## CASE 5

**Short case number: 3_16_5**
**Category: Children and Young People**
**Discipline: Paediatrics_Medicine**
**Setting: Emergency department**
**Topic: Febrile child and vaccination.**

| Case |
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| **Previously well 18 month Charlotte Rutgers presents with her mother Eunice. She is concerned that Charlotte has had high temperatures to $39^0$ C for the last 2 days, paracetamol does not seem to be helping and today she has been quite unsettled and miserable.** |
| **Questions:** |
| 1. What are the key features of history and examination that will assist in determining how unwell Charlotte is?<br>2. What clinical features are suggestive of an unwell child?<br>3. Charlotte's mother has been using paracetamol to lower her fever; summarise the pathophysiology of fever due to an infectious cause and describe the pharmacological action of paracetamol and non-steroidal anti-inflammatory medications in the management of fever in children.<br>4. Charlotte's fevers persist for a further 48 hours, she then develops a morbilliform rash and is diagnosed with roseola infantum. In a table summarise the clinical features of measles, Roseola infantum, Rubella, Erythema infectiosum, Varicella, Herpes Zoster, mumps, scarlet fever and glandular fever, under the following headings; Epidemiology, clinical features, diagnosis, complications and management.<br>5. Charlotte missed her 18 month vaccination because she was unwell with this illness, her mother asks you why children need to be vaccinated, rather than develop their own immunity to these illnesses? Describe the difference between passive and active immunity and outline the key questions that you need to ask before giving a child a vaccination.<br>6. Summarise the current vaccination schedule for children and outline the differences in the schedule for Aboriginal and Torres Strait Islander children.<br>7. Certain vaccine preventable infections are notifiable to public health units, in NSW and Victoria, briefly summarise the process of notification of infectious diseases in NSW and Victoria. |

**Suggested Reading:**


1. Richmond, P. (2012). Immunization. In South, M & Isaacs, D. (Eds) Practical Paediatrics (pp 89 - 96). Edinburgh, Churchill Livingston.
2. Burgner, D., & Isaacs, D. (2012) Infectious diseases. In South, M & Isaacs, D. (Eds) Practical Paediatrics (pp 382- 392). Edinburgh, Churchill Livingston.
3. Australian Immunisation Handbookhttps://immunisationhandbook.health.gov.au/contents
4. NSW Health. Disease Notification, August 2018 available; http://www.health.nsw.gov.au/Infectious/Pages/notification.aspx

## ANSWERS

1. **What are the key features of history and examination that will assist in determining how unwell Charlotte is?**

**HISTORY:**

- Fever history: how was temperature measured? Maximum temp measured?
- Progression of illness over time: increasingly unwell, recent rapid deterioration, waxing and waning?
- Decreasing level of activity/increasing lethargy?. Increased sleepiness/difficulty waking?
- Irritability/inconsolable/difficult to settle
- Pallor/rash?
- Level of hydration :
    - Fluid in: eating and drinking pattern: compare with usual intake? Less than 50% usual?
    - Fluid out: Vomiting or diarrhoea? (Quantity and volume of each, blood?). no of wet nappies in last 24 hours compared to usual?
- Recent overseas travel: illnesses in contacts?
- Treatment with antibiotics during current illness (? partially treated.... signs may be masked)
- Immunization history? Incomplete
- Neonatal History: Born term or premature Any associated neonatal problems
- Past Medical history? background of chronic disease, immunosuppression, previous serious infection.
- How worried is parent/s?
- Note social history as to how parent/s are coping with illness, what supports they have etc. is also important

**EXAMINATION**

- Focus is on vital signs, assessing hydration/perfusion status and identifying the source of infection:

- Vital signs. Temp:( rectal most accurate: axillary most practical ) PR RR O2sats Bare weight

- Appearance: level of alertness, interaction and activity, response to stimuli, irritability consolability.
  Colour: pallor mottling cyanosis

- Hydration peripheral perfusion: cap return (central more accurate than peripheral), skin turgor, sunken eyes, sunken fontanelles (if still open), lack of tearing when crying

- Rash? : (undress infant and look in all areas including axillary and buttocks)? petechiae /purpura

?Focus of infection:

- examine for signs of : otitis media ,pharyngitis, pneumonia, osteomyelitis, septic arthritis, soft tissue infection,intrabdominal sepsis

Note classic signs of meningism do not manifest in children up to 2 yrs of age

Remember palpation of lymphnodes, hernial orifices and careful observation for joint/limb

swelling redness or pain? Tenderness (move each joint separately/observe child at play or on

floor)

?Identifiable viral infection: bronchiolitis, croup, gingivostomatitis, varicella, hand-foot-and-

mouth disease

**2. What clinical features are suggestive of an unwell child?**

| *A, B, C, D, E, $F_3$, $P_3$ questions* | *"'MILDLY UNWELL" answer* | *"SICK CHILD" answer* |
|---|---|---|
| ALERTNESS | - mild lethargy<br>- intermittent play<br>- increased sleep | - drowsy / lethargic<br>- not playing<br>- very sleepy |
| BREATHING (rate/ effort/ noise) | - mildly altered | - slow or fast<br>- stridor |
| COLOUR | - mildly pale / flushed | - pallor<br>- mottling<br>- rash (especially early in illness) |
| DROWSINESS / SLEEP PATTERN | - short daytime sleep<br>- disrupted night sleep | - constant lethargy<br>- no alert periods |
| EMESIS | - occasional vomit | - persistent vomiting |
| FLUIDS IN | - mildly reduced | - markedly reduced<br>- not eating or drinking |
| FLUIDS OUT | - reduced urine<br>- mild diarrhoea | - markedly reduced urine<br>- persistent / bloody diarrhoea |
| FEVER | - mild fevers | - high fevers<br>- prolonged fevers (several days) |
| PAIN | - mild headache<br>- mild aches and pains | - severe headache / neck pain<br>- severe musculoskeletal pain<br>- lower limb pain |
| PATTERN | - gradual onset<br>- gradual deterioration<br>- waxing and waning | - rapid onset<br>- acute deterioration<br>- progressive deterioration<br>- deteriorating despite treatment |
| PARENTS | - mild concern | - very worried |

**From CCS MANUAL Year 2 week 22**

**ADDITIONAL NOTES**

- measure vital signs including bare weight degree of abnormality correlates with severity of illness
- increased height of temp in child with fever without focus assoc with increased risk of bacteraemia: however not a reliable indicator of seriousness of illness
- petechial or purpuric rash (= meningococcaemia until proven otherwise)

- signs of poor peripheral perfusion incicate likely severe dehydration or sepsis syndrome: pallor, mottling, cool peripheries, decreased cap return <2sec (central more accurate than peripheral), tissue turgor, sunken eyes, fontanelles, lack of tearing when crying

- when child is too young to give accurate verbal answers, pain is based on careful observation +/- parental report

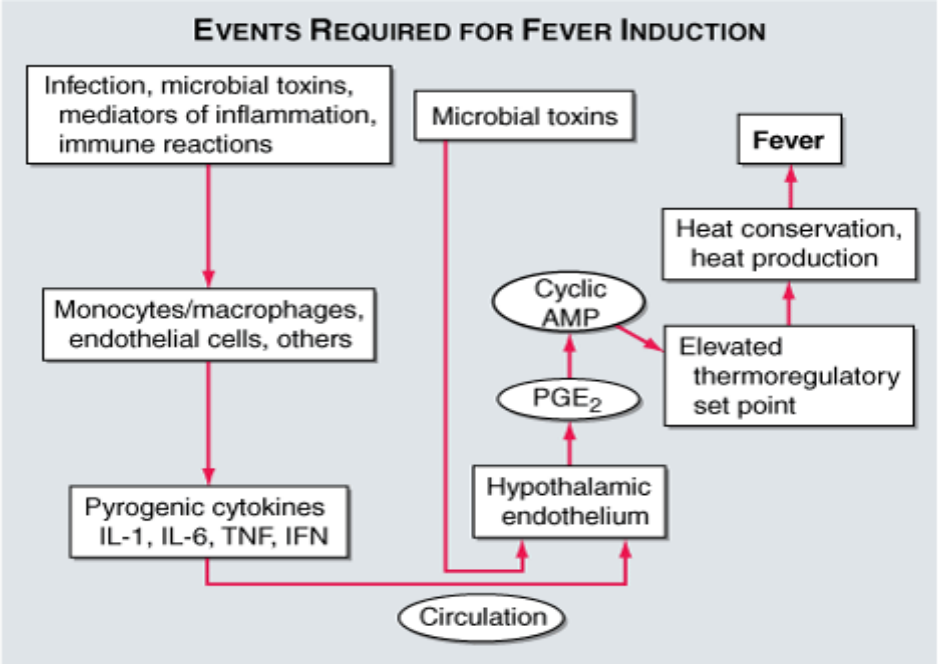
- reduced level of interaction/response to examination, increased irritability reduced sleep /inconsolability also indicative of severity of i

**3. Charlotte's mother has been using paracetamol to lower her fever; summarise the pathophysiology of fever due to an infectious cause and describe the pharmacological action of paracetamol and non-steroidal anti-inflammatory medications in the management of fever in children**

Pyrogens are substances that cause fever. Exogenous pyrogens are usually microbes or their products. The best studied are the lipopolysaccharides of gram-negative bacteria (commonly called endotoxins) and the *Staphylococcus aureus* toxin that produces toxic shock syndrome.

Exogenous pyrogens usually cause fever by inducing release of endogenous pyrogens (IL-1, tumour necrosis factor, interferon-γ, and IL-6). These are immunoregulatory proteins produced by host cells, particularly monocyte-macrophages, that elevate the hypothalamic set point. Prostaglandin $E_2$ synthesis appears to play a critical role.

(Ref: http://www.merck.com/mmpe/sec14/ch167/ch167d.htm)



Nurofen inhibits prostaglandin synthesis by inhibiting cyclooxygenase enzymes known as COX-1 and COX-2

Paracetamol Early work[1] had suggested that the fever reducing action of paracetamol was due to a central activity directly on the hypothalamus Now, recent research[2] has shown the presence of a new, previously unknown cyclooxygenase enzyme COX-3, found in the brain and spinal cord, which is selectively inhibited by paracetamol, and is distinct from the two already known cyclooxygenase enzymes COX-1 and COX-2. It is now believed that this selective inhibition of the enzyme COX-3 in the brain and spinal cord explains the effectiveness of paracetamol in relieving pain and reducing fever without having unwanted GIT side effects

4. **Charlotte's fevers persist for a further 48 hours, she then develops a morbilliform rash and is diagnosed with roseola infantum. In a table summarise the clinical features of measles, Roseola infantum, Rubella, Erythema infectiosum, Varicella, Herpes Zoster, mumps, scarlet fever and glandular fever, under the following headings; Epidemiology, clinical features, diagnosis, complications and management.**

See table next page

[1] Flower RJ and Vane JR 1972 Inhibition of prostaglandin synthetase in brain explains antipyretic activity of paracetamol Nature 240, 410-411

[2] Chandrasekharan NV et. al., 2002, COX-3, a cyclooxygenase-1 variant inhibited by acetaminophen and other analgesic/antipyretic drugs: cloning, structure, and expression, Proc. Natl. Acad. Sci. USA, **99**, 13926-13931

| | CLINICAL FEATURES | EPIDEMIOLOGY | DIAGNOSIS | COMPLICATIONS | MANAGEMENT |
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| **MEASLES** | Incubation 10 days. Prodromal period 3 days: URTI symptoms. Onset of clinical measles characterized by general malaise, systemic toxicity, fever, coryza, conjunctivitis, photophobia, and cough.<br>Exanthem develops about 14thday post exposure: starts behind ears/ hairline of forehead spreads centrifugally from head to feet. Initially erythematous, blanching and maculopapular, rapidly becomes confluent. Initially the rash is red and blanches As it fades, it takes on a copper-to-brownish hue. With healing may be fine desquamation. The rash generally lasts 7 days. | The measles vaccine (1960s) has dramatically decreased incidence in developed countries. Sporadic clusters of cases are the result of improper immunization more so than of vaccine failures, usually related to imported cases. Still widespread in underdeveloped countries where it has a high mortality/morbidity. Highly infectious Spread by respiratory secretions. Patients infectious from 3 days before rash to 4–6 days after onset. Because humans are the sole reservoir of measles, there is the potential to eliminate this disease | Serum IgM (appears 1–2 days after onset rash for about 1 mo) 4-fold rise in IgG antibodies 3-4 weeks apart Viral isolation from blood, urine, or respiratory secretions by culture possible but only at spec lab PCR currently a research tool. | Respiratory (Up to 15% of patients). Bacterial superinfections of lung, middle ear, sinus, and cervical nodes are most common. Bronchospasm, severe croup, and progressive viral pneumonia or bronchiolitis (in infants) also occur. Immunosuppressed patients are at much greater risk for fatal pneumonia than are immunocompetent patients.<br>Cerebral Encephalitis (1 in 2000) cases. Onset <week after appearance of rash. Symptoms include combativeness, ataxia, vomiting, seizures, and coma. 40%die or have severe neurologic sequelae.<br>Subacute sclerosing panencephalitis (SSPE) a | Management of measles is supportive. Antiviral therapy is not effective in the treatment of measles in otherwise normal patients Ribavirin is active in vitro and may be useful in the immunocompromised In malnourished children, vitamin A attenuates illness |

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| | Koplik spots pathognomonic (white, 1-mm spots first appear on the buccal mucosa opposite lower molars spreads to entire buccal mucosa.) | | | slow measles virus infection of the brain: becomes symptomatic years later in about 1 in 100,000 previously infected children. : progressive cerebral deterioration; associated with myoclonic jerks and a typical electroencephalographic pattern. It is fatal in 6–12 months.<br><br>Other: haemorrhagic measles (severe disease with multiorgan bleeding, fever, cerebral symptoms), thrombocytopenia, appendicitis, keratitis, myocarditis, and premature delivery or stillbirth. Mild liver function test elevation has been detected in up to 50% of cases in young adults; frank jaundice may also occur. Measles causes transient | |

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| | | | | immunosuppression; thus, reactivation or progression of tuberculosis (including transient cutaneous anergy) occurs in untreated children. | |
| **ROSEOLA INFANTUM** | Roseola is a mild febrile, exanthematous illness Prodromal period usually asymptomatic, may include mild URTI, (minimal rhinorrhea, slight pharyngeal inflammation, mild conjunctival redness). Mild cervical or, less frequently, occipital lymphadenopathy may be noted. Some children may have mild palpebral oedema. Clinical illness is generally heralded by high temperature, usually ranging from 37.9 to 40°C average of 39°C Some children may become | Roseola occurs throughout the year. Unlike some of the other childhood exanthems, children with roseola rarely report contact with other affected children, and outbreaks are uncommon. The incubation period averages 10 days, with a range of 5–15 days. More than 95% of roseola cases occur in children younger than 3 yr., with a peak at 6–15 mo of age. Transplacental antibodies likely protect most infants until 6 mo of age. | Diagnosis is usually clinical, especially when the characteristic rash appears after fever resolves. Specific testing for HHV-6 or HHV-7 infection may be performed using laboratory methods, including serology, virus culture, and polymerase chain reaction (PCR). | Seizures may occur in 5–10% of children with roseola during this febrile period | In immunocompetent children, no specific therapy is warranted. |

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| | irritable and anorexic during the febrile stage, but most behave normally despite high temperatures. Fever persists for 3–5 days, and then typically resolves rather abruptly A rash appears within 12–24 hr of fever resolution. The rash of roseola is rose coloured, as the name implies, and is fairly distinctive: begins as discrete, small (2–5 mm), slightly raised pink lesions on the trunk and usually spreads to the neck, face, and proximal extremities: not usually pruritic, no vesicles or pustules. Lesions typically remain discrete but occasionally may become almost confluent. After 1–3 days, the rash fades. | | | | |

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| **RUBELLA** | a mild disease not easily discernible from other viral infections, especially in children but devastating when transmitted in utero. Incubation period 14–21 days, prodrome low-grade fever, sore throat, red eyes with or without eye pain, headache, malaise, anorexia, and lymphadenopathy. Suboccipital, postauricular, and anterior cervical lymph nodes are most prominent. In children, the 1st manifestation of rubella is usually the rash, which is variable and not distinctive. It begins on the face and neck as small, irregular pink macules that coalesce, spreads centrifugally to involve the torso and extremities, where it tends to occur as discrete macules | Since rubella vaccine was implemented the incidence has fallen dramatically in developed countries Otherwise endemic and epidemic in non immunised populations Respiratory spread. | Important for epidemiologic reasons, for diagnosis of infection in pregnant women, and for confirmation of the diagnosis of congenital rubella. most common is rubella IgM | Congenital Rubella Syndrome if rubella occurs in a pregnant woman Other in index case infrequent: thrombocytopenia (1 : 3,000) Arthritis (most common female adults, encephalitis (1/5,000) Progressive rubella panencephalitis extremely rare similar to SSPE | No specific treatment available for either acquired rubella or CRS |

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| | About the time of onset of the rash, examination of the throat may reveal tiny, rose-coloured lesions (Forchheimer spots) or petechial haemorrhages on the soft palate. The rash fades from the face as it extends to the rest of the body so that the whole body may not be involved at any 1 time. The duration of the rash is generally 3 days, and it usually resolves without desquamation. Subclinical infections are common, and 25–40% of children may not have a rash. | | | | |
| **ERYTHEMA INFECTIOSUM** | incubation period 4–28 days. Prodromal phase: low-grade fever, mild headache, URTI followed by characteristic rash which occurs in 3 stages<br>1. Erythematous facial flushing, often described as | Infections with parvovirus B19 are common. 70% cases occur between 5 and 15 yr of age. Seasonal peaks: late winter and spring, sporadic infections throughout the year. mildly contagious; the | Usually based on clinical presentation of the typical rash. A mild leukopenia occurs early in some patients, followed by | Arthritis and arthralgia may occur in isolation or with other symptoms (more common in adults and older adolescents) Usually hands, wrists, knees, and ankles, resolves in 2–4 wk In people with chronic haemolytic | There is no specific antiviral therapy. Immunoglobulin (IVIG) have been used with some success to treat B19- |

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| | a “slapped-cheek” appearance. 2. spreads rapidly to trunk+proximal extremities as diffuse macular erythema 3.Central clearing of macular lesions occurs promptly, giving the rash a lacy, reticulated appearance<br>Rash more prominent on extensor surfaces, sparing the palms and soles. Usually afebrile look well. Older children and adults often get mild pruritus. Rash resolves but tends to wax and wane over 1–3 wk. It can recur with exposure to sunlight, heat, exercise, and stress. | secondary attack rate is 50% among susceptible children and 20–30% Transmission usually respiratory route, also transmissible in blood /blood products, | leukocytosis and lymphocytosis. Specific IgM and IgG serum antibody tests are available, Nucleic acid detection tests often definitive. | diseases or the immunosuppressed profound anaemia can occur as Parvovirus B19 replicates primarily in erythroid progenitor cells. Myocarditis (rare) Nonimmune hydrops foetalis following infection of pregnant women. | related episodes of anemia and bone marrow failure in immunocompro mised children. |
| **VARICELLA** | Varicella-zoster virus (VZV) causes primary, latent, and recurrent infections The primary infection is manifested as varicella (chickenpox) and results in | since introduction of vaccine (1995) there has been a substantial decline in varicella morbidity and mortality. | Leukopenia is typical during the 1st 72 hr; followed by lymphocytosis. Mildly elevated liver function | RARE in healthy patients In immunosuppressed: may see progressive varicella: (visceral organ involvement, coagulopathy, severe haemorrhage), Pregnant woman who contract VZ in | Antiviral treatment modifies course of both varicella and herpes zoster |

| | CLINICAL FEATURES | EPIDEMIOLOGY | DIAGNOSIS | COMPLICATIONS | MANAGEMENT |
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| | establishment of a lifelong latent infection of sensory ganglion neurons. Reactivation of the latent infection causes herpes zoster (shingles). Primary disease: incubation p 14–16 days. Prodromal symptoms, (particularly older children/adults): Fever, malaise, anorexia, headache, and occasionally mild abdominal pain may occur 24–48 hr before the rash, a mod fever and other systemic symptoms persist during the 1st 2–4 days after the onset of the rash. Varicella lesions often appear first on scalp, face, or trunk. Exanthem: intensely pruritic erythematous macules that evolve through the papular stage to form clear, fluid-filled vesicles. Clouding and umbilication of the lesions | | tests occur in 75%. VZV can be identified quickly by direct fluorescence assay of cells from cutaneous lesions, and by PCR amplification testing | first trimester are at high risk of having a child born with congenital varicella syndrome. Infants whose mothers develop varicella in the period from 5 days prior to delivery to 2 days afterward are at high risk for severe varicella with high mortality. (transplacental transmission ) | acyclovir therapy is not routinely recommended for treatment of uncomplicated varicella in the otherwise healthy child (cost, marginal benefit, low risk of complications) Any child ill enough to be hospitalization and who has new vesicle formation should be treated with acyclovir Consider oral acyclovir for children >12 years, those |

| | CLINICAL FEATURES | EPIDEMIOLOGY | DIAGNOSIS | COMPLICATIONS | MANAGEMENT |
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| | begin in 24–48 hr. While the initial lesions are crusting, new crops form on the trunk and then the extremities; the simultaneous presence of lesions in various stages of evolution is characteristic of varicella Ulcerative lesions involving the mucosa of oropharynx and vagina are also common; many children have vesicular lesions on the eyelids and conjunctivae, Hypopigmentation or hyperpigmentation of lesion sites persists for days to weeks in some children, but severe scarring is unusual unless the lesions were secondarily infected. | | | | with chronic cutaneous or pulmonary disorders, newborn infants, and selected immunocompromised persons at risk for severe varicella. Children with varicella should not receive salicylates because of the association with Reye syndrome |
| **HERPES ZOSTER** | manifests as vesicular lesions clustered within 1, less commonly 2 adjacent dermatomes Unlike **herpes** | RARE in healthy children | Rapid Immunofluorescence or PCR amplification testing testing of | In contrast to adults, post herpetic neuralgia is very unusual in children. Approximately 4% of patients suffer a 2nd episode | Strict isolation for the duration of vesicular eruption |

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| | **zoster** in adults, in children it is infrequently associated with localized pain, hyperesthesia, pruritus, and low-grade fever. In children, the rash is mild, with new lesions appearing for a few days; symptoms of acute neuritis are minimal; and complete resolution usually occurs within 1–2 wk. | | vesicular fluid Culture of fluid | of **herpes zoster**; 3 or more episodes are rare. Transverse myelitis with transient paralysis rarely occurs | Acyclovir arguable for otherwise healthy children but is effective in decreasing duration/severity. Antiviral drugs should be used in adults and immunocompromised, most effective <72hrs prior to onset |
| **MUMPS** | Range from nonspecific symptoms to typical illness associated with parotitis with or without complications Usual prodrome 1–2 days): fever, headache, vomiting, myalgia Parotitis then appears unilateral initially becomes bilateral in about 70%: | Incidence once very common disease has declined dramatically post immunization. Outbreak still occur<br>Spread by respiratory secretions.<br>Infectious: 7 days before to 9 days after onset of parotid swelling Incubation period: 12–25 days after | Usually clinical diagnosis because lack of sensitivity of laboratory tests, particularly in vaccinated persons. A confirmation can made by viral culture from | aseptic meningitis –usually benign (30%), orchitis (25%), usually unilateral, pancreatitis (usually mild), oophoritis, thyroiditis, neuritis, hepatitis, myocarditis, thrombocytopenia, migratory arthralgias (noted infrequently among adults and rarely in children), and | Treatment is symptomatic. Topical application of compresses may relieve parotid discomfort. |

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| | parotid gland tender and swollen, Ingestion of sour or acid may enhance pain. As swelling progresses, the angle of the jaw is obscured and the ear lobe may be lifted upward and outward: peaks 3 days, subsides over 7. Fever resolves in 3 to 5 days along with the other systemic symptoms. A morbilliform rash is rarely seen. Submandibular salivary glands may also be involved | exposure<br>Humans are the only known host for mumps. | parotid duct. Increased IgM diagnostic but often delayed 2-3 weeks in prev immunized. Fourfold rise in paired IgG also confirms infection. | nephritis, rare neurologic complications Mumps in early pregnancy can cause abortion or foetal abnormalities | |

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| **SCARLET FEVER** | **Scarlet fever** is an URTI, usually pharyngitis associated with a characteristic rash, caused by an infection with a Group A strep pyogenes that produces erythrogenic toxin in individuals who do not have antitoxin antibodies. Now less common/less virulent than in past: incidence is cyclic, depending on the prevalence of toxin-producing strains and the immune status of the population. Occurs uncommonly before 3years or after15 (possibly related to requirement for prior /exposure and sensitization and development of toxin-specific immunity) | Characteristic rash appears 24–48 hr after onset typical pharyngitis symptoms: Usually begins on the trunk and spreads to involve almost the entire body within hours to days.<br>Diffuse, finely papular, erythematous producing a bright red discoloration, blanches on pressure. (often more intense along skin creases) goose-pimple appearance, feels rough: face usually spared, cheeks may be red with prominent perioral pallor After 3–4 days, rash fades followed by desquamation, (often resembling mild sunburn peeling) Oropharynx; typical GpAS pharyngitis Dorsum of tongue: white coat early in illness with edematous red papillae. White covering desquamates and reveals swollen, red, and mottled | Clinical diagnosis of rash in association with positive gp A strep pharyngitis Rapid streptococcal antigen tests: 50–80% sensitivity and >95% specificity Throat culture: The gold standard with best sensitivity (>90%) for group A β-hemolytic streptococci. | generally benign if treated: severe cases: sepsis, rheumatic fever can occur after untreated infection, glomerulonephritis | Identical to therapy for strep pharyngitis i.e. antibiotic Rx e.g. penicillin |

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| | | "strawberry tongue". | | | |
| **GLANDULAR FEVER** | The majority of primary EBV infection in infants and young children are clinically silent. In older patients, the onset of illness usually insidious and vague. Prodromal phase (1-2 weeks): malaise, fatigue, acute or prolonged (>1 wk) fever, headache, sore throat, nausea, abdominal pain, and myalgia. The complaints of sore throat and fever gradually increase, Splenic enlargement may be rapid enough to be symptomatic<br>**Examination:** generalized lymphadenopathy (90% of cases), splenomegaly (50% of cases), | Worldwide distribution<br>Humans are the only known reservoir.<br>Transmission occurs through saliva and genital secretions<br>Incubation period is 30–50 days.<br>Most common in adolescence or young adults<br>Antibodies to EBV are almost universally present in adult populations.<br>Areas with a high population density or low socioeconomic status usually become primarily infected within the 1st 3 years of life and have a minor or subclinical atypical course. | Atypical lymphocytosis in the peripheral blood indicative, usually confirmed by serologic testing, either for heterophile antibody(mons pot or monalert) or specific EBV antibodies.<br>Mild thrombocytope nia and neutropenia are common | Airway compromise <5%<br>Splenic rupture rare <0.5% history of preceding trauma in 50% of the cases.<br>Hemolytic anemia3%<br>Fulminant hepatitis, pericarditis, myocarditis, neurologic involvement—including transverse myelitis, encephalitis, and Guillain–Barré syndrome—is infrequent. Prolonged and debilitating fatigue, malaise, and some disability that may wax and wane for several weeks to 6 mo are common complaints. Occasional persistence of fatigue for a few years after infectious **mononucleosis** is well recognized. There is no convincing evidence linking chronic fatigue syndrome | Rest and symptomatic treatments are the mainstays of management |

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| | hepatomegaly (10% of cases). Lymphadenopathy occurs most commonly in the anterior and posterior cervical nodes and the submandibular lymph nodes and less commonly in the axillary and inguinal lymph nodes. Symptomatic hepatitis or jaundice uncommon, but elevated liver enzymes are common. Splenomegaly to 2–3 cm below the costal margin is typical; massive enlargement is uncommon, exudative pharyngitis with marked tonsillar enlargement, Petechiae at the junction of the hard and soft palate frequent Rashes (3–15%) | | | | |

REFERENCES;

- Kliegman: Nelson Textbook of Paediatrics, 18th ed. Copyright © 2007 Saunders, An Imprint of Elsevier

- Murtaghs General Practice 4th edition McGraw Hill Australia 2007.
- CURRENT Diagnosis & Treatment: Paediatrics Chapter 38. Infections: Viral & Rickettsial William W. Hay, Jr., Myron J. Levin, Judith M. Sondheimer, Robin R. Deterding 19th Edition 2009 Lange books

5. **Charlotte missed her 18 month vaccination because she was unwell with this illness, her mother asks you why children need to be vaccinated, rather than develop their own immunity to these illnesses? Describe the difference between passive and active immunity and outline the key questions that you need to ask before giving a child a vaccination.**

**Passive and active immunity:**

ACTIVE IMMUNITY: When a healthy person becomes infected with a virus, e.g. measles, the body recognises the virus as an invader, produces antibodies which eventually destroy the virus and recovery occurs. If contact with the measles virus occurs again in the future, the body's immune system 'remembers' the measles virus and produces an increase in antibodies to destroy the virus.

Vaccination is the process that is used to stimulate the body's immune system in the same way as the real disease would, but without causing the symptoms of the disease. Most vaccines provide the body with 'memory' so that an individual doesn't get the disease if exposed to it.

PASSIVE IMMUNITY:
Immunity can also be acquired passively by the administration of immunoglobulins. Such immunity is immediate and is dose-related and transient. For example, measles or hepatitis B immunoglobulin can be used promptly after exposure in an unimmunised person to help reduce the chance of catching measles or hepatitis B from the exposure.

**The key questions to ask before giving a child a vaccination:**
These can be provided as a check list to give to parents/carers which act as screening questions pre vaccination

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Is/does the child to be immunised:

- an Aboriginal or Torres Strait Islander
- unwell today
- have a disease which lowers immunity (e.g. leukaemia, cancer, HIV/AIDS)or is having treatment which lowers immunity (e.g. oral steroid medicines such as cortisone and prednisone, radiotherapy, chemotherapy) or does he/she live with someone who has a disease which lowers immunity (e.g. leukaemia, cancer, HIV/AIDS), or lives with someone who is having treatment which lowers immunity (e.g. oral steroid medicines such as cortisone and prednisone, radiotherapy, chemotherapy)
- have they had a severe reaction following any vaccine
- have they any severe allergies (to anything)
- have had any vaccine in the past month
- have they had an injection of immunoglobulin, or received any blood products or a whole blood transfusion within the past year
- do they have a past history of Guillain-Barré syndrome

- were they a preterm infant
- so they have  a chronic illness
- do they have a bleeding disorder
- does he/she have a functioning spleen

The immunisation provider then only needs to ask:

- Did you understand the information provided to you about immunisation?
- Do you need more information to decide whether to proceed?
- Did you bring your/your child's vaccination record card with you

**Table 1.3.2: Responses to relevant conditions or circumstances identified by the pre-vaccination screening checklist**

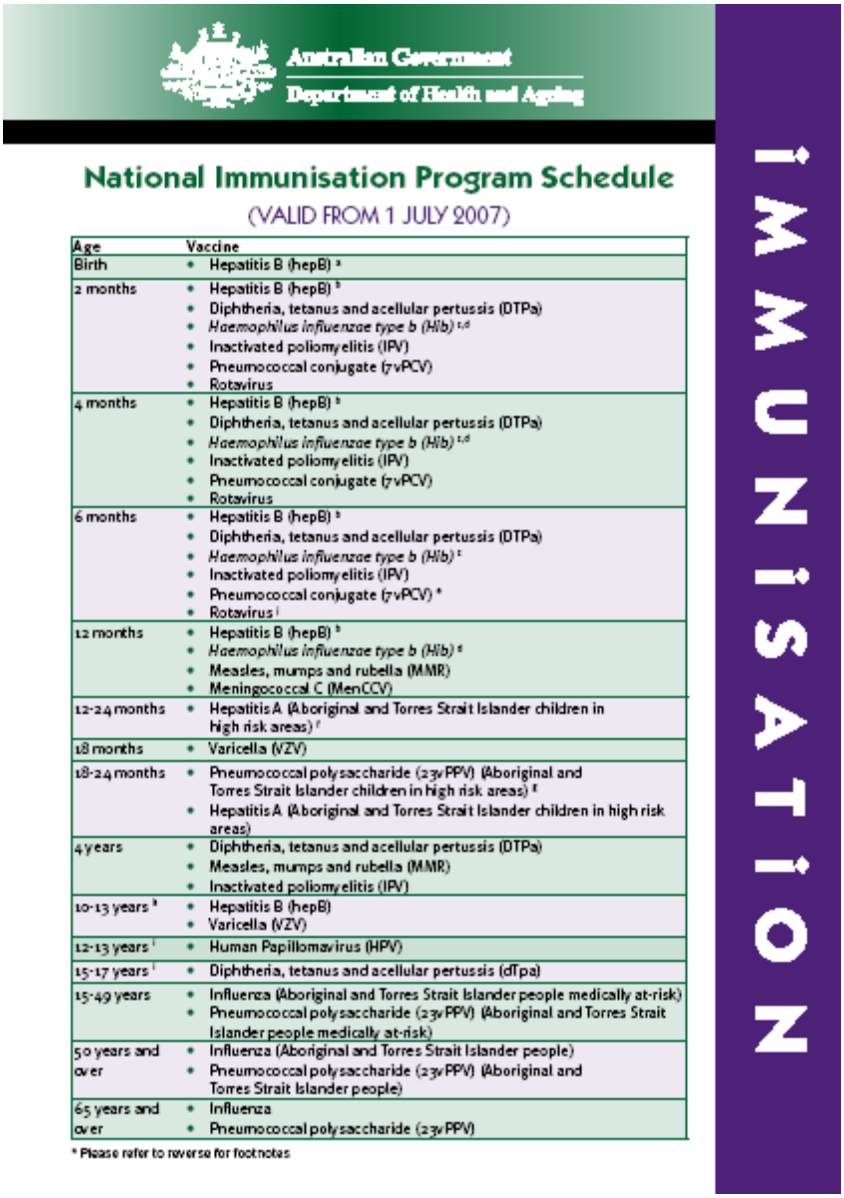
| Condition or circumstance | Action | Rationale[13-15] |
|---|---|---|
| Unwell today:<br>• Acute febrile illness (current T ≥38.5°C).<br>• Acute systemic illness. | Defer all vaccines until afebrile.<br>NB. Children with minor illnesses (without acute systemic symptoms/signs) should be vaccinated. | To avoid an adverse event in an already unwell child, or to avoid attributing symptoms to vaccination. |
| Has a disease which lowers immunity or receiving treatment which lowers immunity.<br>See Section 2.3.3, *Vaccination of individuals with impaired immunity due to disease or treatment.* | Seek expert advice before vaccination (see Appendix 1).<br>NB. People living with someone with lowered immunity should be vaccinated, including with live viral vaccines. | The safety and effectiveness of the vaccine may be suboptimal in people with impaired immunity. |
| Anaphylaxis following a previous dose of the relevant vaccine. | Do not vaccinate.<br>See also 'Contraindications to vaccination' below. | Anaphylaxis to a previous dose of vaccine is a contraindication to receiving the vaccine. |
| A severe (anaphylactic) allergy to a vaccine component. | Do not vaccinate (seek specialist advice as per Appendix 1). | Anaphylaxis to a vaccine component is |

| Condition or circumstance | Action | Rationale[13-15] |
|---|---|---|
| Refer to Appendix 4 for vaccine component checklist. | See also 'Contraindications to vaccination' below. | a contraindication to receiving the vaccine. |
| Received live parenteral vaccine or BCG vaccine in past 4 weeks. | Delay live vaccines by 4 weeks. | The immune response to a live viral vaccine may interfere with the response to a second live viral vaccine if given within 4 weeks of the first. |
| Has had any blood product in the past 7 months, or has had IM or IV immunoglobulin in the past 11 months. Refer to Table 2.3.5 *Recommended intervals between either immunoglobulins or blood products and MMR, MMRV or varicella vaccination.* | Make a return appointment for this vaccination, and send a reminder later if necessary. | Antibodies within these products may interfere with the immune response to these vaccines. |
| History of Guillain-Barré syndrome (GBS). See Chapter 3.9, *Influenza*. | Risks and benefits of influenza vaccine should be weighed against the potential risk of GBS recurrence (seek specialist advice as per Appendix 1). | People with a history of GBS may be at risk of recurrence of the condition following influenza vaccine. |
| Was born preterm. . | Preterm infants born at <28 weeks' gestation or <1500 g birth weight require an extra dose of PRP-OMP Hib vaccine at 6 months of age. Preterm infants born at <28 weeks' gestation and/or with chronic lung disease require extra | Preterm infants may be at increased risk of vaccine preventable diseases (e.g. invasive pneumococcal disease (IPD)), and may not mount an optimal immune response to certain vaccines (eg. hepatitis B, PRP-OMP). |

| Condition or circumstance | Action | Rationale[13-15] |
|---|---|---|
| | pneumococcal vaccinations. Preterm infants born at <32 weeks' gestation or <2000 g birth weight may require an extra dose of hepatitis B vaccine. | |
| Has a severe or chronic illness. See Chapter 2.3, *Groups with special vaccination requirements.* | These people should receive pneumococcal vaccine and annual influenza vaccination. If there is significantly impaired immunity, they should not receive live vaccines, but inactivated vaccines should be considered (seek expert advice). | People with a severe or chronic illness may be at increased risk of vaccine preventable diseases (e.g. IPD) but may not mount an optimal immune response to certain vaccines. |
| Has a bleeding disorder. See Section 2.3.6, *Vaccination of patients with bleeding disorders.* | The subcutaneous route could be considered as an alternative to the intramuscular route (seek specialist advice as per Appendix 1). | Intramuscular injection may lead to haematomas in patients with disorders of haemostasis. |
| Identifies as an Aboriginal or Torres Strait Islander. | See the National Immunisation Program Indigenous schedules. | Some groups of Indigenous people are at increased risk of some of the vaccine preventable diseases. |
| Does not have a functioning spleen. | Check vaccination status for pneumococcal, meningococcal and Hib vaccinations. | Individuals with an absent or dysfunctional spleen are at an increased risk of severe bacterial infections, most notably IPD. |
| | | |
| | | |
| Lives with someone who has impaired immunity. | Ensure all vaccines (in particular MMR, varicella | Household members are the most likely sources of vaccine-preventable |

| Condition or circumstance | Action | Rationale[13-15] |
|---|---|---|
| | and influenza vaccines) recommended for their age-group have been offered to household members of people with impaired immunity. | diseases among people with impaired immunity (who often are unable to be vaccinated, especially with live viral vaccines). |

**6. Summarise the current vaccination schedule for children and outline the differences in the schedule for Aboriginal and Torres Strait Islander children.**



Australian Government
Department of Health and Ageing

IMMUNISATION

## National Immunisation Program Schedule

(VALID FROM 1 JULY 2007)

| Age | Vaccine |
|---|---|
| Birth | • Hepatitis B (hepB) [a] |
| 2 months | • Hepatitis B (hepB) [b]<br>• Diphtheria, tetanus and acellular pertussis (DTPa)<br>• *Haemophilus influenzae type b (Hib)* [c,d]<br>• Inactivated poliomyelitis (IPV)<br>• Pneumococcal conjugate (7vPCV)<br>• Rotavirus |
| 4 months | • Hepatitis B (hepB) [b]<br>• Diphtheria, tetanus and acellular pertussis (DTPa)<br>• *Haemophilus influenzae type b (Hib)* [c,d]<br>• Inactivated poliomyelitis (IPV)<br>• Pneumococcal conjugate (7vPCV)<br>• Rotavirus |
| 6 months | • Hepatitis B (hepB) [b]<br>• Diphtheria, tetanus and acellular pertussis (DTPa)<br>• *Haemophilus influenzae type b (Hib)* [c]<br>• Inactivated poliomyelitis (IPV)<br>• Pneumococcal conjugate (7vPCV) [e]<br>• Rotavirus [f] |
| 12 months | • Hepatitis B (hepB) [b]<br>• *Haemophilus influenzae type b (Hib)* [g]<br>• Measles, mumps and rubella (MMR)<br>• Meningococcal C (MenCCV) |
| 12-24 months | • Hepatitis A (Aboriginal and Torres Strait Islander children in high risk areas) [f] |
| 18 months | • Varicella (VZV) |
| 18-24 months | • Pneumococcal polysaccharide (23vPPV) (Aboriginal and Torres Strait Islander children in high risk areas) [g]<br>• Hepatitis A (Aboriginal and Torres Strait Islander children in high risk areas) |
| 4 years | • Diphtheria, tetanus and acellular pertussis (DTPa)<br>• Measles, mumps and rubella (MMR)<br>• Inactivated poliomyelitis (IPV) |
| 10-13 years [h] | • Hepatitis B (hepB)<br>• Varicella (VZV) |
| 12-13 years [i] | • Human Papillomavirus (HPV) |
| 15-17 years [i] | • Diphtheria, tetanus and acellular pertussis (dTpa) |
| 15-49 years | • Influenza (Aboriginal and Torres Strait Islander people medically at-risk)<br>• Pneumococcal polysaccharide (23vPPV) (Aboriginal and Torres Strait Islander people medically at-risk) |
| 50 years and over | • Influenza (Aboriginal and Torres Strait Islander people)<br>• Pneumococcal polysaccharide (23vPPV) (Aboriginal and Torres Strait Islander people) |
| 65 years and over | • Influenza<br>• Pneumococcal polysaccharide (23vPPV) |

* Please refer to reverse for footnotes

**ABORGINAL AND TORRES STRAIT ISLANDER CHILDREN**

Aboriginal and Torres Strait Islander children living in certain regions require extra protection against some diseases. Children from these states should receive all the routine vaccines given to other children, with the following differences and additions:

**Pneumococcal infection**

In Queensland, the Northern Territory, Western Australia and South Australia an additional booster dose of the pneumococcal vaccine (PneumoVax®23) is required between 18 and 24 months. This is required because Aboriginal and Torres Strait Islander children living in these areas continue to be at risk of pneumococcal disease for a longer period than other children. The vaccine used for this dose is different from the one used for babies.

**Hepatitis A vaccination program**

In Queensland, the Northern Territory, Western Australia and South Australia the Government provides free hepatitis A vaccine for all Aboriginal and Torres Strait Islander

children less than five years of age because hepatitis A is more common among Aboriginal and Torres Strait Islander children in these areas than it is among other children. Two doses of vaccine are given between 12 and 24 months of age.

**Hib (Haemophilus influenzae type b)**

In the Northern Territory and certain remote areas of South Australia the preferred vaccine is a specific type, called Hib PRP-OMP. This vaccine provides increased protection to very young infants and is used because there is an increased risk for this age group among Aboriginal and Torres Strait Islander children living in these areas. This vaccine should be given at 2, 4 and 12 months of age, at the same time as other routine vaccines.

**7. Certain vaccine preventable infections are notifiable to public health units, in NSW and Victoria, briefly summarise the process of notification of infectious diseases in NSW and Victoria.**

**NSW NOTIFICATION MECHANISMS:**

The Department's policy directive mandates that notifiable diseases must be reported to the local Public Health Unit (PHU).

http://www.health.nsw.gov.au/infectious/pages/notification.aspx

***Notifiable diseases***

Notifiable diseases are classified according to the urgency of notification (some must be notified by telephone ideally as soon as diagnosis made) All case notification must be made within 24 hours. Infectious diseases are also classified according to who can/must notify: this may include treating doctor, hospital chief executive officers or general managers, laboratories and even school principles/directors of child care centres.

***Notification Mechanism***

**Case notification must be initiated within 24 hours of diagnosis**

**1- Download the relevant notification form from the Notifiable disease list** All infectious diseases notification forms are available from Public Health Units
and on the NSW Health website
http://www.health.gov.au/internet/main/publishing.nsf/Content/cdna-casedefinitions.htm

**2- Fill in the form**

**3- Notify the PHU by phone if the notifiable disease is followed by a**

**4- Send in the form**

Doctor and Hospital Notifications should be directed to the local Public Health Unit, except for HIV, which is notifiable only by reference laboratories direct to the Communicable Diseases Branch, NSW Department of Health.

In order to protect patient confidentiality, notifications **must not be made by facsimile machine** except in exceptional circumstances and when confidentiality is ensured.

**VICTORIA NOTIFICATION MECHANISMS**
http://ideas.health.vic.gov.au/notifying.asp
**(NSW similar – see** http://www.health.nsw.gov.au/infectious/pages/notification.aspx

**Step 1 (Mandatory for Group A diseases / optional for Group B/C/D diseases)**

- Telephone our priority number 1300 651 160
  This number connects you directly to the Department of Health's Communicable Disease Prevention and Control Unit for the cost of a local call from most fixed phones (additional charges may apply for calls made from mobile phones).
  For urgent notifications outside office hours, please telephone the departments after hours service on 1300 790733 and advise the operator that you wish to make an 'urgent infectious disease notification'.

**Step 2 (Mandatory for all diseases) see**

- **Notify online using the secure e-Form**

**OR**

**Complete the Notification of Infectious Disease Form**
Fax the completed form to our priority number **1300 651 170**, which connects you directly through to the Communicable Disease Prevention and Control Unit for the cost of a local call from most fixed phones (additional charges may apply for calls made from mobile phones) Notifiable infectious diseases are included in Schedule 3 of the Health (Infectious Diseases) Regulations 2001 and are divided into four groups on the basis of the method of notification and the information required. With the exception of HIV and AIDS, these groups are all included on the standard Notification of Infectious Disease form.

**Disease groupings**

**Group A -** Diseases require immediate notification to the Department of Health by telephone or fax upon initial diagnosis (presumptive or confirmed) with written notification to follow within five days.

**Group B** - Diseases require written notification only within five days of diagnosis (presumptive or confirmed).

**Group C** - Diseases include the sexually transmissible diseases and should be notified using the same form. To preclude identification of the patient, only the first two letters of the given and family name of the patient are required.

**Group D** - Diseases include HIV infection (Human Immunodeficiency Virus) and AIDS (Acquired Immunodeficiency Syndrome) and written notification is required within five days of confirmation of diagnosis. A separate form is used for this purpose due to the need to have national uniformity in collection of data.