IGPG1 INFECTION PREVENTION AND CONTROL
PROCEDURAL GUIDELINES
SECTION 1: INFECTION AND COMMON INFECTIOUS DISEASES

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PROCEDURE SUMMARY
The purpose of this document is to ensure that every member of staff has an understanding of the principles of infection prevention and control, the most common infectious diseases and healthcare associated infections, and the management thereof.

The Trust monitors the implementation of and compliance with this procedure in the following ways;
The responsibility for monitoring and reviewing this Policy lies with the Director responsible for Infection Prevention and Control. Compliance with this procedure will be audited. Audit results will be presented to the Infection Prevention and Control Group.

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<tr>
<th>Services</th>
<th>Applicable</th>
<th>Comments</th>
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The Director responsible for monitoring and reviewing this policy is
The Executive Director of Mental Health
SECTION 1: INFECTION & COMMON INFECTIOUS DISEASES

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ESSEX PARTNERSHIP UNIVERSITY NHS FOUNDATION TRUST

SECTION 1: INFECTION & COMMON INFECTIOUS DISEASES

Assurance Statement
The purpose of this document is to ensure that every member of staff has an understanding of the principles of infection prevention and control, the most common infectious diseases and healthcare associated infections, and the management thereof.

1.0 INFECTION AND COMMON INFECTIOUS DISEASES

This section supports ICP1 - Infection Control Policy by explaining the causes and spread of infection and setting out the procedures for staff to follow for common infectious diseases.

2.0 THE CAUSES OF INFECTION

2.1 Micro-organisms that cause infections are known as pathogens. They may be classified as follows:

- **Bacteria** are minute organisms about one-thousandth to five thousandth of a millimetre across. They are susceptible to a greater or lesser extent to antibiotics.

- **Viruses** are much smaller than bacteria and although they may survive outside the body for a time they can only grow inside cells of the body. Viruses are not susceptible to antibiotics, but there are a few anti-viral drugs available which are active against a limited number of viruses.

- **Pathogenic Fungi** can be either moulds or yeasts. For example, a mould which causes infections in humans is *Trichophyton rubrum* which is one cause of ring-worm and which can also infect nails. A common yeast infection is thrush caused by an organism called *Candida albicans*.

- **Protozoa** are microscopic organisms, but larger than bacteria. Free-living and non-pathogenic protozoa include amoebae and paramecium. Examples of medical importance include: *Giardia lamblia*, which causes an enteritis.

- **Worms** are not always microscopic in size but, pathogenic worms do cause infection and some can spread from person to person. Examples include: threadworm and tapeworm.

- **Prion** are infectious protein particles. Example: New Variant Creutzfeldt-Jakob Disease
3.0 THE SPREAD OF INFECTION

3.1 It is important to remember that the one feature that distinguishes infection from all other disease is that it can be spread, i.e. one person can ‘catch’ it from another or via a vector (crawling and flying insects).

3.2 It is convenient to classify the modes of spread of infection as follows:

- **Direct Contact**: Direct spread of infection occurs when one person infects the next by direct person-to-person contact. Sexually transmitted diseases are obvious examples.

- **Inhalation**: Inhalation spread occurs when microbes exhaled or discharged into the atmosphere by an infected person are inhaled by and infect another person. The common cold and influenza are often cited as examples, but it is likely that hands and fomites (inanimate objects) are also important in the spread of respiratory viruses.

- **Ingestion**: Infection can occur when organisms capable of infecting the gastrointestinal tract are ingested. When these organisms are excreted faecally by an infected person, faecal-oral spread is said to occur. Organisms may be carried on fomites, hands or in food and drink e.g. Hepatitis A, salmonella, campylobacter.

- **Inoculation**: Inoculation infection can occur following a “sharps” injury when blood contaminated with, for example Hepatitis B virus, is directly inoculated into the blood stream of the victim, thereby causing an infection. Bites from humans can also spread infection by the inoculation mode.

- **Indirect**: Indirect spread of infection is said to occur when an intermediate carrier is involved in the spread of pathogenic microbes from the source of infection to another person. e.g.

  - **Fomite**: A fomite is defined as an object, which becomes contaminated with infected organisms and which subsequently transmits those organisms to another person. Examples of potential fomites are bedpans, urinals, thermometers, oxygen masks or practically any inanimate article.

  - **Hands**: The hands of health and social care workers are probably the most important vehicles of cross-infection. The hands of patients can also carry microbes to other body sites, equipment and staff.

  - **Air**: Aerosol spread of infection undoubtedly occurs causing inhalation spread e.g. Chickenpox, Mumps and Measles.
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- **Vectors:** Crawling and flying insects are an obvious example of intermediate carriers and need to be controlled. Insect bites may also cause infections such as malaria.

### 4.0 PORTALS OF ENTRY FOR INFECTION

4.1 In order to cause disease a pathogen must have a way to enter the body - a portal of entry. To transmit to another host it must be able to leave the body via a portal of exit. The route of entry and exit may be different, for example enteric infections enter the mouth and leave in the faeces, or they may be the same; for example respiratory tract infections.

4.2 Micro-organisms use a range of different routes to find new hosts and one microbe may be able to spread by using more than one method.

4.3 A broad distinction can be made between transmission through direct physical contact with an infected individual and transmission indirectly on other vehicles, objects and equipment.

4.4 This diagram indicates the portals of entry:

- **Inhalation**
  Small particles of dust or droplets of water carry microbes into the respiratory tract via the mouth or nose (e.g. influenza, measles, and tuberculosis)

- **Inoculation**
  Microbes may be introduced via skin and mucous membranes by accidental injury, bites, during surgical incision or via artificial devices (e.g. hepatitis B, malaria)

- **Transplacental**
  Microbes may cross the placenta from the maternal to the foetal circulation to cause congenital infection (e.g. rubella, syphilis)

- **Ingestion**
  Microbes enter the gastrointestinal tract with contaminated food or water (e.g. salmonella cholera, polio)

- **Sexual intercourse**
  Microbes may be transferred from the genital tract of one partner to the other during sexual intercourse (e.g. gonorrhoea, herpes simplex)
5.0 COMMON INFECTIOUS DISEASES

5.1 Please see Appendix 1 which describes the most common infectious diseases and there:
- Incubation period
- Method of spread
- Period of infectivity
- Exclusion period
- Management of contacts

5.2 Advice on treatment should be sought from a medical practitioner or the Infection Prevention and Control Nurses or contact:

   Public Health England on 0300 303 8537

6.0 TRANSMISSIBLE SPONGIFORM ENCEPHALOPATHIES

6.1 Transmissible spongiform encephalopathies (TSE's), sometimes known as prion diseases, are fatal degenerative diseases, which occur in humans and other animals. A common feature is the appearance of microscopic vacuoles (holes) in the grey matter of the brain, giving a sponge-like appearance.

6.2 TSE's include the following: Creutzfeldt-Jacob Disease (CJD), new variant Creutzfeldt-Jacob Disease (vCJD), Gerstmann-Straussler-Scheinker (GSS), Fatal Familial Insomnia (FFI), KURU and Alpers Syndrome.

6.3 In addition to these human diseases, prion related diseases have been recognised in several animal hosts, e.g. Bovine Spongiform Encephalopathy (BSE) in cattle.

6.4 TSE's are uniquely different from other microbiological diseases is that:
- The mode of transmission is via a protein particle called a prion.
- These prions are unusually resistant to conventional decontamination methods.
- Are not highly contagious but may be transmitted to patients via contaminated medical instruments, pituitary hormones, corneal or dura mater grafts obtained from an infected patient.
- Have no known treatment or prophylaxis.
- 95% of TSE’s are CJD.

6.5 Transmission of TSE
Prions do not behave like bacteria and viruses and much is still unknown about how TSE’s are transmitted. Prions are not uniformly distributed throughout the body. Certain tissues are thought to be more infectious than other, in general, and at the later stages of the illness. This is summarised below.
CJD – Probable Infectivity of Human Tissue and other Body Substances

<table>
<thead>
<tr>
<th>High Risk</th>
<th>Medium Risk</th>
<th>Low Risk</th>
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</thead>
<tbody>
<tr>
<td>Neural tissue</td>
<td>Anterior eye and cornea</td>
<td>Peripheral nerves</td>
</tr>
<tr>
<td>- brain</td>
<td>Olfactory epithelium</td>
<td>Blood and bone marrow</td>
</tr>
<tr>
<td>- spinal cord</td>
<td>Tonsil (vCJD)</td>
<td>Tonsil</td>
</tr>
<tr>
<td>- spinal ganglia</td>
<td>Appendix (vCJD)</td>
<td>Appendix</td>
</tr>
<tr>
<td>- dura mater</td>
<td>Spleen and Thymus</td>
<td>Spleen and Thymus</td>
</tr>
<tr>
<td>- cranial nerves</td>
<td>(vCJD)</td>
<td>Other Lymphoid tissue</td>
</tr>
<tr>
<td>- cranial ganglia</td>
<td>Other lymphoid tissue</td>
<td>Dental pulp</td>
</tr>
<tr>
<td>Posterior eye</td>
<td></td>
<td>Gingival Tissue</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CSF</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Urine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Other tissue</td>
</tr>
</tbody>
</table>

In addition the kidney, lung, pituitary gland and placenta are also or assumed to be infectious.

6.6 There is no evidence at present that saliva, body secretions or excreta are infectious. These conditions are not transmitted by close person-to-person contact.

6.7 Types of CJD

**Sporadic CJD**
- Cause unknown.
- Usually occurs in middle age.
- Rapid onset of disease (within one week).
- Early features include: **memory loss, cognition impairment and incoordination**.
- Other symptoms include: **involuntary movements, loss of ability to walk and speech impairment**.
- Illness duration less than 6 months.

**Iatrogenic CJD**
- Result of: neurosurgery (including use of EEG depth leads), corneal grafting, human dura mata implants and exposure and use of human pituitary gonadotrophin (hGNH).
- Very rare.
- Symptoms vary depending on route of transmission.
- hGNH – causes **unsteadiness with late onset dementia**.
- Dura mata grafts – **rapid onset dementia** with other neurological features. Possibly indistinguishable from sporadic cases.
- Investigations to include history of treatments.

**Variant CJD**
- Caused by BSE agent, probably with contaminated food.
- Affects younger age group (median 28 years).
- Slower progression of symptoms.
- Symptoms include first **psychiatric symptoms, then behaviour changes**.
- Other symptoms include **anxiety, agitation, delusions and hallucinations**.
- **Dementia, together with other neurological features develop towards the end of the disease**.
- Illness duration lasts approximately 14 months.
**Genetic CJD**

Caused by genetic mutation in prion protein gene. Children of infected people have 50% of inheriting the abnormal disease-causing gene. Cases have been identified where there is no obvious history. *Symptoms similar to Sporadic CJD.* Disease progression slower and age onset younger.

### 6.8 Management and Control

Where CJD or related disorders are suspected/known:

- Inform: Service area Infection Prevention and Control Team
- Inform: Public Health England on 0300 303 8537

### 6.9 Patients with CJD or vCJD can be cared for in an open ward, using standard precautions. Evidence to date suggests that normal social or routine clinical contact such as washing does NOT present a risk to patients, relatives or healthcare workers.

### 6.10 It is not necessary for the patient to be isolated, unless appropriate for privacy.

### 6.11 Minimise invasive procedures and involve minimum numbers of staff if performing invasive procedures. Only trained staff, aware of the hazards, should carry out invasive procedures that may lead to contact with infectious tissue (e.g. venepuncture, lumbar punctures). SINGLE-USE instruments ONLY must be used.

### 6.12 Linen from patients who have TSE, or are at high risk of having TSE, is handled according to existing laundry guidelines.

### 6.13 Waste generated on the hospital ward is unlikely to contain high risk material and should be disposed of into a tiger stripe waste bag.


### 6.15 Labelling of specimens:

All TSE’s are categorised as hazard group 3 organisms. Blood samples, cerebro-spinal fluid and tissue from patients who are identified in the table above must be labelled as ‘Danger of Infection’. The laboratory should be contacted for advice before specimens are sent.

### 6.16 Care of deceased patients

Care of a deceased patient, suspected/diagnosed with TSE, is as follows:

- Standard precautions apply.
- Place deceased in **BODY BAG** *(not standard envelope)*
- Complete notification sheet: See [Appendix 2](#)
6.17 Relatives of the deceased may wish to view the body. This may involve touching or kissing the face and such superficial contact is permitted.

6.18 Embalming procedures should be avoided.

6.19 It is not necessary to discourage burial of the body - there are no special arrangements for burial or cremation

6.20 If a post-mortem examination is to be carried out, the Consultant Histopathologist will initiate transfer arrangements to a mortuary with facilities for performing specialist procedures.

6.21 Confidentiality regarding diagnosis must be maintained at all times. (Undertakers should follow their own professional universal precaution guidelines when handling patients in BODY BAGS).

7.0 GUIDELINES FOR THE MANAGEMENT OF INFECTIONS ASSOCIATED WITH SPECIFIC ALERT ORGANISMS

7.1 This section relates specifically to:
- Glycopeptide resistant enterococci (GRE)
- Multiresistant Acinetobacter spp.
- Viruses causing Viral Haemorrhagic Fever

7.2 Infections caused by these agents are expected to occur mainly within the acute hospital setting, where patients are generally more physically unwell and susceptible to contracting serious opportunistic or healthcare-associated infections.

7.3 Taking local epidemiology and risk into account, it is considered unlikely that such infectious conditions would ever present problems requiring cross-infection precautions within the community services and mental health care environment. Should this occur, discussions with colleagues in Infection Prevention and Control in acute medical facilities and the local Public Health England Team will take place, and the patient will be managed and treated in a timely fashion within the most appropriate care setting.

8.0 GUIDELINES FOR THE MANAGEMENT OF INFECTIONS ASSOCIATED WITH EXTENDED SPECTRUM BETA LACTAMASE

8.1 What does ESBL mean?
ESBL stands for extended-spectrum beta-Lactamase. These are enzymes produced by many species of bacteria which destroy and make them resistant to, some of the most widely used antibiotics, e.g. cephalosporins. This can cause problems when treating infected patients as there are only 2 oral antibiotics (e.g. nitrofurantoin) and a limited group of IV antibiotics that remain effective. This can complicate and/or delay appropriate treatment.
8.2 ESBLs were first described in mid 1980s -1990s and were found mostly in Klebsiella species, in hospitals treating the most vulnerable patients. Since 2003 a new class (CTX-M enzymes) has emerged, found in Escherichia coli bacteria, most often causing urinary tract infections, which are also resistant to penicillin’s. This can progress to the serious illness, septicaemia. ESBLs have been found in the community as well as in hospitals although “community acquired” infections may have had previous contact with hospitals.

8.3 How are they spread?
There is some evidence suggesting they can be found in the faeces of farm animals as well as some humans. This means that it is possible that transmission may occur from contamination of food, e.g. raw meat and by bacteria from animal faeces, leading to infections in humans.

8.4 It is also possible that these bacteria are passed from person to person on contaminated hands (of patients or health care workers) or by poor practice in urinary catheter care.

8.5 Spread is made easier if the bacteria normally present in the gut (and which help protect against invasion by other strains) are killed by taking antibiotics. Use of some newer antibiotics appears to predispose patients to infection with ESBL producing bacteria which may explain why this has become an issue now.

8.6 Who gets ESBL producing bacteria?
The majority of those with an ESBL producing strain are elderly and unwell with another underlying medical condition. Often these patients have had multiple courses of antibiotics for repeated infections and have been in hospital

8.7 It is possible that the ESBL producing bacteria are acquired months or even years before they cause infection. They live harmlessly in the gut until the patient becomes ill and requires antibiotics.

8.8 How can we control spread?
Effective control measures are less well understood than for other types of antibiotic resistant bacteria for example MRSA.

8.9 Until we know more about how to control these bacteria, the following are sensible precautions to take:

- It should be ensured that hand washing and other infection control procedures are rigorously enforced.
- Encourage regular assessment of urinary catheter use; if the patient no longer has a clinical need for a urinary catheter, remove it.
- Antibiotics should only be prescribed when needed, in the right dose, for the right duration, so as to reduce resistance developing in bacteria.
In some circumstances patients with ESBL producing bacteria will be isolated whilst in the acute hospital.

8.10 **Can a person be cleared of an ESBL producing strain?**
Sometimes the strain will be lost naturally. In those with serious illnesses, ESBL producing strains may be present for months or even years. Use of antibiotics probably does not help; they can treat infections but do not necessarily eliminate the bacteria from the body especially if there are some in the gut.

8.11 **Treating a patient with ESBL infection.** Due to the infrequency and complexity of such cases arising, management requires discussion and liaison with the Infection Prevention and Control Team.

### 9.0 CARBAPENEMASE-PRODUCING ENTEROBACTERIACEAE (CPE)

9.1 Enterobacteriaceae are bacteria that usually live harmlessly in the gut of humans. This is called ‘colonisation’ (a person is said to be a ‘carrier’). However, if the bacteria get into the wrong place, such as the bladder or bloodstream they can cause infection.

9.2 Carbapenems are one of the most powerful types of antibiotics. Carbapenemases are enzymes (protein catalysts), made by some strains of these bacteria, which allow them to destroy carbapenem antibiotics and so become resistant to these and most other penicillin-like (beta-lactam) antibiotics.

9.3 If a person is a carrier of Carbapenemase-producing Enterobacteriaceae, they do not need to be treated. However, if the resistant bacteria cause an infection then treatment to manage the infection e.g. antibiotics and sometimes other treatment e.g. wound management, will be required.

9.4 If a person is a carrier, the bacteria can get into the environment when poor hygiene leads to faecal contamination, allowing spread to other people. Where hygiene is poor, the bacteria can be passed on by carers or by the affected individual having direct contact with others. Therefore, good personal hygiene including hand hygiene by carers and the affected individual (particularly after visiting the toilet) is important. Keeping the environment scrupulously clean is also an important measure in preventing spread.

9.5 Most people will be unaware that they are a carrier and, for most healthy carriers, the chance of developing an infection with the bacteria is low. However, immunocompromised individuals and those receiving complex and intensive hands-on care in the community (very often with frequent visits to hospital) will be more vulnerable. This means that they are at greater risk of becoming a carrier in the first place and potentially, being vulnerable, suffering more serious consequences if they develop an infection.
Therefore, ‘CPE’ pose two main risks:

- Firstly, to an individual, who may develop a serious infection needing more intensive treatment in hospital to clear (e.g. antibiotics intravenously). There are few antibiotics which work against most CPE strains.
- Secondly, if the levels of hygiene in the community setting are inadequate, then the resistant bacteria may spread among individuals who congregate together, e.g. in care homes.

Therefore, it is especially important that consistent good hygiene is practiced and maintained by staff, service users and visitors, at all times.

Treating a patient with CPE infection. Due to the infrequency and complexity of such cases arising, management requires discussion and liaison with the Infection Prevention and Control Team.

**GUIDELINES FOR THE SURVEILLANCE OF HEALTH CARE ASSOCIATED INFECTIONS (HCAIS)**

The purpose of this guidance is to describe the procedures for the systematic gathering of information about HCAIs within in-patient wards.

This guidance outlines why surveillance is needed and how data on HCAI within the organisation will be collected, stored, analysed, reported and reviewed.

**Introduction**

Surveillance is the cornerstone of all infection control activities, enabling the identification of priorities for intervention.

The Infection Prevention and Control Team conducts continuous, priority driven, targeted surveillance. It is an active method of detecting, reporting and analysing information concerning HCAI in patients and staff.

**Definition**

Surveillance of HCAI is defined by the Public Health England as “the continuous monitoring of the frequency and the distribution of disease and death in patients and staff, due to infections that can be transmitted from human to human or from animals, food, water or the environment to humans, and the monitoring of risk factors for those infections”.

This implies:

- A precise definition of the events to be surveyed
- Systematic collection of data
- Analysis and interpretation of data
- Dissemination of the results to those who need to know, so that appropriate action can be taken.
10.7 The objectives of HCAI surveillance are:
- The assessment of infection levels over time in order to determine the need for and measure the effect of, preventable or control measures
- The prevention and early detection of outbreaks in order to allow timely investigation and control

10.8 Surveillance also allows the IPCT to prioritise infection control activities by identifying areas with a higher risk of infection where HCAI could be reduced by appropriate control measures and thereby improve the quality of patient care.

10.9 Data Collection
In most instances laboratory data forms the basis of surveillance.

10.10 Patients with invasive devices are screened for MRSA (with the patient’s permission). Individual risk assessments are carried out for each patient to ensure the patient is treated in a timely and appropriate manner.

10.11 There are certain conditions in which clinical syndromes are surveyed e.g. outbreaks of diarrhoea and vomiting and in these cases information gathering is dependant on reporting by clinical staff.

10.12 Organisms with specific relevance to infection control are designated as ‘Alert organisms’

10.13 ALERT ORGANISMS AND CONDITIONS
Bacterial isolates with unusual anti-microbial resistance
Beta Lactamase producing enterococci
Campylobacter enteritis
Clostridium difficile – See ICPG 6
Cryptosporidiosis
ESBLs (extended spectrum Beta lactamase producers)
Gentamicin – Resistant ‘coliforms’
Group A Streptococcal infections
Influenza - See ICPG 4
Legionella sp.
Methicillin resistant staphylococcus aureus - See ICPG 5
Mycobacteria sp
Norwalk Virus (Norovirus) – See ICPG 4
Pseudomonas sp.
Penicillin – Resistant gonococci
Penicillin resistant Strep. Pneumoniae
Pseudomonas aeruginosa
Rotavirus
Respiratory syncytial virus (RSV)
Salmonella spp.
Scabies – See ICPG 8
Severe soft tissue infections
Shigella species
Streptococcus pyogenes
Suspected infective diarrhoea/vomiting – See ICPG 4
Vancomycin Resistant enterococci
Varicella-zoster
Vero-toxin producing strains of *E. coli
Whooping cough

10.14 When one of these organisms is identified in an in-patient, the result is sent to the in-patient unit from the reporting laboratory. Receipt of a positive isolate should initiate any action required; advice can be sought from the IPCT.

10.15 In the event of an MRSA bacteraemia result, the IPCT will complete the Post Infection Review form with input from the relevant clinical staff involved in the care of the patient presenting with the bacteraemia.

### 11.0 REFERENCES


END