Infection Prevention and Control Assurance Standard Operating Procedure 23 (IPC SOP 23)

Alert Organisms – Transmissible Spongiform Encephalopathies (TSEs) e.g. Creutzfeldt-Jakob Disease (CJD)

Why we have a procedure?

To ensure employees of the Black Country Partnership NHS Foundation Trust have a standard procedure to follow when caring for patients affected by Transmissible Spongiform Encephalopathies (TSEs), to ensure that effective measures are in place to prevent patients, staff and others being put at risk of contracting TSEs, as a consequence of healthcare delivered by the Trust (TSEs are also known as prion diseases).

The Health and Social Care Act 2008: Code of Practice for the NHS for the Prevention and Control of Healthcare Associated Infections (revised January 2015) stipulates that NHS bodies must, in relation to preventing and controlling the risk of Health Care Associated Infections (HCAI), have in place appropriate core policies/procedures. Implementation of this procedure will contribute to the achievement and compliance with the Act.

What overarching policy the procedure links to?

- This procedure is supported by the Infection Prevention and Control Assurance Policy

Which services of the trust does this apply to? Where is it in operation?

<table>
<thead>
<tr>
<th>Group</th>
<th>Inpatients</th>
<th>Community</th>
<th>Locations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mental Health Services</td>
<td>✓</td>
<td>✓</td>
<td>all</td>
</tr>
<tr>
<td>Learning Disabilities Services</td>
<td>✓</td>
<td>✓</td>
<td>all</td>
</tr>
<tr>
<td>Children and Young People Services</td>
<td>x</td>
<td>✓</td>
<td>all</td>
</tr>
</tbody>
</table>

Who does the procedure apply to?

This document applies to all staff employed by or working on behalf of the Trust caring for patients as part of their role and job description.

When should the procedure be applied?

Effective prevention and control of healthcare associated infection (HCAI) must be embedded into everyday practice and applied consistently. This procure should be applied when caring for patients known or suspected to have TSEs.
Additional Information/ Associated Documents

- Infection Prevention and Control Assurance Policy
- Hand Hygiene Policy
- Infection Prevention and Control Assurance - Standard Operating Procedure 1 (IPC SOP 1) - Standard Infection Control Precautions
- Infection Prevention and Control Assurance - Standard Operating Procedure 2 (IPC SOP 2) - Transmission Based Precautions
- Infection Prevention and Control Assurance - Standard Operating Procedure 3 (IPC SOP 3) - Surveillance of Infection and Data Collection
- Infection Prevention and Control Assurance - Standard Operating Procedure 4 (IPC SOP 4) - Reporting Incidents of Infection to Public Health England and/or the Local Authority
- Infection Prevention and Control Assurance - Standard Operating Procedure 14 (IPC SOP 14) - Undertaking a Patient Infection Risk Assessment
- Infection Prevention and Control Assurance - Standard Operating Procedure 16 (IPC SOP 16) Sharing Information with other Health and Social Care Providers
- Infection Prevention and Control Assurance - Standard Operating Procedure 18 (IPC SOP 18) - Post Infection Review (PIR)
- Infection control of CJD, vCJD and other human prion diseases in healthcare and community settings. (Feb 2015), GOV.UK
- Part 4 of the above document HTTPS://ASSETS.PUBLISHING.SERVICE.GOV.UK/GOVERNMENT/UPLOADS/SYSTEM/UPLOADS/ATTACHM
T_DATA/FILE/427854/INFECTION_CONTROLV3.0.PDF

Aims
To reduce the risk of transmission of infection by ensuring that Trust staff are:

- Alert to the risks of individual patients carrying or being infected by TSEs
- Alert to the risks of transmission of TSEs
- Undertaking a post infection review (PIR) on patients with TSEs to review contributory factors and lessons learned when deemed necessary by the Infection Prevention and Control Team
- Informing other healthcare providers of the patients infectious status when any transfers of care are planned either internally within the Trust or to external acute care providers
Transmissible Spongiform Encephalopathies (TSEs), also known as prion diseases, are a group of rare degenerative brain disorders characterized by tiny holes that give the brain a "spongy" appearance. These holes can be seen when brain tissue is viewed under a microscope. The human TSEs have a pre-clinical phase which may last for many years (when they do not show any symptom of disease). Once clinical signs appear there is no known treatment or prophylaxis.

Research suggests that TSEs are caused by an abnormal version of a protein called a prion (prion is short for proteinaceous infectious particle). Prion proteins occur in both a normal form, which is a harmless protein found in the body's cells, and in an infectious form, which causes disease. The harmless and infectious forms of the prion protein are nearly identical, but the infectious form takes on a different folded shape from the normal protein.

Human TSEs can occur three ways: sporadically; as hereditary diseases; or through transmission from infected individuals:
Sporadic TSEs may develop because some of a person's normal prions spontaneously change into the infectious form of the protein and then alter the prions in other cells in a chain reaction.

Inherited cases arise from a change, or mutation, in the prion protein gene that causes the prions to be shaped in an abnormal way. This genetic change may be transmitted to an individual's offspring.

Transmission of TSEs from infected individuals is relatively rare. TSEs cannot be transmitted through the air or through touching or most other forms of casual contact. However, they may be transmitted through contact with infected tissue, body fluids, or contaminated medical instruments. Normal sterilization procedures such as boiling or irradiating materials do not prevent transmission of TSEs.

Symptoms of TSEs vary, but they commonly include:

- Personality changes
- Psychiatric problems such as depression
- Lack of coordination, and/or an unsteady gait

Patients also may experience involuntary jerking movements called myoclonus, unusual sensations, insomnia, confusion, or memory problems. In the later stages of the disease, patients have severe mental impairment and lose the ability to move or speak.

Creutzfeldt-Jakob disease (CJD) is the most well-known of the human TSEs. It is a rare type of dementia that affects about one in every one million people each year, CJD accounts for 95% of all TSEs. TSEs tend to progress rapidly and usually culminate in death over the course of a few months to a few years.

For almost all routine clinical contacts no special precautions are required for the care of patients with or identified as being at risk of developing CJD/vCJD.

Diagnostic criteria can be seen in Appendix 1.

Types of CJD and Routes of Transmission

There are four types of CJD, not all types have the same disease characteristics and are not necessarily transmitted in the same way. In addition their infectivity varies at different phases of disease. The risk of transmission is dependent on the type of procedure undertaken and the type of tissue involved.

To prevent transmission of TSEs it is necessary to take a two stage risk based approach. First patients affected with or at risk of developing a TSE must be reliably identified, and second measures must be taken to ensure that any invasive device used on such patients are not re-used on other patients.

Types of CJD

Classical or sporadic CJD

This is the most common type and its cause is unknown (85-90% of cases). It is not thought to be found outside the brain. Subsequent transmission of the prion is thought to be instruments which have been contaminated by brain tissue or by the transplant of tissue itself e.g. Dura mater.
### Familial CJD
This type is very rare and is caused by an inherited abnormality in the gene that produces normal protein (10-15% of cases). In the majority of cases this is known within the family because of family history. Subsequent transmission is the same as for sporadic CJD.

### Iatrogenic CJD
Caused by medical procedures such as injections with human pituitary hormones, Dura mater grafts, neurosurgical instruments and blood/blood products (less than 1% of cases).

CJD patients typically present with rapidly progressive dementia, usually accompanied by Myoclonus and cerebellar ataxia. Most patients die within 4 months of disease onset, in a mute and immobile state.

### Variant CJD (vCJD)
This was first recognised in 1996 and generally affects young adults. It is associated with the same transmissible agent that causes Bovine Spongiform Encephalopathy (BSE) in cattle. Primary infection is thought to be caused by consuming BSE contaminated food products. This prion is found throughout the body in the lymphatic tissue and central nervous system. Transmission may occur via instruments contaminated with infective tissue and following transfusion of contaminated blood or blood products.

The median duration of illness in vCJD is longer than that for sporadic CJD (14 months compared with 4 months). Death in an immobile and mute state is a typical outcome.

### Categorisation of Patients by Risk

<table>
<thead>
<tr>
<th>Patient Groups</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Symptomatic patients</strong></td>
</tr>
<tr>
<td>• Patients who fulfil the diagnostic criteria for definite, probable or possible CJD</td>
</tr>
<tr>
<td>• Patients with neurological disease of unknown aetiology, who do not fit the criteria for possible CJD, but where the diagnosis of CJD is being actively considered</td>
</tr>
<tr>
<td><strong>Patients “at increased risk” from genetic forms of CJD</strong></td>
</tr>
<tr>
<td>• Individuals who have been shown by specific genetic testing to be at significant risk of developing CJD</td>
</tr>
<tr>
<td>• Individuals who have a blood relative known to have a genetic mutation indicative of genetic CJD</td>
</tr>
<tr>
<td>• Individuals who have or have had two or more blood relatives affected by CJD or other prion disease</td>
</tr>
</tbody>
</table>
### Patients “at increased risk” of vCJD through receipt of blood from a donor who later developed vCJD

- Individuals who have received labile blood components (whole blood, red cells, white cells or platelets) from a donor who later went on to develop vCJD

### Patients identified as “at increased risk” of CJD through iatrogenic exposures

- Recipients of hormone derived from human pituitary glands, *e.g.* growth hormone, gonadotrophin, are “at increased risk” of transmission of sporadic CJD. In the UK the use of human-derived gonadotrophin was discontinued in 1973, and use of cadaver-derived human growth hormone was banned in 1985. However, use of human-derived products may have continued in other countries after these dates

- Individuals who underwent intradural brain or intradural spinal surgery before August 1992 who received (or might have received) a graft of human-derived Dura mater are “at increased risk” of transmission of sporadic CJD (unless evidence can be provided that human-derived Dura mater was not used)

- Individuals who have had surgery using instruments that had been used on someone who went on to develop CJD, or was “at increased risk” of CJD

- Individuals who have received an organ or tissue from a donor infected with CJD or “at increased risk” of CJD

- Individuals who have been identified as having received blood or blood components from 300 or more donors since January 1990

- Individuals who have given blood to someone who went on to develop vCJD

- Individuals who have received blood from someone who has also given blood to a patient who went on to develop vCJD

- Individuals who have been treated with certain implicated UK sourced plasma products between 1990 and 2001

### Transmission

TSEs including CJD/vCJD, exhibit unusual and extreme resistance to conventional decontamination methods such as heat (e.g. autoclaving) or chemical disinfection. Therefore, transmission may occur if a surgical instrument or flexible endoscope which has been in contact with infected tissue is subsequently re-used on another patient. (The level of infectivity of various tissues is outlined in Appendix 3).

All patients undergoing surgical or endoscopic procedures in a hospital/surgery involving medium or high risk tissue must be assessed for their risk of having CJD/cud, or of increased risk of developing CJD/cud, prior to their procedure being undertaken, in order to prevent such transmission taking place.
Any patient identified as confirmed, probable or possible CJD/cud must undergo further assessment by the Consultant Microbiologist/Infection Prevention and Control Team regarding the level of tissue infectivity involved and the procedure to be undertaken.

**Risk Factors for Transmission**
The use of instruments and endoscopes (e.g. as used in acute healthcare) may pose a risk of transmission of CJD. These procedures have been classified as follows based on the risk of transmission:

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>High-risk procedures</strong></td>
<td>Procedures that involve handling of tissue considered to be at high risk of transmission of CJD. High-risk procedures are intradural neurosurgical operations on the brain (excluding operations on the spine and peripheral nerves), neuroendoscopy and posterior eye procedures that involve the retina or optic nerve</td>
</tr>
<tr>
<td><strong>Medium-risk procedures</strong></td>
<td>All procedures on tonsils, spleen, lymphoid tissue, spinal cord, anterior eye and peripheral nerves</td>
</tr>
<tr>
<td><strong>Low-risk procedures</strong></td>
<td>All procedures other than the high and medium risk procedures</td>
</tr>
</tbody>
</table>

**Identifying Patients at Risk of CJD/vCJD**
In order to prevent the transmission of CJD / vCJD in the healthcare setting it is of the utmost importance that all patients are assessed for risk factors according to Department of Health criteria.

All patients about to undergo any elective or emergency surgical or endoscopic procedure (excluding arthroscopy) should be asked the following questions at the earliest opportunity:

- “Have you ever been notified that you are at increased risk of CJD or vCJD for public health purposes?”
- “Have you received multiple (300 or more donors) transfusions of blood or blood components (red cells, cryoprecipitate or platelets) or plasma (150 or more donors) since January 1990”

Patients suspected of having CJD or vCJD should be referred to a neurologist. The National CJD Surveillance Unit must also be informed of all suspect cases.

**Care of CJD Patients in the Hospital or Community**

There is no evidence that normal social or routine clinical contact with a CJD patient presents a risk to healthcare workers, relatives or others.

- In hospital isolation of patients with CJD is not necessary, and they can be nursed in an open ward using standard infection prevention and control precautions in line with those used for all other patients [see Infection Prevention and Control Assurance - Standard Operating Procedure 1 (IPC SOP 1) - Standard Infection Control Precautions]
- There is no evidence of infectivity in saliva, body secretions or excreta. Any potential exposure to these body fluids should be handled in line with the standard infection control precautions procedure [see Infection Prevention and Control Assurance - Standard Operating Procedure 1 (IPC SOP 1) - Standard Infection Control Precautions]
The agents of CJD/vCJD are classified as Hazard Group 3 pathogens by the Advisory Committee on Dangerous Pathogens. For this reason all diagnostic specimens from known or at risk patients must be labelled with a ‘risk of infection’ sticker – seek advice from the Consultant Microbiologist.

When certain invasive procedures are performed there is the potential for exposure. Although there is no evidence of any transmission it is essential that control measures are in place to prevent iatrogenic transmission, through good standard infection control practices, with the control of sharps and protective equipment to prevent splashing and droplet contamination of mucosal surfaces.

Normal standard infection control precautions should be followed to clean up spillages, including spillages of blood and cerebrospinal fluid [see Infection Prevention and Control Assurance - Standard Operating Procedure 7 (IPC SOP 7) - Decontamination - Cleaning, Disinfection and Sterilisation].

These should be cleaned immediately. Potentially infectious material should be removed using disposable absorbent material and any waste, including cleaning tools such as mop heads, disposed of as infectious waste for incineration. Disposable gloves and apron should be worn when removing spillages, eye protection should be worn for procedures where splashing may occur. Advice should be sought from the Infection Prevention and Control Team (IPCT) and the Consultant Microbiologist.

Late stage CJD patients may experience tissue breakdown and the development of pressure sores. These lesions should be dressed regularly and contaminated dressings disposed of as clinical waste – N.B. yellow clinical waste bags must be used.

*Within the Trust high risk procedures using surgical instruments/endoscopes etc. do not generally occur therefore specialist care relevant to the use, decontamination, quarantine and disposal of these has not been covered in this procedure but advice can be sought from the Infection Prevention and Control Team.*

**Death of a Patient with Known or Suspected CJD/vCJD**

On the death of a patient with risk factors as detailed in the table in 2.2 of this procedure, the removal of the deceased from the ward/community setting should be carried out using standard infection control precautions [see Infection Prevention and Control Assurance - Standard Operating Procedure 1 (IPC SOP 1) - Standard Infection Control Precautions].

National guidelines recommend that patients with CJD/vCJD or at risk of CJD/vCJD, need to be placed in a cadaver bag (body bag) and to have labels attached to both the deceased and the outside of the cadaver bag stating “Risk of Infection”, prior to transportation to the mortuary/undertakers.

Viewing the deceased by relatives, friends or carers can take place as required. Such viewing and superficial contact such as touching or kissing need not be discouraged even if a post-mortem has taken place. Body bags may be rolled down to allow superficial contact.

If a post mortem is required for anyone who falls into the ‘risk groups’, arrangements should be made for the body to be removed to a regional neuropathology centre where specialist expertise exists. Liaison between local pathologists and the National CJD Surveillance Unit is preferable. The Infection Prevention and Control Team or Consultant Microbiologist can provide further information as/when required.
Where there is a possibility that a patient could have been exposed to CJD/vCJD through contaminated surgical instruments that were previously used on a patient with or at increased risk of CJD/vCJD the infection prevention and control team must be informed. A “look-back” exercise may be required. Advice will be given by the IPCT and the local Public Health England office.

The Infection Prevention and Control Team may recommend that a Post Infection Review (PIR) is completed to identify any critical points and contributory factors, and determine whether any preventative action(s) and improvement action(s) can be undertaken to reduce or control incidents of HCAI [See Infection Prevention and Control Assurance - Standard Operating Procedure 18 (IPC SOP 18) - Post Infection Review (PIR) and Infection Prevention and Control Assurance - Standard Operating Procedure 4 (IPC SOP 4) - Reporting Incidents of Infection to Public Health England and/or the Local Authority]

**Where do I go for further advice or information?**

- Infection Prevention and Control Team
- Your Service Manager, Matron, General Manager, Head of Nursing, Group Director
- Your Group Governance Staff

**Training**

Staff may receive training in relation to this procedure, where it is identified in their appraisal as part of the specific development needs for their role and responsibilities. Please refer to the Trust’s Mandatory and Risk Management Training Needs Analysis for further details on training requirements, target audiences and update frequencies.

**Monitoring / Review of this Procedure**

In the event of planned change in the process(es) described within this document or an incident involving the described process(es) within the review cycle, this SOP will be reviewed and revised as necessary to maintain its accuracy and effectiveness.

**Equality Impact Assessment**

Please refer to overarching policy

**Data Protection Act and Freedom of Information Act**

Please refer to overarching policy.
Diagnostic Criteria

1. Symptomatic Patients
Those who fulfil internationally accepted diagnostic criteria set out below for definite, probable and possible CJD/vCJD. Neuropathological / immunocytochemical confirmation is required for a diagnosis of definite sporadic CJD.

2. Sporadic CJD
Probable sporadic CJD patients will have rapidly progressive dementia, and at least two of the following four symptoms:
   a) Myoclonus
   b) Visual or cerebellar problems
   c) Pyramidal or extra pyramidal feature
   d) Akinetic mutism
Plus typical electroencephalogram (EEG) or clinical criteria for possible sporadic CJD (see below) and a positive assay in the cerebrospinal fluid (CSF). Possible sporadic CJD patients will have rapidly progressive dementia, two of the above symptoms (a-d) and duration of less than two years.

3. Latrogenic CJD
Iatrogenic CJD patients display progressive cerebellar syndrome in a pituitary hormone recipient or sporadic CJD with a recognised exposure risk (e.g. Dura mater transplant). A definitive diagnosis of iatrogenic CJD still requires a neuropathological examination.

4. Familial CJD
Patients with familial CJD will have definite or probable CJD (see definitions above), plus definite or probable CJD in a first degree relative (i.e. a parent, child or sibling) or a neuropsychiatric disorder plus a disease – specific mutation in the prion protein gene.

5. Definite vCJD Patients
Will have a progressive neuropsychiatric disorder and neuropathological confirmation of the disease, showing spongiform change and extensive PrPC deposition with florid plaques throughout the cerebrum and cerebellum.

6. Probable vCJD Patients
Can be classified under two sets of criteria:
   a) They will have progressive neuropsychiatric disorder of duration greater than six months where routine investigations do not suggest an alternative diagnosis. They will also have at least four of the following five symptoms:
      1. Early psychiatric symptoms (depression, anxiety, apathy withdrawal, delusions).
      2. Persistent painful sensory symptoms (frank pain and/or unpleasant dysaesthesia.
      3. Ataxia
      4. Monoclonus, chorea or dystonia
      5. Dementia
      6. These patients would have had no history of potential iatrogenic exposure
   b) Alternatively, a probable vCJD patient will have had a progressive neuropsychiatric disorder for a period of longer than six months, where routine investigations do not support an alternative diagnosis, and where there is no history of potential of iatrogenic exposure, plus a positive tonsil biopsy which is positive for PrP-res. Possible vCJD patients will have progressive neuropsychiatric disorder of duration greater than six months where routine investigations do not suggest an alternative diagnosis, and no history of potential iatrogenic exposure. They will also have at least four out of five of the symptoms listed above and an EEG does not show the typical appearance of sporadic CJD or no EEG has been performed.
Pre-procedure Risk Assessment for all Patients about to Undergo Elective or Emergency Surgical or Endoscopic Procedures (Excluding Nasoendoscopy or Cystoscopy without Biopsy)

**N.B these procedures are usually undertaken in an acute Trust setting**

<table>
<thead>
<tr>
<th>Question to Patient</th>
<th>Notes to clinician</th>
</tr>
</thead>
</table>
| • Have you a history of CJD or other prion disease in your family? If yes, please specify. | Patients should be considered to be at risk from genetic forms of CJD if they have or have had:  
  - Genetic testing, which has indicated that they are at significant risk of developing CJD or other prion disease  
  - A blood relative known to have genetic mutation indicative of genetic CJD or other prion disease  
  - 2 or more blood relatives affected by CJD or other prion disease.                                                                                   |
| • Have you ever received growth hormone or gonadotropin treatment? If yes please specify:  
  - Whether the hormone was derived from human pituitary glands  
  - The year of treatment  
  - Whether the treatment was received in the UK or in another country | Recipients of hormone derived from human pituitary glands e.g. growth hormone or gonadotropin, have been identified as at increased risk of sporadic CJD.  
In the UK, the use of human derived growth hormone was discontinued in 1985 but human derived products may have continued to be used in other countries.  
In the UK, the use of human derived gonadotropin was discontinued in 1973 but may have continued in other countries after this time. |
| • Have you ever had surgery on your brain or spinal cord?                              | Patients who underwent intradural neurosurgical or spinal procedures before August 1992 may have received a graft of human-derived Dura mater and should be treated as at increased risk, unless evidence can be provided that human-derived Dura mater was not used.  
Patients who received a graft of human-derived Dura mater before 1992 are at increased risk of transmission of sporadic CJD, but not vCJD. |

All people identified “at increased risk” of developing CJD/vCJD are asked to help prevent any further transmission by:

- Not donating blood  
- Not donating organs or tissues, including bone marrow, sperm, eggs or breast milk  
- Informing whoever is treating them if they are to undergo any medical, surgical or dental procedures  
- Informing family members so that they can pass the information on if required and the person is unable to tell them themselves
## Distribution of TSE Infectivity in Human Tissues and Body Fluids

<table>
<thead>
<tr>
<th>Tissue</th>
<th>Presence of abnormal Prion Protein and level of infectivity</th>
<th>TSE other than vCJD</th>
<th>V CJD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PrP TSE detected</td>
<td>Assumed level of infectivity</td>
<td>PrP TSE detected</td>
</tr>
<tr>
<td>Brain</td>
<td>+ve</td>
<td>High P</td>
<td>+ve</td>
</tr>
<tr>
<td>Spinal cord</td>
<td>+ve</td>
<td>High P</td>
<td>+ve</td>
</tr>
<tr>
<td>Spinal ganglia</td>
<td>+ve</td>
<td>High</td>
<td>+ve</td>
</tr>
<tr>
<td>Dura mater</td>
<td>-ve</td>
<td>High</td>
<td>-ve</td>
</tr>
<tr>
<td>Cranial nerves</td>
<td>+ve</td>
<td>High</td>
<td>+ve</td>
</tr>
<tr>
<td>Cranial ganglia</td>
<td>+ve</td>
<td>High</td>
<td>+ve</td>
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<tr>
<td>Posterior eye</td>
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<td>High (?), NT</td>
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</tr>
<tr>
<td>Anterior eye and cornea</td>
<td>-ve</td>
<td>Medium</td>
<td>-ve</td>
</tr>
<tr>
<td>Olfactory epithelium</td>
<td>+ve</td>
<td>Medium</td>
<td>NT</td>
</tr>
<tr>
<td>Tonsil</td>
<td>-ve</td>
<td>Low</td>
<td>+ve</td>
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<td>Appendix</td>
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<td>Low</td>
<td>+ve</td>
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<tr>
<td>Spleen</td>
<td>+ve</td>
<td>Low P*</td>
<td>+ve</td>
</tr>
<tr>
<td>Thymus</td>
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<td>+ve</td>
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<td>Other lymphoid tissue</td>
<td>-ve</td>
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<td>+ve</td>
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<tr>
<td>Peripheral nerve</td>
<td>+ve</td>
<td>Low</td>
<td>+ve</td>
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<td>Skeletal muscle</td>
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<td>Dental pulp</td>
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<td>Blood and bone marrow</td>
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<td>CSF</td>
<td>-ve**</td>
<td>Low P**</td>
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<td>Placenta</td>
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<td>Low</td>
<td>-ve</td>
</tr>
<tr>
<td>Urine</td>
<td>-ve</td>
<td>Low</td>
<td>-ve</td>
</tr>
<tr>
<td>Other tissues</td>
<td>-ve</td>
<td>Low P*</td>
<td>-ve</td>
</tr>
</tbody>
</table>

**KEY:**

- **+ve** = tested positive
- **-ve** = tested negative
- **NT** = not tested
- **P** = infectivity proven in experimental transmission studies
Standard Operating Procedure Details

<table>
<thead>
<tr>
<th>Unique Identifier for this SOP is</th>
<th>BCPFT-COI-POL-05-23</th>
</tr>
</thead>
<tbody>
<tr>
<td>State if SOP is New or Revised</td>
<td>Revised</td>
</tr>
<tr>
<td>Policy Category</td>
<td>Control of Infection</td>
</tr>
<tr>
<td>Executive Director who portfolio this SOP comes under</td>
<td>Executive Director of Nursing, AHPs and Governance</td>
</tr>
<tr>
<td>Policy Lead/Author Job titles only</td>
<td>Infection Prevention and Control Team</td>
</tr>
<tr>
<td>Committee/Group Responsible for Approval of this SOP</td>
<td>Infection Prevention and Control Committee</td>
</tr>
<tr>
<td>Month/year consultation process completed</td>
<td>July 2019</td>
</tr>
<tr>
<td>Month/year SOP was approved</td>
<td>July 2019</td>
</tr>
<tr>
<td>Next review due</td>
<td>July 2022</td>
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<tr>
<td>Disclosure Status</td>
<td>‘B’ can be disclosed to patients and the public</td>
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Review and Amendment History

<table>
<thead>
<tr>
<th>Version</th>
<th>Date</th>
<th>Description of Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.1</td>
<td>June 2019</td>
<td>Policy reviewed. References on page 2 updated</td>
</tr>
<tr>
<td>1.0</td>
<td>July 2016</td>
<td>New Procedure established to supplement Infection Control Assurance Policy</td>
</tr>
</tbody>
</table>