FAQs re PIM2 fields and diagnoses

Below is a list of Registry-related questions and their responses which have been compiled over a period of time, which may be of use to data collectors and recorders. Most deal with fields related to PIM2, although there are some which relate to diagnosis coding as well. Included also are the Registry definitions for septic shock, chromosomal abnormalities and neurodegenerative disorders. (August, 2009)

Q: Would a critical care PNP count for first contact or just doctor?
A: PNP's would initiate ICU level care therefore include PNP for first contact.

Q: How should we handle start time if we have no record of first face-to-face time?
A: If the time of face to face contact can be assumed (eg Trauma team call or MET call) then use the assumed time. Otherwise if no documentation re face-to-face time, then use PICU admission time

Q: What about the infrequent but possible scenario of a patient being in the ICU but never under the critical care service – should they be omitted if realistically there is no time of first face to face contact with an ICU doctor?
A: Start collecting data with time of ICU admission regardless of whether or not an ICU doctor is following the patient

Q: For SBP should we use arterial measurements in preference to cuff measurements?
A: If there is an arterial line in situ with an appropriate wave form, use the SBP recorded from the arterial line in preference to SBP measured non-invasively.

Q: Should we delete SBPs during agitation or crying?
A: Use the first SBP recorded in the observation chart.

Q: Re ventilation in 1st hour - assume tracheostomy is included in this as long as mechanically ventilated?
A: yes, but tracheostomy & breathing spontaneously does not constitute ventilation

Q – Can a patient have both a PIM high risk and low risk score?
A. Yes, both can be present in an admission record.

Q: Please clarify PIM2 timing requirements for physiological variables in retrievals
A: For retrieved patients, use the 1st measurement recorded after time of first face to face contact with the retrieval team doctor up until 1 hour post admission to ICU. (eg if retrieval takes 3 hours from 1st face to face contact until admission then use the first measurement during the 4 hour period.)

Q: Does T-bar constitute mechanical ventilation?
A: A T-bar or T-piece that is not connected to a ventilator or CPAP device does NOT constitute mechanical ventilation.

Q: Would BiPAP and CPAP be acceptable for FiO2?
A: Yes (mask sealed tightly)

Q: What if the patient has a mixing lesion? Should PaO2 and FiO2 be recorded as 0?
A: Record values measured (not 0)

Q: Re pupil assessment - What about a child that has received atropine during intubation?
A: If one or both pupils were reactive to light before atropine then do NOT code as fixed to light.

Q: Should we qualify the time period for post-cardiac arrest prior to ICU admission under High Risk Diagnoses (i.e. pick some interval within 6, 24, or 96 hours) Example – patient has cardiac arrest at another hospital and transfers the next day or patient has cardiac arrest out of the hospital and takes 8 hours to be transported.
A: Cardiac arrest is relevant if the admission to ICU was related to arrest and all related codes should be used

Q: Many of our surgical patients do not use the Pediatric Critical Care team at all...for example our cardiovascular team would be primary docs with anesthesia running the vent. The PIM 2 asks for that first face-to-face contact with the ICU Doctor. Can I therefore assume that the primary surgeon would be that ICU doc?
A: Yes

Q: And if so, many of them write orders prior to them leaving the recovery room or operating room, and never get to our floor until the next day for rounds. So what time and date would I put for our first doctor face-to-face meeting? Would it be the start of surgery, end of surgery, or something else? And if it's the surgical time, then what SBP would I put on my PIM 2....the first ICU SBP or the first one of the operating room or recovery room?
A: First contact would be on arrival in PICU. The definition of first face to face contact aims to standardize the time PICU therapy commences which is particularly important if PICU staff commence PICU management outside the PICU eg in the emergency department or on a retrieval. For patients admitted from the operating room the standardized time ICU management commences is the time of arrival in the PICU.

Q: If an open heart surgery patient comes from the OR and is intubated, should I mark that his main reason to be in the ICU is to recover from surgery or should I assume that because he might be haemodynamically unstable as well as his inability to regulate his respiratory status, that he his surgery is NOT the main reason why he's in the ICU?
A: Recovery from surgery is principal reason for admission.

Q: About the definition of cardiac arrest – Is that requiring compressions so a respiratory arrest would not be counted?
A: Correct

Q: If a patient is breathing room air and you have a PaO2 for them- shouldn't you enter .21 for FiO2 - or is it strictly only via ETT? Example - A patient has pneumonia and you are drawing PaO2s but they are breathing on their own with no supplemental O2.
A: Record 0.21 for FiO2

Q: RE the 3rd High Risk Diagnosis (Leukemia or lymphoma after 1st induction): should malignancy after first induction be included or just leukaemia or lymphoma?
A: Code only or leukemia or lymphoma after 1st induction

Q: Clarification between genetic and chromosomal disorder
A: There are many disorders known to have a genetic basis that have a normal result to chromosome testing – eg cystic fibrosis. A chromosomal disorder is one where an abnormality is detected on chromosome testing (see Appendix 2 for further details)

Q: PIM high risk code 9 Neurodegenerative disorders – examples?
A: See Appendix 3 for list of neurodegenerative disorders and motor neuron (note that not all Neurodegenerative disorders will have a specific Dx at the time of ICU admission)

Q: High flow O2 (via nasal prong) – should this be considered non-invasive ventilation? Flows are high enough to produce some CPAP?
A: If flow is ≥ 6 litres/min via nasal prong then classed as a form of mechanical respiratory support (ie CPAP).

Q: FiO2 measurement – can it only be via ETT or headbox?
A: If FiO2 can be measured accurately then it can be recorded. This includes spontaneous breathing of room air (FiO2 0.21)

Q: How to code admission after T&A because of OSA. (ie should OSA be PDX or T&A the PDX?)
A: Code T&A as PDX, with OSA as UDX and PIM Low Risk as "4".

Q: How to code patient with bronchiolitis and subsequent pneumonia – which is the PDX?
A: If the medical diagnosis at the time of ICU admission is pneumonia and this is the primary reason for admission, then code Pneumonia as PDX, with bronchiolitis as an associated diagnosis if a recent history of bronchiolitis is contributing to the current illness. In this situation do not code PIM low risk diagnosis as bronchiolitis as bronchiolitis is not the main reason for admission.

Q: definition of septic shock versus sepsis
A: see Appendix 1 for definition of septic shock

Other diagnosis coding queries:

Q: How do you use the underlying diagnosis field
A: In many cases the reason for admission has its route cause in a primary underlying medical condition. For example an ex-preterm infant with BPD and RSV infection, the underlying diagnosis is prematurity. In other situations the underlying diagnosis and the reason for admission are the same (eg asthma)

Q: How to make decisions re inclusion of historical diagnoses in notes for subsequent admissions.
A: If the condition is contributing to the need for current admission, then include it.

Q: post-op bleeding (how much to get this diagnosis?)
A: If ICU treatment is required is response to bleeding eg, airway protection, return to theatre or transfusion required.

Q: SMA – code as myopathy or neuropathy?
A: neuropathy

Q: Spinal instrumentation - does this include 1st stage scoliosis repair without rods? Does it include removal of rods?
A: yes to both questions

Appendix 1

Definition of septic shock:

Septic shock: is sepsis with cardiovascular dysfunction despite the administration of > 40ml/kg of isotonic fluid in one hour

Sepsis: SIRS in the presence of or as a result of suspected or proven infection

SIRS: widespread inflammatory response that may or may not be associated with infection.
The presence of 2 or more of the following criteria (one of which must be abnormal temperature or leukocyte count)

- core temp > 38.5oC or < 36oC
- tachycardia (HR > 2 SD above normal for age or for children < 1 yr – bradycardia
- Mean respiratory rate > 2 SD above normal for age
- WBC increased or decreased for age, or > 10% immature neutrophils

Appendix 2

Chromosomal abnormalities

Chromosomal abnormalities: Code for conditions that can be detected on routine chromosomal analyses (including FISH studies) eg Trisomies, translocations, deletions.

Examples of most common chromosomal abnormalities
Trisomies : Down syndrome is among the most common of these disorders, affecting about 1 in 800 to 1000 live-born babies.
Babies also can be born with extra copies of chromosomes 13 or 18. These trisomies are usually much more severe than Down syndrome, but fortunately less common, each affecting about 1 in 5000 babies. Babies with trisomies 13 or 18 generally have severe mental retardation and many physical birth defects.

Turner syndrome is a sex chromosome abnormality that affects about 1 in 2500 girls. Girls with Turner syndrome have only one X chromosome, instead of the normal two.
About 1 in 1000 to 2000 females has an extra X chromosome, referred to as triple X.

Klinefelter syndrome is a sex chromosome abnormality that affects about 1 in 600 to 800 boys. Boys with Klinefelter syndrome have two, or occasionally more, X chromosomes along with their Y chromosome.

Appendix 3

Neurodegenerative disorders

For the purposes or recording a neurodegenerative disorder in the PIM high risk field, a patient requires a history of progressive loss of milestones (even if no specific condition has yet been diagnosed), or a diagnosis where this will inevitably occur.

Alpers' Disease
Synonym(s): Progressive Sclerosing Poliodystrophy
Alpers' disease is a rare, genetically determined disease of the brain that causes progressive degeneration of grey matter in the cerebrum. The first sign of the disease usually begins early in life with convulsions. Other symptoms are developmental delay, progressive mental retardation, hypotonia (low muscle tone), spasticity (stiffness of the limbs), dementia, and liver conditions such as jaundice and cirrhosis that can lead to liver failure. Optic atrophy may also occur, often causing blindness. Researchers believe that Alpers' disease is caused by an underlying metabolic defect. Some patients have mutations in mitochondrial DNA.

Cerebro-Oculo-Facio-Skeletal Syndrome
Synonym(s): COFS, Pena Shokeir II Syndrome, Cockayne Syndrome Type II
Cerebro-oculo-facio-skeletal syndrome (COFS) is a paediatric, genetic, degenerative disorder that involves the brain and the spinal cord. It is characterized by craniofacial and skeletal abnormalities, severely reduced muscle tone, and impairment of reflexes. Symptoms may include large, low-set ears, small eyes, microcephaly (abnormal smallness of the head), micrognathia (abnormal smallness of the jaws), clenched fists, wide-set nipples, vision impairments, involuntary eye movements, and mental retardation, which can be moderate or severe. Respiratory infections are frequent. COFS is diagnosed at birth.

Leigh's Disease
Leigh's disease is a rare inherited neurometabolic disorder characterized by degeneration of the central nervous system. Leigh's disease can be caused by mutations in mitochondrial DNA or by deficiencies of the enzyme pyruvate dehydrogenase. Symptoms of Leigh's disease usually begin between the ages of 3 months to 2 years and progress rapidly. In most children, the first signs may be poor sucking ability and loss of head control and motor skills.

Monomelic Amyotrophy
Synonym(s): Benign Focal Amyotrophy, Hirayama Syndrome, O'Sullivan-McLeod Syndrome
Monomelic amyotrophy (MMA) is characterized by progressive degeneration and loss of motor neurons, the nerve cells in the brain and spinal cord that are responsible for controlling voluntary muscles. It is characterized by weakness and wasting in a single limb, usually an arm and hand rather than a foot and leg. There is no pain associated with MMA. While some physicians contend that mild sensory loss may be associated with this disease, many experts suggest that such symptoms actually indicate a cause other then MMA. MMA occurs in males between the ages of 15 and 25. Onset and progression are slow. MMA is seen most frequently in Asia, particularly in Japan and India; it is much less common in North America. In most cases, the cause is unknown, although there have been a few published reports linking MMA to traumatic or radiation injury. There are also familial forms of MMA. Diagnosis is made by physical exam and medical history. Electromyography (EMG), a special recording technique that detects electrical activity in muscles, shows a loss of the nerve supply, or denervation, in the affected limb; MRI and CT scans may show muscle atrophy. People believed to have MMA should be followed by a neuromuscular disease specialist for a number of months to make certain that no signs of other motor neuron diseases develop.

Progressive Multifocal Leukoencephalopathy
Progressive Multifocal Leukoencephalopathy (PML) is an infrequent disorder of the nervous system that primarily affects individuals with suppressed immune systems (including, allograft recipients such as kidney transplant patients; patients with cancers such as leukaemia or lymphoma; and nearly 10% of patients with acquired immune deficiency syndrome (AIDS). The disorder, which is caused by a common human polyomavirus, JC virus, is characterized by demyelination or destruction of the myelin sheath that covers nerve cells. The myelin sheath is the fatty covering – which acts as an insulator – on nerve fibres in the brain. Symptoms of PML include mental deterioration, vision loss, speech disturbances, ataxia (inability to coordinate movements), paralysis, and, ultimately, coma reflecting the multifocal distribution of brain lesions. In rare cases, seizures may occur.

Motor Neuron Diseases

(include as PIM high risk diagnosis of neurodegenerative disorder)

Motor neuron diseases (MNDs) are progressive, degenerative disorders that affect nerves in the upper or lower parts of the body. Some of the diseases are inherited, while others may be acquired. Common MNDs include amyotrophic lateral sclerosis (ALS), progressive muscular atrophy, and postpolio syndrome.

Amyotrophic Lateral Sclerosis
Synonym(s); Lou Gehrig's Disease
Amyotrophic lateral sclerosis (ALS) is a rapidly progressive, invariably fatal neurological disease that attacks the neurons responsible for controlling voluntary muscles. In ALS, both the upper motor neurons and the lower motor neurons degenerate or die, ceasing to send messages to muscles. Unable to function, the muscles gradually weaken, waste away, and twitch. Eventually the ability of the brain to start and control voluntary movement is lost. Individuals with ALS lose their strength and the ability to move their arms, legs, and body. When muscles in the diaphragm and chest wall fail, individuals lose the ability to breathe without the ventilatory support.

Spinal Muscular Atrophy
Spinal muscular atrophy (SMA) is a genetic, motor neuron disease caused by progressive degeneration of motor neurons in the spinal cord. The disorder causes weakness and wasting of the voluntary muscles. Weakness is often more severe in the legs than in the arms.
The childhood SMAs are all autosomal recessive diseases. The gene for SMA has been identified and accurate diagnostic tests exist. There are many types of SMA; some of the more common types are described below.

SMA type 1, also called Werdnig-Hoffmann disease, is evident before birth or within the first few months of life. There may be a reduction in fetal movement in the final months of pregnancy. Symptoms include floppiness of the limbs and trunk, feeble movements of the arms and legs, swallowing and feeding difficulties, and impaired breathing. Affected children never sit or stand and usually die before the age of 2. Symptoms of ***SMA type II*** usually begin between 3 and 15 months of age. Children may have respiratory problems, floppy limbs, decreased or absent deep tendon reflexes, and twitching of arm, leg, or tongue muscles. These children may learn to sit but will never be able to stand or walk. Life expectancy varies. Symptoms of ***SMA type III*** (Kugelberg-Welander disease) appear between 2 and 17 years of age, and include abnormal manner of walking; difficulty running, climbing stairs, or rising from a chair; and slight tremor of the fingers. ***Progressive spinobulbar muscular atrophy*** or Kennedy syndrome may occur between 15 and 60 years of age. Features of this type may include weakness of muscles in the tongue and face, difficulty swallowing, speech impairment, and excessive development of the mammary glands in males. The course of the disorder is usually slowly progressive. Kennedy syndrome is an X-linked recessive disorder.
Congenital SMA with arthrogryposis (persistent contracture of joints with fixed abnormal posture of the limb) is a rare disorder. Manifestations include severe contractures, curvature of the spine, chest deformity, respiratory problems, an unusually small jaw, and drooping upper eyelids.

Rett syndrome

Rett syndrome is a childhood neurodevelopmental disorder characterized by normal early development followed by loss of purposeful use of the hands, distinctive hand movements, slowed brain and head growth, gait abnormalities, seizures, and mental retardation.

Rett syndrome is caused by mutations in the MECP2 gene, which is found on the X chromosome. The MECP2 gene contains instructions for the synthesis of a protein called methyl cytosine binding protein 2 (MeCP2), which acts as one of the many biochemical switches that tell other genes when to turn off and stop producing their own unique proteins. Because the MECP2 gene does not function properly in those with Rett syndrome, insufficient amounts or structurally abnormal forms of the protein are formed.