clinical shock, leakage of serosanguinous fluid, tissue necrosis and presence of gas in the tissues.
- 3) **Non-sporing anaerobes** - are particularly important causes of infection in the pelvic and scrotal areas (when the term Fournier's gangrene is applied) and are common causes of gangrene in ischaemic and diabetic limbs. They often occur in infections mixed with Enterobacteriaceae, streptococci and *Clostridium* species⁶⁴.
- 4) **Spontaneous gangrene** occurs either with no apparent relation to trauma or following mild, non-penetrating trauma. It is most commonly associated with patients with colonic carcinoma, leukaemia or neutropenia. The main causative organisms are *C. perfringens* and *septicum*⁶⁵.

Intra-abdominal sepsis

Intra-abdominal sepsis is infection occurring in the normally sterile peritoneal cavity⁶⁶. The term covers primary and secondary peritonitis, as well as intra-abdominal abscesses.

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Primary peritonitis is infection of the peritoneal fluid in which no perforation of a viscus has occurred. Infection usually arises via haematogenous spread from an extra-abdominal source and is often caused by a single pathogen⁶⁶. It is common in patients with ascites following hepatic failure. In females it may also result from organisms ascending the genital tract, for example *N. gonorrhoeae* and *Chlamydia trachomatis*. pneumococci, actinomycetes, enterobacteriaceae and streptococci have been associated with peritonitis in women with IUCDs but can cause primary peritonitis in any patient group at any age.

Secondary peritonitis is acute, suppurative inflammation of the peritoneal cavity usually resulting from bowel perforation or postoperative gastrointestinal leakage. Secondary peritonitis is most often treated with a combination of surgery and antibiotics.

The most frequent isolates encountered in intra-abdominal sepsis with secondary peritonitis are derived from the normal gastrointestinal flora. Anaerobic bacteria are isolated from the majority of cases with *Bacteroides* species being isolated. However, infections are usually polymicrobial⁶⁷ and organisms that have been isolated include:

- *Enterococcus* species
- *Bacteroides* species
- Pseudomonads
- *Peptostreptococcus* species
- Yeasts
- β -haemolytic streptococci
- *Clostridium* species
- *Enterobacteriaceae*

Tuberculous peritonitis is a rare disease in the UK. It is more common on the Indian sub-continent, so it is important to consider this in immigrants from that area. In most cases a primary pulmonary focus is present with secondary spread of *Mycobacterium tuberculosis* (see [BSOP 40 – Investigation of specimens for Mycobacterium species](#)).

TECHNICAL INFORMATION/LIMITATIONS

In National Standard Methods, the term “CE marked leak proof container” is used to describe containers bearing the CE marking and which are used for the collection and transport of clinical specimens. The requirements of the EU *in vitro* Diagnostic Medical Devices Directive (98/79/EC Annex 1 B 2.1)⁶⁸ state that such devices must “reduce as far as possible contamination of, and leakage from, the device during use and, in the case of specimen receptacles, the risk of contamination of the specimen. The manufacturing processes must be appropriate for these purposes”.

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1 SAFETY CONSIDERATIONS⁶⁹⁻⁷⁴

1.1 SPECIMEN COLLECTION

Avoid accidental injury when pus is aspirated

1.2 SPECIMEN TRANSPORTATION AND STORAGE

Pus in a CE Marked leak proof container^a in a sealed plastic bag

1.3 SPECIMEN PROCESSING

Containment Level 2 unless infection with a Hazard Group 3 organism is suspected on clinical grounds, in which case Containment Level 3 is required

All specimens likely to contain Hazard Group 3 organisms must be processed in a microbiological safety cabinet in a Containment Level 3 (CL3) laboratory.

Thus initial examination and all follow up work on specimens from patients with suspected *Mycobacterium* species, or suggesting a diagnosis of blastomycosis, coccidioidomycosis, histoplasmosis, paracoccidioidomycosis or penicilliosis must be performed inside a microbiological safety cabinet in a CL3 laboratory.

Any grinding of sulphur granules should be performed in a microbiological safety cabinet

Prior to staining, fix smeared material by placing the slide on an electric hotplate (65 to 75°C), under the hood, until dry. Then place in a rack or other suitable holder

Note: Heat-fixing may not kill all *Mycobacterium* species⁷⁵. Slides should be handled carefully

Centrifugation must be carried out in sealed buckets, which are subsequently opened in a microbiological safety cabinet

Specimen containers must also be placed in a suitable holder

Laboratory procedures that give rise to infectious aerosols must be conducted in a microbiological safety cabinet

Refer to current guidance on the safe handling of all organisms documented in this NSM

The above guidance should be supplemented with local COSSH and risk assessments

Compliance with postal and transport regulations is essential

2 SPECIMEN COLLECTION

2.1 OPTIMAL TIME FOR SPECIMEN COLLECTION

Before antimicrobial therapy where possible

2.2 CORRECT SPECIMEN TYPE AND METHOD OF COLLECTION

The specimen will usually be collected by a medical practitioner

Samples of pus are preferred to swabs. However, pus swabs are often received (when using swabs, the deepest part of the wound should be sampled, avoiding the superficial microflora)

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2.3 ADEQUATE QUANTITY AND APPROPRIATE NUMBER OF SPECIMENS

Ideally, a minimum volume of 1mL of pus

Swabs should be well soaked in pus

3 SPECIMEN TRANSPORT AND STORAGE

3.1 TIME BETWEEN SPECIMEN COLLECTION AND PROCESSING

Specimens should be transported and processed as soon as possible

The volume of specimen influences the transport time that is acceptable. Large volumes of purulent material maintain the viability of anaerobes for longer^{76,77}

The recovery of anaerobes is compromised if the transport time exceeds 3 h

3.2 SPECIAL CONSIDERATIONS TO MINIMISE DETERIORATION

Swabs should be transported in Amies transport medium with charcoal⁷⁸

If processing is delayed, refrigeration is preferable to storage at ambient temperature. Delays of over 48 h are undesirable

4 SPECIMEN PROCESSING

4.1 TEST SELECTION

Divide specimen on receipt for appropriate procedures such as examination for parasites ([BSOP 31 – Investigation of specimens other than blood for parasites](#)) and culture for *Mycobacterium* species ([BSOP 40 – Investigation of specimens for Mycobacterium species](#)), depending on clinical details

4.2 APPEARANCE

Describe presence or absence of sulphur granules (if sought)

4.3 MICROSCOPY

4.3.1 STANDARD

Swab

Prepare a thin smear on a clean microscope slide for Gram staining after performing culture (see [QSOP 52 – Inoculation of culture media](#))

Pus

Using a sterile pipette place one drop of neat specimen or centrifuged deposit (see 4.4.1), as applicable, on to a clean microscope slide

Spread this using a sterile loop to make a thin smear for Gram staining (see [BSOPTP 39 – Staining procedures](#))

4.3.2 SUPPLEMENTARY

Gram stain of sulphur granules

With care, either squash the sulphur granules that have been washed in saline (see Section 4.4.1) between two slides using gentle pressure, or use the homogenised granules (see Section 4.4.1) and make a thin smear for Gram staining.

Note: Any grinding of sulphur granules should be performed in a microbiological safety cabinet

For microscopy, *Mycobacterium* species ([BSOP 40 – Investigation of specimens for Mycobacterium](#)

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[species](#)) and parasites ([BSOP 31 – Investigation of specimens other than blood for parasites](#)). For fungi and other staining procedures see [BSOPTP 39 – Staining procedures](#).

4.4 CULTURE AND INVESTIGATION

4.4.1 PRE-TREATMENT

Standard

Exudates

Centrifuge in a sterile, capped, conical-bottomed container at 1200 x g for 5-10 mins

Transfer the supernatant with a sterile pipette, leaving approximately 0.5 mL, to another CE Marked leak proof container^a in a sealed plastic bag for additional testing if required

Resuspend the deposit in the remaining fluid

Supplementary

Wash any sulphur granules that are present in saline

Suspend an aliquot of pus containing sulphur granules in sterile water or saline in a CE Marked leak proof container^a in a sealed plastic bag. Agitate gently to wash pus from the granules

Grind the washed granules in a small amount of sterile water or saline, with a sterile tissue grinder (Griffiths tube or unbreakable alternative) or a pestle and mortar

Use this homogenised sample to make a smear for Gram staining and to inoculate agar plates

Note 1: All grinding of sulphur granules should be performed in a microbiological safety cabinet

Note 2: If a fungal infection is suspected then grinding of the whole specimen should be avoided. This is to prevent damaging hyphae that would result in a reduced yield, particularly with zygomycetes.

4.4.2 SPECIMEN PROCESSING

Pus

Inoculate agar plates and enrichment broth with the pus or centrifuged deposit with a sterile pipette (see [QSOP 52 – Inoculation of culture media](#))

If sulphur granules are present, these should be ground (see Section 4.4.1) and included in the culture

For the isolation of individual colonies, spread inoculum with a sterile loop

All additional pus/fluid from the specimen should be stored for up to 7 days at 4°C

Swabs

Inoculate each agar plate with swab (see [QSOP 52 – Inoculation of culture media](#))

For the isolation of individual colonies, spread inoculum with a sterile loop

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4.4.3 CULTURE MEDIA, CONDITIONS AND ORGANISMS FOR ALL SPECIMENS

Clinical details/ conditions	Standard media	Incubation			Cultures read	Target organism(s)
		Temp °C	Atmos	Time		
All specimens	Blood agar	35-37	5 – 10% CO ₂	40-48 h	daily	<i>S. aureus</i> β-haemolytic streptococci Enterococci
	CLED/ MacConkey agar	35-37	air	16-24 h	≥16 h	Enterobacteriaceae Pseudomonads
	Neomycin fastidious anaerobe agar with metronidazole 5 µg disc	35-37	anaerobic	5 d	≥40 h and at 5 d	Anaerobes
All pus and exudates (not swabs)	Fastidious anaerobe broth then subcultured where appropriate on to above media (excluding CLED/ MacConkey agar)	35-37	air	5 d	N/A	Any organism
		35-37	as above	40-48 h	≥40 h	
For these situations, add the following:						
Clinical details/ conditions	Supplementary media	Incubation			Cultures read	Target organism(s)
		Temp °C	Atmos	Time		
Submandibular abscess Empyema Normally sterile sites such as: Brain abscess Liver abscess Lung abscess Psoas abscess Spinal abscess	Fastidious anaerobe agar	35-37	anaerobic	5 d	≥40 h and at 5 d	Anaerobes
	Chocolate agar	35 – 37	5 – 10% CO ₂	40 – 48 h	≥40 h	Fastidious organisms
Actinomycosis (or where microscopy suggestive of actinomycetes)	Blood agar supplemented with metronidazole and nalidixic acid	35-37	anaerobic	10 d	≥40 h, at 7 d and 10 d	<i>Actinomyces</i> species
Nocardiosis	Blood agar	35-37	air	up to 7 d	at 3 d and 7 d	<i>Nocardia</i> species
Immunocompromised	Sabouraud agar	35-37	air	5 d	≥40 h	Fungi
Prostatic abscess Primary peritonitis in females	GC selective/ Chocolate agar	35-37	5-10% CO ₂	40-48 h	≥40 h	<i>N. gonorrhoeae</i>

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Optional media		Incubation			Cultures read	Target organism(s)
		Temp °C	Atmos	Time		
When clinical details or microscopy suggestive of mixed infection	Staph/strep selective agar	35-37	air	40-48 h	daily	<i>S. aureus</i> Streptococci
	GN medium (NAV)	35-37	anaerobic	Up to 5 d	48 h and 5 d	Gram-negative anaerobes
Other organisms for consideration - Fungi (BSOP 39 – Investigation of dermatological specimens for superficial mycoses) and <i>Mycobacterium</i> species (BSOP 40 – Investigation of specimens for Mycobacterium species)						

4.5 IDENTIFICATION

4.5.1 MINIMUM LEVEL IN THE LABORATORY

Actinomycetes	species level BSOPID 10 – Identification of aerobic <i>Actinomycetes</i> species BSOPID 15 – Identification of anaerobic <i>Actinomycetes</i>
Anaerobes	"anaerobes" level
β-haemolytic streptococci	Lancefield group level
Coagulase-negative staphylococci	"coagulase-negative" level
Enterobacteriaceae	"coliforms" level
Fungi	species level (except yeast to yeast level)
Neisseria	species level
Pseudomonads	species level
S. aureus	species level
" <i>S. anginosus</i> " group	" <i>S.anginosus</i> " group level
<i>Mycobacterium</i>	BSOP 40 - Investigation of specimens for Mycobacterium species
Parasites	BSOP 31 - Investigation of specimens other than blood for parasites

Organisms may be further identified if clinically or epidemiologically indicated or if isolated from normally sterile site.

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4.5.2 REFERRAL TO REFERENCE LABORATORIES

For information on the tests offered, turn around times, transport procedure and the other requirements of the reference laboratory [click here for user manuals and request forms](#).

Organisms with unusual or unexpected resistance, and whenever there is a laboratory or clinical problem, or anomaly that requires elucidation should be sent to the appropriate reference laboratory.

4.6 ANTIMICROBIAL SUSCEPTIBILITY TESTING

Refer to NSM on Antimicrobial Susceptibility Testing ([BSOP 45 - Susceptibility Testing](#)).

5 REPORTING PROCEDURE

5.1 MICROSCOPY

Report on WBCs and organisms detected

For the reporting of microscopy for fungi, *Mycobacterium* species and parasites ([BSOP 40 – Investigation of specimens for *Mycobacterium* species](#)) and parasites ([BSOP 31 – Investigation of specimens other than blood for parasites](#)).

5.1.1 MICROSCOPY REPORTING TIME

Urgent microscopy results to be telephoned or sent electronically

Written report, 16 – 72 h

5.2 CULTURE

Report clinically significant organisms isolated or

Report other growth or

Report absence of growth

Report on the presence of sulphur granules

Also, report results of supplementary investigations

5.2.1 CULTURE REPORTING TIME

Clinically urgent culture results to be telephoned or sent electronically

Written report, 16 – 72 h stating, if appropriate, that a further report will be issued

Supplementary investigations: Fungi, *Mycobacterium* species and parasites ([BSOP 31 – Investigation of specimens other than blood for parasites](#))

5.3 ANTIMICROBIAL SUSCEPTIBILITY TESTING

Report susceptibilities as clinically indicated

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6 REPORTING TO THE HPA (LOCAL AND REGIONAL SERVICES AND CENTRE FOR INFECTIONS)⁷⁹

Refer to the following:

Individual NSMs on organism identification

Health Protection Agency publications:

"Laboratory reporting to the HPA: A guide for diagnostic laboratories"

"Hospital infection control: Guidance on the control of infection in hospitals"

Local guidelines

Report all clinically significant isolates from deep-seated abscesses and metastatic infections to CDSC

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7 ACKNOWLEDGEMENTS AND CONTACTS

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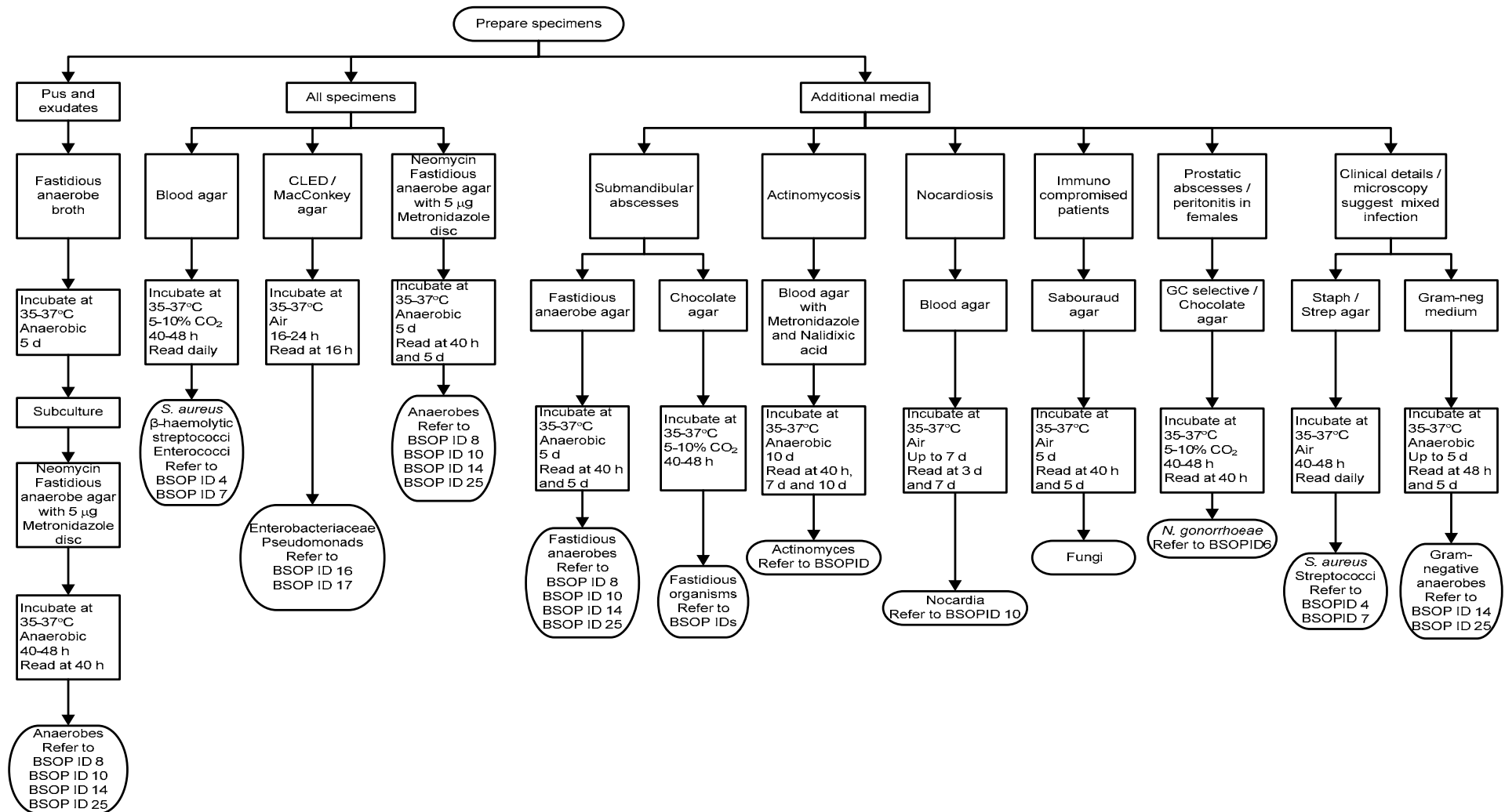
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APPENDIX⁸⁰



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