This guideline has been produced by the paediatric south London Network, and is an expert document which covers in detail the treatment of children with HIV. The management pathway for neonates born to HIV positive mothers is detailed on pages 6-11 in a number of scenarios to guide the risk to the infant based on the maternal treatment and viral load.

Pages 12 – 14 give specific instructions on what to prescribe based on this risk assessment, and when follow up and repeat blood tests are required. Further on in the document is some very useful information about drug doses, counselling and general management which is relevant.

The neonatal consultant should be informed about infants born to HIV positive mothers. Rarely there may be a suspicion that a mother may be HIV infected due to high risk activities, where the mother has refused HIV testing. These cases must also be discussed with the neonatal consultant.
INTRODUCTION

Welcome to the 2005/06 edition of ‘Paediatric HIV Management’ from PHILSnet (Paediatric HIV South London Network). Given the expansion of our network beyond the borders of Tooting, PHILSnet no longer seems appropriate! A new acronym is in order. We welcome your suggestions on renaming our network.

It is always a pleasure interacting with you either by phone, at our network meetings or at our respective centres. We feel networking in this way has enhanced clinical care for the children we care for and their families. The following guidelines are intended to provide a brief synopsis on selected topics in Paediatric HIV care, and are not comprehensive. They have been designed with the ‘on-call’ clinician in mind when faced with an unwell HIV-infected child, or an infant who could potentially become HIV-infected without appropriate intervention.

The manual reflects the current standard of care within the UK in 2005. In a field as rapidly evolving as Paediatric HIV care some of the recommendations will become outdated. Further literature can be obtained from the Children’s HIV Association at http://www.bhiva.org/chiva/ and from PENTA (The Paediatric European Network for Treatment of AIDS) http://www.pentatrials.org

In recent years a number of new challenges have evolved that are updated in the guidelines including management of multi-drug resistant virus in HIV-infected children and care of vertically infected adolescents. However these areas are evolving, and sharing your experience on these and other topics is invaluable.

We can be contacted through any of the numbers/email addresses on Page 3, and welcome your questions and feedback. The attending paediatric infectious diseases consultant at St George’s is available through switchboard 020 8672 1255 if advice on management is needed.

We would like to thank our staff, colleagues, collaborators, and the families.

Rana Chakraborty and Mike Sharland.        April 2005
# TABLE OF CONTENTS

CONTACT DETAILS .................................................................................................................. 4

PERINATAL GUIDELINES 2005/06 .......................................................................................... 5

PHILS-NET ART GUIDELINES 2005/06 ............................................................................... 15

MANAGEMENT OF CLINICAL ILLNESS ............................................................................. 27  
(a) The infant/child with HIV and fever ............................................................................. 27  
(b) The infant/child with advanced HIV and gastroenteritis (GE) ................................... 28  
(c) The infant/child with HIV and respiratory illness ..................................................... 30

GUIDELINES FOR HIV TESTING OF CHILDREN AND YOUNG PEOPLE ....... 36
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**Paediatric Infectious Diseases Unit Ward – Pinckney Ward 5th Floor Lanesborough Wing**

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<td>NICU:</td>
<td>1936</td>
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<td>Pinckney ward office:</td>
<td>3935</td>
<td>1724/2953</td>
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Urgent (or non-urgent) contact: - To air-call anyone, you need to dial 07699 119700, you will then be asked to leave a message, or contact the switchboard at St George’s Hospital 0208 672 1255.
PERINATAL GUIDELINES 2005/06

Scale of the HIV epidemic among children in the UK and globally

Within a 20-year period HIV infection has spread from a few high-risk groups to become a worldwide pandemic. According to statistics released from UNAIDS and the WHO more than 60 million people have been infected with HIV-1. An estimated 14,000 new infections occur daily. 95% of these occur in developing nations where access to newer medical treatments are not readily available or affordable.

HIV infection is now the leading cause of death in sub-Saharan Africa. Vertical transmission is the primary means by which infants become infected with HIV either in utero, during delivery or by breast-feeding. At the end of 2003 between 2.1-2.9 million children (<15 years) were infected with HIV globally.

In the UK since 1999 there have been more diagnoses of heterosexually acquired infection than of infections acquired through sex between men with an increase in the numbers of women diagnosed. A few HIV-infected women remain undiagnosed until testing is prompted by HIV related symptoms late in the course of illness. Women who remain unaware of their infection status are unable to benefit from interventions, which can reduce the risk of mother-to-child-transmission (MTCT) of HIV to well under 2%. As of December 2004, 934 children in the UK had been reported with confirmed HIV infection. 75% of these children were living within the London area.

Prevention of mother-to-child-transmission (MTCT) of HIV – an overview

With the improved uptake of antenatal HIV testing, identification of infected women during pregnancy has increased throughout the UK. At the same time MTCT of HIV has been dramatically reduced by providing antiretroviral therapy (ART) to mother antenatally, during delivery and to the baby postnatally; delivery by elective caesarean section (ELCS) and by avoidance of breast-feeding. These interventions reflect the recommendations of many preceding studies including the landmark trial from the Paediatric AIDS Clinical Trials Group (PACTG 076), which demonstrated the efficacy of Zidovudine (AZT) in pregnancy & postnatally, reducing vertical transmission by 67%.

Risk factors for MTCT of HIV

Maternal HIV viral load (VL) appears to be the primary determinant in the risk of transmission with a higher VL increasing the risk. Other risk factors include advanced maternal HIV disease, vaginal delivery, prolonged rupture of membranes (>4 hours); premature delivery (<36/40); breastfeeding, chorioamnionitis, placental abruption and foetal scalp monitoring.

Specific UK guidelines

The guidelines below are edited from the current pregnancy guidelines of the British HIV Association (www.bhiva.org).
**TESTING POLICY**
The Department of Health recommends that all women should be offered HIV testing during pregnancy. Women have the option to decline testing after counselling. Some women are already aware of their status, and may have conceived on combination ART. All units should aim to be achieving over 90% of women routinely tested for HIV in pregnancy.

It is good practice for a senior member of the obstetric, midwifery, adult HIV and paediatric team to meet to discuss HIV infected women’s care antenatally, and produce a clear birth plan.

**ANTENATAL, INTRAPARTUM AND POSTNATAL GUIDELINES BASED ON POSSIBLE SCENARIOS**

If women are identified during pregnancy, the treatment offered is dependent on their disease status.

1. Women with non/slowly progressive disease (CD4 >2-300 and VL<10,000 copies/mL), and naïve to Highly Active ART (HAART).

   (a) Antenatally, women may be offered Zidovudine (or AZT) monotherapy or PI-based Short-Term ART (START) in the 2nd trimester between 20-32/40.

   (b) i) With AZT monotherapy, maternal VL is unlikely to be < 50 copies/mL, so offer elective Caesarean section (PLCS) at 38/40 and intrapartum AZT. The **dose of maternal intravenous AZT is 2mg/kg for 1 hour**, followed by **1 mg/kg per hour** until the cord is clamped. With PLCS the infusion should be started 4 hours before the operation.

   ii) At 36/40 if VL < 50 copies/mL in a pregnant women receiving HAART or START, oral HAART should be continued during delivery. Infant can be delivered vaginally. The benefit of PLCS has been clearly demonstrated. However, it is unclear as to whether this intervention provides significant added benefit for women on HAART with an undetectable viral load. Intrapartum AZT is no longer routine in this scenario.

   iii) At 36/40 if VL > 50 copies/mL in a pregnant women receiving HAART or START, obtain a genotype and change therapy to the best available option using expert advice. The infant should be delivered by PLCS at 38/40, and IV AZT offered at the above dose [1 (b) i], provided no AZT resistance is documented on the genotype.

   (c) i) Term infants born to women in scenarios 1(b) i and 1(b) ii should receive oral AZT **monotherapy 4mg/kg/dose twice daily for 4 weeks** postnatailly.

   ii) If the infant is born prematurely at >30/40, scenario 1 (b) iii is likely to apply, the dose of oral AZT is **2mg/kg/dose twice daily for 2 weeks**, then **2mg/kg/dose three times daily for 2 weeks** as part of combination post-exposure prophylaxis.
iii) If the infant is born prematurely at <30/40, scenario 1 (b) iii is likely to apply, the dose of oral AZT is **2mg/kg/dose twice daily for 4 weeks**.

iv) In sick infants unable to tolerate oral medication if at term provide **intravenous AZT at 1.5 mg/kg/dose four times daily** and **1.5 mg/kg/dose twice daily** in a premature infant (<36 weeks).

v) Combination post-exposure prophylaxis should be given to infants born to women where scenario 1 (b) iii applies. Combination post-exposure prophylaxis includes **AZT for 4 weeks** (doses discussed above) + **Lamivudine** (or 3TC) at **2mg/kg/dose twice daily for 4 weeks** + **Nevirapine** at **2mg/kg OD from birth to 7 days** and **Nevirapine 4mg/kg OD for 7 – 14 days**. Use **Nevirapine 4mg/kg OD for 2 weeks** if the mother has received **>3 days of Nevirapine**. Nevirapine has a long half-life and is not continued beyond 2 weeks as part of combination post-exposure prophylaxis.

2. Women with advancing disease and naïve to HAART (CD4 >2-300 & VL >10,000 copies/mL)

   (a) Antenatally, women should be offered PI-based START in the 2nd trimester between 20-32/4.

   (b) i) At 36/40 if VL < 50 copies/mL in a pregnant women receiving START, oral HAART should be continued during delivery. Infant can be delivered vaginally. Intrapartum AZT is no longer routine in this scenario.

   ii) At 36/40 if VL > 50 copies/mL in a pregnant women receiving START, obtain a genotype and change therapy to the best available option using expert advice. The infant should be delivered by PLCS at 38/40, and IV AZT offered at the above dose [1 (b) i)], provided no AZT resistance is documented on the genotype.

   (c) i) Infants born to women in scenario 2(b) i should receive oral AZT for 4 weeks postnatally if maternal HAART includes AZT.

   ii) If maternal HAART does not contain AZT, AZT may be used provided there is no documented maternal resistance to this NRTI or provided d4T does not constitute part of maternal HAART.

   iii) AZT should not be given to an infant born to a mother who is receiving d4T because of the theoretical negative competitive interaction. The **dose of d4T** (if this NRTI is used as postnatal prophylaxis) is **1mg/kg dose twice daily**.

   iv) Avoid ddI (if possible) as part of postnatal prophylaxis because of the feeding restriction associated with using this NRTI, since ddI is much better absorbed on an empty stomach. The **dose of ddI** (if used as postnatal prophylaxis) is **60mg/m²/dose twice daily for monotherapy**, and **100mg/m² once daily as part of combination post-exposure prophylaxis with nelfinavir**. The **dose of nelfinavir** (if used as postnatal prophylaxis) is **50-75mg/kg twice daily**.
v) Combination post-exposure prophylaxis with **AZT, 3TC and nevirapine** should be offered to infants born to women in scenario 2 (b) ii. See scenario 1 (c) for dosages.

vi) If maternal HAART does not contain AZT, use the NRTI most often used within the mother’s regimen. Generally, 3TC>d4T>Abacavir>ddI.

vii) The dose of Abacavir is **2mg/kg twice daily** (if used as postnatal prophylaxis as monotherapy). The associated hypersensitivity reaction has not been documented in infants although only small numbers have been treated.

3. Women with more advanced disease and naïve to HAART (CD4 <200 and VL >10,000 copies/mL).

   (a) Should be commenced on HAART (usually AZT-containing) after the 1st trimester.

   (b) i) At 36/40 if VL < 50 copies/mL in a pregnant women receiving HAART, treatment should be continued during delivery. Infant can be delivered vaginally. Intrapartum AZT is no longer routine in this scenario.

   ii) At 36/40 if VL > 50 copies/mL in a pregnant women receiving HAART, obtain a genotype and change therapy to the best available option using expert advice. The infant should be delivered by PLCS at 38/40, and IV AZT offered at the above dose [1 (b) i)], provided no AZT resistance is documented on the genotype.

   (c) i) Infants born to women in scenario 3 (b) i should receive oral AZT **4mg/kg/dose twice daily for 4 weeks** postnatally if maternal HAART includes AZT.

   ii) If maternal HAART does not contain AZT, AZT may be used provided there is no documented maternal resistance to this NRTI or provided d4T does not constitute part of maternal HAART (see 2 (c) ii).

   iii) Combination post-exposure prophylaxis with **AZT, 3TC and nevirapine** should be offered to infants born to women in scenario 3 (b) ii. See scenario 1 (c) for dosages

4. Women who present at any time in pregnancy on effective HAART (VL <50 copies/mL, or increasing CD4 counts).

   (a) Should continue HAART with their current regimen. New BHIVA guidelines recommended continuation with efavirenz as there are no human data to suggest an increased risk of neural tube abnormalities. Furthermore switching to nevirapine as an alternative NNRTI may risk additional toxicity from hepatitis or skin rash, particularly if the mother’s CD4 count has been increased due to her prior antiretroviral therapy.
(b) i) At 36/40 if VL < 50 copies/mL in a pregnant women receiving HAART, treatment should be continued during delivery. Infant can be delivered vaginally. Intrapartum AZT is no longer routine in this scenario.

ii) At any stage if VL > 50 copies/mL in a pregnant women receiving HAART, obtain a genotype and change therapy to the best available option using expert advice. If VL reverts to < 50 copies/mL as a result follow 4 (b) i. If VL remains > 50 copies/mL, the infant should be delivered by PLCS at 38/40, and IV AZT offered at the above dose [1 (b) i]], provided no AZT resistance is documented on the genotype.

(c) i) Infants born to women in scenario 4 (b) i should receive oral AZT 4mg/kg/dose twice daily for 4 weeks postnatally if maternal HAART includes AZT.

ii) If maternal HAART does not contain AZT, AZT may be used provided there is no documented maternal resistance to this NRTI or provided d4T does not constitute part of maternal HAART (see 2 (c) ii).

iii) Combination post-exposure prophylaxis should be offered to infants born to women in scenario 4 (b) ii. Seek expert advice from the network lead for the appropriate combination.

5. Women who present on non-suppressive HAART

(a) At 36/40 if VL > 50 copies/mL in a pregnant women receiving HAART, obtain a genotype and change therapy to the best available option using expert advice.

(b) The infant should be delivered by PLCS at 38/40, and IV AZT offered at the above dose [1 (b) i]], provided no AZT resistance is documented on the genotype.

(c) Combination post-exposure prophylaxis should be offered to all infants born to women in scenario 5. Seek expert advice from the network lead for the appropriate combination.

6. Women who present late after 32/40 but before delivery, with advancing (VL > 10,000 copies/mL) or advanced disease (CD4 < 200), and are naïve to HAART.

(a) Should be commenced on HAART ASAP.

(b) i) At 36/40 if VL < 50 copies/mL, treatment should be continued during delivery. Infant can be delivered vaginally. Intrapartum AZT is no longer routine in this scenario.

ii) At 36/40 if VL > 50 copies/mL, obtain a genotype and change therapy to the best available option using expert advice. The infant should be delivered by PLCS at 38/40, and IV AZT offered at the above dose [1 (b) i]], provided no AZT resistance is documented on the genotype.
(c) i) Infants born to women in scenario 6 (b) i should receive oral AZT 4mg/kg/dose twice daily for 4 weeks postnatally.

ii) Combination post-exposure prophylaxis for 4 weeks should be offered to infants born to women in scenario 6 (b) ii. Seek expert advice from the network lead for the appropriate combination.

7. Women who present in labour with intact or ruptured membranes with VL>50 copies/mL, CD4 <250 cells/µL or unknown CD4 count and VL, and are naïve to HAART

(a) Should be commenced on HAART (Combivir and Nevirapine) ASAP prior to intrapartum AZT. Obtain blood for CD4, VL and resistance assay. Give a STAT oral dose of nevirapine (200 mg orally at onset of labour) because of a very rapid antiviral effect and high transplacental concentrations. Start antibiotics early with rupture of membranes.

- Please see the Trust antibiotic guidelines

(b) Optimise obstetric management (antibiotics stat with rupture of membranes and emergency CS if not about to deliver, otherwise active management of labour) and infuse IV AZT at the above dose [1 (b) i)].

(c) Combination post-exposure prophylaxis with AZT, 3TC (for 4 weeks) and nevirapine (for 2 weeks) should be offered to all infants born to women in scenario 7.

8. Women who present in labour with intact or ruptured membranes with VL>50 copies/mL, CD4 <250 cells/µL or unknown CD4 count and VL, and are HAART-experienced.

(a) Seek expert advice to optimise HAART and give a STAT oral dose of nevirapine (200 mg orally at onset of labour). Start antibiotics early with rupture of membranes.

(b) Optimise obstetric management (antibiotics stat with rupture of membranes and emergency CS if not about to deliver, otherwise active management of labour) and infuse IV AZT at the above dose [1 (b) i)].

(c) Combination post-exposure prophylaxis should be offered to all infants born to women in scenario 8. Seek expert advice from the network lead for the appropriate combination.

9. Women who present in labour with intact or ruptured membranes with VL<50 copies/mL and are HAART-experienced.

Optimise obstetric management and follow the same guidelines as for women on effective HAART in scenario 4. Postnatally provide the monotherapy component of the mother's regimen for 4 weeks. Generally, 3TC>d4T>Abacavir>ddI – see 2 (c) vi.
10. Women who are diagnosed after delivery.

Combination post-exposure prophylaxis with AZT, 3TC (for 4 weeks) and nevirapine ASAP in the newborn (for 2 weeks).

Postnatal infant ART is much less effective in reducing MTCT of HIV after 48 hours.

11. Women who present in labour with an unconfirmed positive HIV test and are treatment naïve.

Same management as in scenario 7.

**INDICATIONS FOR COMBINATION POSTNATAL PROPHYLAXIS**

1. Women who first present in labour or after birth naïve to HAART.

2. If maternal Viral Load either is, or is predicted to be > 50 copies/ml at delivery.

3. If maternal CD4 count continues to decline below 200 in the 2nd & 3rd trimester (and VL cannot be obtained).

4. If HAART has been refused by the mother during her pregnancy. Note if the mother strongly opposes combination post-exposure prophylaxis in the newborn after counselling, a treatment order should be considered.

5. If the mother has received < 4 weeks of ART during pregnancy either through late booking or premature delivery.

6. Consideration should be given for combination post-exposure prophylaxis if the intrapartum AZT (when indicated) is not administered to the expectant mother, or with documented risk factors (prolonged rupture of membranes (>4 hours); premature delivery (<36/40); breastfeeding, chorioamnionitis, placental abruption and foetal scalp monitoring).

**PNEUMOCYSTIS JIROVECII PNEUMONIA (PCP) PROPHYLAXIS**

As transmission rates for mothers who fully take up interventions in pregnancy in some centres is <1% it is no longer necessary to administer co-trimoxazole to these infants as PCP prophylaxis. Generally, infants who received AZT monotherapy do not need to take co-trimoxazole.

Infants who are at higher risk of becoming infected, because there is inadequate control of maternal VL, should receive co-trimoxazole. Generally, infants who received combination post-exposure prophylaxis need to start co-trimoxazole after stopping their ART at 4 weeks of age.

Generally for most term infants body surface area 0.25 m\(^2\), the dose will be 120 mg once daily, and for term infants over 6 months, the dose will be 240 mg once
daily. Septrin is given once daily 3 times a week (Monday, Wednesday, Friday) until 3 negative PCR’s.

**OTHER NON-PHARMACOLOGICAL INTERVENTIONS**

Breast-feeding approximately doubles the risk of vertical transmission and the risk increases with the duration of feeding. Therefore all HIV positive mothers should be strongly encouraged to formula feed and to not start breast-feeding. If breast-feeding has been initiated, women should be encouraged to discontinue ASAP. The formula feed, bottles and sterilizer should be provided particularly for asylum seeking mothers.

**Teratogenic effects and toxicities associated with ART in infants and follow-up investigations**

Effective intervention with perinatal AZT, PLCS and exclusive formula feeding has reduced vertical transmission rates to <1%. There is however limited safety data currently available on the use of other antiretrovirals in pregnancy and a cautious approach to the use of HAART in early pregnancy is recommended.

Nevertheless to date, neither an increase in total number, nor any specific foetal abnormality has been identified. No long-term increased risk of carcinogenicity has been reported after NRTI exposure. So far, no adverse developmental effects of ART exposure have been demonstrated in children. Reported short-term side effects in asymptomatic ART-exposed infants include acute mitochondrial toxicity and lactic acidosis, elevated transaminases, and symptomatic anaemia.

**Testing and follow-up (Figure 1)**

Infants born to mothers with HIV will have passively acquired IgG antibodies to HIV-1. Blood should therefore be obtained from the baby in the first day or two of life for HIV-1 DNA, amplified by polymerase chain reaction (PCR) (at least 1ml in an EDTA bottle and not from cord blood).

HIV RNA PCR (or viral load) may lead to false positive results and is not ideal at birth, but can be useful later in infancy especially in settings where HIV DNA amplification is not possible.

A sample of the mother’s blood (EDTA) should be sent with the baby’s first PCR to ensure the PCR primers used can detect maternal virus. The infant should receive a full neonatal check prior to discharge.

The second PCR is taken at 6 weeks of age (at least 2 weeks after stopping ART).

The third PCR should be taken at 3-4 months of age.

Infants who received triple ART should have a fourth PCR taken at 4-6 months of age.

A HIV antibody test should be taken at 18 months of age (2mls clotted blood).
Baseline investigations in the ART-exposed newborns should include full blood count, liver function tests, and a urine sample to detect cytomegalovirus (CMV) if possible. Serum pH and lactate should be obtained in an unwell ART-exposed newborn.

![Figure 1: Investigation of the indeterminate infant.](image)

If the baby has three negative PCR’s there is a 98-99% chance that the baby is not infected with HIV. It must be stressed however, that a negative HIV diagnosis cannot be 100% confirmed until the final 18-month HIV antibody test is performed. For this reason the parents should be encouraged to make contact if they are concerned about their child’s health during this period.

**National Surveillance**

All infants born to mothers with HIV should be reported through the British Paediatric Surveillance Unit (BPSU) study of HIV in children using the “orange card” system. As the majority of these infants will have been exposed to ART both in utero and in the first weeks of life, they will be followed up clinically each year to school age to monitor for long-term effects. This is currently a pilot study by the National Study of HIV in Pregnancy and Childhood (NSHPC) but is likely to be national soon.

Please telephone one of the Paediatric HIV consultants as soon as possible if a positive PCR test is identified, as the ART may need to be altered. Contact details for our unit are provided on Page 3 of this manual.

**Vaccinations**

It is considered safe for the baby to continue the usual vaccination programme, and to have Hepatitis B if indicated. Subsequently, anti-HBs levels should be checked yearly and booster doses given if the anti-HBs level falls below 100i.u./l
BCG vaccination should only be given when the infant has had 3 negative PCR’s.

**Figure 2: Protocol for Infants of HIV Positive Mothers.**

*Note: The figure summarises the steps: full details are in the text, which should be consulted as the algorithm is not a replacement.*

**Protect yourself**

**DELIVERY**

WEAR APPROPRIATE PROTECTIVE CLOTHING

**Resuscitate as necessary**

**RESUSCITATION**

(as atraumatic as possible)

**Clarify the level of risk for the baby**

**LOWER RISK BABY**

(SEE TEXT)

**HIGH RISK BABY**

(SEE TEXT)

**Treatment to start on day 1**

**Zidovudine** for 4 weeks

(See text for doses)

IM Vitamin K after bathing in water and drying

**Zidovudine** for 4 weeks

**Lamivudine** for 4 weeks

**Nevirapine** for 2 weeks

(See text for doses)

**OR**

Other HAART dependant on viral resistance etc

IM Vitamin K after bathing in water and drying

**Bloods to be taken**

- Baseline HIV markers from the infant should be taken within 48 hours of birth, but **not** from cord blood.
- At least 1 ml of EDTA blood should be sent to Virology, for ‘Pro-Viral DNA’.
- A FBC should be sent.
- A sample of the mother’s blood (8 ml in EDTA) should be sent with the baby’s first Pro-Viral DNA, to ensure the PCR primers used can detect the maternal virus.

**Arrangements for follow up**

**LONG TERM FOLLOW UP: DR …………**

Ask for a 1st follow up appointment at 6 weeks of age
Disease progression in HIV-infected children

20% of AIDS deaths globally are in children. There is a bimodal distribution: 25% develop AIDS within 1 year, with a median time for 75% ~ 7 years. Perinatally infected infants and children with HIV-1 have accelerated disease progression (compared with adults) as a result of a higher viral set-point and an active thymus (with a larger pool of cells permissive to HIV-Infection). In addition, naïve T cells have an impaired functional phenotype and are unable to process pathogens effectively.

Goals of therapy

The goals of HAART are to maximally reduce plasma VL below the limit of detection (<50 copies/mL), prevent selection of drug resistant strains and maintain good immunological status (repopulation with CD4+ naïve T cells) to prevent clinical disease progression and a good quality of life.

Therapeutic challenges

The difficulties with implementation of HAART and adherence relate to social circumstances, excess pill burden, and poor taste; variable absorption and metabolism of drugs, drug-drug interactions and toxicities and the emergence of drug resistant virus.

Challenges therefore include: 1. Introduction of suitable formulations, in the setting of previous ART-exposure and immunosuppression, which minimise cost, toxicity, & drug interactions. 2. Effective salvage therapy after virological, immunological, and clinical failure. 3. Treating Adolescents. 4. Disclosure of diagnosis.

INFANTS

When to start HAART in infants (< 12 months of age)

Always: CDC Stage C, CD4<25% or rapid decrease in CD4% (irrespective of number) and/or VL persistently > 10⁶ copies/mL and/or clinical signs and symptoms including HIV-encephalopathy and neurodevelopmental delay.

Consider: ART in any infant irrespective of clinical/ immunological stage.
Problems with administering HAART to infants

These include variable drug administration, absorption & metabolism; maternal ART and vertical transmission of drug resistant virus; acceptability and palatability of formulations; refrigeration of syrup formulations in warm climates.

Therapeutic options in infants

1. **AZT + 3TC + Abacavir (ABC) + Nevirapine (NVP)** – well tolerated and very potent.

   Within PHILSnet this is our preferred option as 1st line HAART in infants.

Disadvantage: Both ABC and NVP cause a rash and ABC hypersensitivity may very rarely be fatal with re-challenge. If an infant develops fever & rash on this combination, it is important to admit the infant, investigate thoroughly with blood cultures etc, and strongly consider withdrawing ABC if there are no signs of infection. Discuss with network lead.

   ABC hypersensitivity is more commonly reported in Caucasians. A report of hypersensitivity to ABC among 18 caucasian individuals found the 57-1 haplotype (defined by the presence of HLA-B*5701, HLA-DR7, & HLA-DQ3) to be highly predictive of ABC hypersensitivity.

2. **3TC + ABC + NVP**: Also well tolerated and efficacious in reducing and maintaining undetectable VL in infants and children. Advantage: AZT & ddI are spared as an NRTI backbone for a 2nd line regimen.

Disadvantages: ABC hypersensitivity.

We have commonly recorded lipodystrophy syndrome (LDS) in children with exposure to Stavudine (d4T) and didanosine (ddI) and this NRTI combination is no longer recommended.

CHILDREN

When to start HAART in children > 12 months and < 8 years of age

**Always:** Clinical Stage C or CD4 < 15%.

**Consider:** Clinical Stage B or CD4 < 20% or VL > 5 log.

**Defer:** Clinical Stage N or A, CD4 > 20%, VL < 5 log.

When to start HAART in children > 8 years of age
CD4% is less helpful. It is better to rely on absolute CD4 number (like adults).

**Always:** Clinical Stage C or CD4 < 200 cells/µl.

**Consider:** Clinical Stage B or CD4 < 500 cells/µl or VL > 5 log

**Defer:** Clinical Stage N or A. CD4 > 500 cells/µl, and VL < 5 log.

**Therapeutic options in children > 12 months**

1. **3TC + ABC + NNRTI (EFV or NVP)** –

   **Within PHILSnet this is our preferred option as 1st line HAART.**

   Advantages: Potent and AZT & ddI form a 2nd line NRTI backbone.

   Disadvantages: ABC hypersensitivity.

   3TC and ABC can now be given once daily (PENTA 13), so this can be a once daily regimen. A fixed dose combination consisting of 3TC and ABC is now available called Kivexa. Efavirenz is not given to children under 3 years of age. There is little evidence for improved efficacy between EFV or NVP. The decision is based on formulation, licensing (EFV 6 years and older only) and predicted toxicity (NVP-hepatitis, EFV - CNS).

2. **AZT + 3TC + ABC:** superior to AZT + 3TC after 24 weeks. Well tolerated. However, ABC Hypersensitivity in 2%, and may be less potent compared to AZT + 3TC + ABC + EFV (ACTG 5095 Gulick et al).

3. **Kaletra (Lopinavir with Ritonavir [RTV boosting]) + 2 NRTIs.**

   Advantages: Potent, safe and well tolerated.

   Disadvantage: pill burden, taste, and toxicity

**Changing regimens**

Indicated with the emergence of drug-resistant strains associated with clinical or immunological failure, and concurrent virological failure.

Depends on prior drug history, toxicity and availability of new drugs, and guidance from resistance testing.

**PHILSnet regimens for children 2005**

1. **1st line:**
   3TC + ABC + NNRTI (Either EFV or NVP)

2. **2nd line:**
   AZT + ddI + Kaletra (NFV if Lopinavir intolerant).
Summary of drug dosages and toxicity

<table>
<thead>
<tr>
<th>Name of drug</th>
<th>Total daily dose (frequency)</th>
<th>Major toxicities</th>
<th>Other comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nucleoside Reverse Transcriptase Inhibitors (NRTIs)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zidovudine (AZT)</td>
<td>Oral 360 mg/m²/day (divide bid)</td>
<td>Neutropenia; anaemia; nausea; headaches; myopathy (rare).</td>
<td>Large volume of syrup not well tolerated in older children. Double dose for HIV encephalopathy.</td>
</tr>
<tr>
<td>Didanosine (ddl)</td>
<td>240 mg/m²/day (od or divide bid). (&gt; 60 kg 200 mg bid or 400 mg qd). 125/200/250/400 mg capsules qd for older children.</td>
<td>Pancreatitis rare (dose related); peripheral neuropathy rare (dose related); diarrhoea and abdominal pain: LDS in association with d4T. Lactic acidosis.</td>
<td>Constituted suspension stable for 30 days in fridge. Ideally taken 1 h before food or 2 h after, but may be less important in children.</td>
</tr>
<tr>
<td>Zalcitabine (ddC)</td>
<td>0.03 mg/kg/day (divide bid).</td>
<td>Headache, GI upset, peripheral neuropathy; pancreatitis rare in children; hepatic toxicity; oral ulcers.</td>
<td>Small tablets. Rarely used in Paediatric HIV therapy. Not to be used with AZT.</td>
</tr>
<tr>
<td>Stavudine (d4T, Zerit)</td>
<td>2 mg/kg/day (up to 30 kg) (divide bid) 30-60 kg - 30 mg bid &gt; 60 Kg, 40 mg bid.</td>
<td>LDS (common). Lactic acidosis/hepatic steatosis. Headache, GI upset, rash, peripheral neuropathy and pancreatitis (rare).</td>
<td>Less often used because of LDS, lactic acidosis/ hepatic steatosis. Large volume of suspension.</td>
</tr>
<tr>
<td>Lamivudine (3TC, Epivir)</td>
<td>8 mg/kg/day (divide bid). In neonates &lt; 30 days 4 mg/kg/day q 12 hours. &gt; 60 Kg 150 mg (bid) Combivir ZDV 300 mg + 3TC 150 mg</td>
<td>Headache, abdominal pain, pancreatitis, peripheral neuropathy, and neutropenia, abnormal LFTs - all rare.</td>
<td>Well tolerated. Store solution at room temperature (use within 1 month of opening).</td>
</tr>
<tr>
<td>Abacavir (ABC)</td>
<td>16 mg/kg/day (divide bid). Adult 300 mg bid. Trizivir - ZDV 300 mg + 3TC 150 mg + ABC 300 mg</td>
<td>1-3% may develop hypersensitivity: fever, malaise, mucositis ± rashes, usually in first 6 weeks STOP DRUG - DO NOT RECHALLENGE.</td>
<td>Syrup well tolerated or crush tablets. MUST WARN PARENTS ABOUT HYPERSENSITIVITY REACTION.</td>
</tr>
<tr>
<td><strong>Non-NRTIs: SEE ADDENDUM AFTER THIS TABLE</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nevirapine (NVP)</td>
<td>400 mg/m²/day (divide bid). Maximum 400 mg qd. For 1st 14 days give half dose ie 200 mg/m²/day then if no rash increase to full dose.</td>
<td>Rash 10-20% can treat through, Stevens-Johnson very rare, but STOP drug. Hepatotoxicity - monitor liver enzymes. Induces cytochrome P450. Drug interactions.</td>
<td>Can be given with food. Few data on use with PI. Practice is to increase PI dose by about 30%.</td>
</tr>
<tr>
<td>Efavirenz (EFV)</td>
<td>See addendum</td>
<td>Rash (mild). CNS toxicity,</td>
<td>Syrup available. Best given as bedtime</td>
</tr>
<tr>
<td>Name of drug</td>
<td>Total daily dose (frequency)</td>
<td>Major toxicities</td>
<td>Other comments</td>
</tr>
<tr>
<td>--------------------------</td>
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</tr>
<tr>
<td>Delavirdine (DLV, Rescriptor)</td>
<td>Paediatric dose under study. Adult dose 600 mg bid.</td>
<td>Somnolence, abnormal dreams. Drug interactions.</td>
<td>Dosing to reduce CNS side-effects. Dispersible tablets can be dissolved in water/cola.</td>
</tr>
<tr>
<td><strong>Protease inhibitors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indinavir (IDV, Crixivan)</td>
<td>Do not use in neonates. 1500 mg/m²/day (divide tid). Adult 800 mg every 8 h.</td>
<td>Nausea; hyperbilirubinaemia (10%) Renal stones/nephritis (4%); haemolytic anaemia, liver dysfunction rare. Abnormal lipids.</td>
<td>Do not take with meals. Rarely used in Paediatrics. Complex formula for syrup available. Drug interactions.</td>
</tr>
<tr>
<td>Ritonavir (RTV)</td>
<td>800 mg/m²/day (divide bid). Start with 250 mg/m²/dose q 12 hours and ↑ over 5 days. Infants 900 mg/m²/day. (Syrup 80 mg/mL).</td>
<td>GI intolerance ++, headache; increased liver enzymes; abnormal lipids.</td>
<td>Take with food but liquid tastes bitter. Can help to take with peanut butter and follow with chocolate sauce or cheese. Drug interactions.</td>
</tr>
<tr>
<td>Saquinavir (SQV, Fortovase)</td>
<td>150 mg/kg/day (divide tid).</td>
<td>Rash; headache; GI upset; abnormal lipids.</td>
<td>Give with food. Sun photosensitivity. Drug interactions.</td>
</tr>
<tr>
<td>Nelfinavir (NFV, Viracept)</td>
<td>120 mg/kg/day (divide bid). Adolescents need &gt; than adult doses. Crush tablets; powder available. Infants 150 mg/kg/day.</td>
<td>Mild/moderate diarrhoea; vomiting; rash; abnormal lipids. LDS with ddI and d4T.</td>
<td>Take with food. Drug interactions.</td>
</tr>
<tr>
<td>Lopinavir/ritonavir (LPV/r, Kaletra)</td>
<td>450/112.5-600/150 mg/m²/day (divide bid). Higher dose used with NNRTI. (Syrup 80/20 mg/mL).</td>
<td>Rash (2%), GI intolerance, abnormal lipids.</td>
<td>Liquid formulation - low volume bitter taste. Capsules large. Take with food. Drug interactions.</td>
</tr>
<tr>
<td>Amprenavir (APV, Agenerase)</td>
<td>40 mg/kg/day (divide bid) capsules Increase dose for syrup.</td>
<td>GI upset. Abnormal lipids.</td>
<td>Large volume of syrup - bitter taste. 150 mg capsules are very large; alternatively many small 50 mg capsules may be taken.</td>
</tr>
</tbody>
</table>

*bid, twice daily; GI, gastrointestinal; IV, intravenously; LFT, liver function tests; qd, once daily; PK, pharmacokinetic; tid, three times daily.
## Summary of Prescribing and Administration Information for NNRTIs

<table>
<thead>
<tr>
<th>Names of Drug</th>
<th>Dosage (Oral unless specified)</th>
<th>Formulations/Special Instructions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nevirapine (NVP, Viramune)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neonatal (&lt;30 days)</td>
<td>Inadequate data but 2-5mg/kg OD has been used. Post Exposure Prophylaxis (combined with 2 NRTIs) 2mg/kg OD for 14 days then stop due to long half-life. Continue NRTIs for 4 weeks in total. If treatment is to continue increase to 4 – 5mg/kg OD after 1 4 days &amp; increase again at 2 months</td>
<td>Tablets: 200mg Suspension: 10mg in 1ml Few data on use with PI. Practice is to increase PI dose by about 30%. Suspension: Shake well. Store at room temperature</td>
</tr>
<tr>
<td>Infant (1 - 12 months)</td>
<td>Inadequate data. 150-200 mg/m²/day OD for 14 days then, if no rash, increase to 300-400mg/m²/day in 2 divided doses. Alternatively: 2 months – 8 years: 4mg/kg OD for 14 days then 7mg/kg BD. Maximum 400mg daily. 8-16 years &amp; &lt;50kg: 4mg/kg for 14 days then 4mg/kg BD. ≥50kg: adult dose.</td>
<td></td>
</tr>
<tr>
<td>Paediatric (Tanner Stages 1 - 3)</td>
<td>Over 16 years: 200mg OD for 14 days then 200mg BD 400mg OD unlicensed</td>
<td></td>
</tr>
<tr>
<td>Adolescent (Tanner Stages 4 -5) / Adult</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Tablet: 200mg Suspension: 10mg in 1ml
Few data on use with PI.
Practice is to increase PI dose by about 30%.
Suspension: Shake well. Store at room temperature
| Drug               | Unknown | Inadequate data in children <3 years or <13kg. | 600mg OD | Capsules: 50mg, 100mg, 200mg  
Tablets: 600mg  
Oral solution: 30mg in 1ml |
|--------------------|---------|-----------------------------------------------|---------|----------------------------------------------------------------------------------|
| Efavirenz (EFV, Sustiva) | Unknown | **Capsules:**  
**Over 3 years:**  
13 - 15kg - 200mg OD  
15 - 20kg - 250mg OD  
20 - 25kg - 300mg OD  
25 - 32.5kg - 350mg OD  
32.5 - 40kg - 400mg OD  
Over 12 years and / or ≥ 40kg : 600mg OD  
**Oral Solution:**  
**Over 5 years:**  
13 - 15kg - 270mg OD (9ml)  
15 - 20kg - 300mg OD (10ml)  
20 - 25kg - 360mg OD (12ml)  
25 - 32.5kg – 450mg OD (15ml)  
32.5 - 40kg – 510mg OD (17ml)  
≥ 40kg : 720mg OD (24ml)  
**3 - <5 years:**  
13 - 15kg - 360mg OD (12ml)  
15 - 20kg - 390mg OD (13ml)  
20 - 25kg - 450mg OD (15ml)  
25 - 32.5kg – 510mg OD (17ml)  
32.5 – 40kg – 400mg OD  
15mg/kg OD |
| Delavirdine (DLV, Rescriptor) | Unknown | Unknown | 400mg TDS or 600mg BD | Tablets: 100mg, 200mg  
Rarely used.  
100mg tablets can be dispersed in water or cola & taken promptly. 200mg tablets are not readily dispersible.  
Should be taken one hour before or after ddl or antacids. |
Lactic acidosis and severe hepatomegaly with steatosis from mitochondrial toxicity is most often associated with prolonged therapy (>6 months) with ddI and d4T. However, other NRTIs alone and in combination, and Tenofovir have also been implicated.

Other toxicities include osteopenia and osteonecrosis, associated with prolonged NRTI usage, and cardiomyopathy, and renal tubular leak with Tenofovir.

**Guidelines for the management of possible allergic reactions on commencing the 3TC, Abacavir, NNRTI regimen**

The potential area of concern is that both Abacavir and Nevirapine can produce hypersensitivity reactions. Both reactions can present in the most severe form with fever and systemic illness and in children particularly over the winter respiratory viral period, minor upper respiratory infections could mimic the early phases of hypersensitivity reactions to these agents. However, losing these drugs, both of which are extremely useful in paediatric antiretroviral practice, if a child has a minor cold can severely compromise their future long-term HIV management. Therefore before stopping any antiretroviral the following issues need to be considered.

**Nevirapine hypersensitivity:** this is an idiosyncratic reaction to the drug and is not dose related. A mild rash, looking like an allergic macular, papular rash is common, around 10% when first starting Nevirapine. This is usually on the trunk and limbs and is itchy, but the child is systemically well with no fever and no other clinical signs. This most commonly occurs within the first two weeks of starting therapy. Very rarely (approximately 1 in 300) a child can progress to develop a full Stephen-Johnson Syndrome. Signs of this would include severe conjunctivitis, mucositis, desquamation, peeling, oedema and a systemically unwell child. This is extremely rare in children. The rash can be safely managed with a non-sedating antihistamine, for example Loratadine given for 3 to 5 days with daily monitoring of the child is adequate. If the rash then fades to normal there is no indication to stop the Nevirapine.

Occasional rare hepatitis or severe hepatitis has been reported as part of the Nevirapine hypersensitive reaction, which may not be associated with a rash. Routine liver functions tests should be monitored on all children starting Nevirapine. These should be checked between 2 – 4 weeks of commencing therapy and again in a month’s time. Evidence of a markedly rising ALT is taken as evidence of Nevirapine hypersensitivity reaction and the drug needs to be stopped. Routine monitoring of liver function tests should continue on all patients on a NNRTI every 2 to 3 months.

Abacavir hypersensitivity reactions are also less common in children than adults. There is evidence of lower frequency in those of black African ethnic background. It is likely that severe Abacavir sensitivity reactions occur only in around 1-2% of children starting Abacavir. These usually occur from between 2 – 6 weeks after commencing Abacavir. The clinical features are that the child is most commonly systemically unwell with a fever as the predominant clinical sign. They may or may not have a rash, but severe Abacavir hypersensitivity reactions have been
reported in the absence of a rash. Other symptoms may include respiratory symptoms with a cough and wheeze, gastrointestinal symptoms with vomiting or diarrhoea, listlessness, fatigue and flu-like symptoms often predominate. Severe Abacavir hypersensitivity reactions occur when Abacavir continues to be given as the patient clinically deteriorates.

If a child has a minor upper respiratory tract infection and is symptomatically improving it is completely safe to continue Abacavir. Indeed, Abacavir should not be stopped for children with mild viral upper respiratory tract infections. If the child is Abacavir allergic and then re-starts Abacavir after treatment interruption then this can exacerbate a severe formulate hypersensitivity reaction. If a child does not have an Abacavir hypersensitivity reaction, but the parents stop the drug there is no risk to re-starting this again after a period of treatment interruption. All attempts therefore, should be made to confirm a virological or bacteriological diagnosis if a child is unwell. If the illness can be clarified as being due to influenza or other respiratory virus then there is no risk to re-starting the Abacavir. If there is any doubt children should be admitted to hospital and monitored very closely. Signs that would suggest an Abacavir hypersensitivity reaction would include deteriorating liver function tests with rising ALT, increasing lactate and steady clinical deterioration. This usually occurs over the course of a few days, but given the rarity of this reaction the priority is to try and confirm another clinical diagnosis for the child’s intercurrent illness.

If a child is on a 3TC, Abacavir, Nevirapine containing regimen and has an illness of sufficient severity that stopping therapy is warranted then all drugs should be stopped.

**Formula to calculate body surface area (BSA in m2)**

\[
BSA \ (m^2) = \left( \frac{\text{Height (cm)} \times \text{Weight (kg)}}{3600} \right)^{1/2}, \ OR
\]

\[
= \sqrt{\frac{\text{height (cm)} \times \text{weight (kg)}}{3600}}
\]

**PROPHYLAXIS**

**PCP prophylaxis in an immune-reconstituted infant and child**

All children with a CD4 % less than 15% or a CD4 count < 200 should receive co-trimoxazole.

It is simplest to give infants under one year aged under 5 kg 120 mg once daily three times a week usually on Monday, Wednesday and Friday.

The dose for average children aged 1-3 is 240 mg, for 4-9 years 480 mg, and for 10 years and over 960 mg.
Discontinue prophylaxis with TMP/SMZ if CD4 >500 cells/µl in children >8 yrs, or >15% in child < 8yrs for over 6 months.

Do not discontinue prophylaxis in an HIV-infected infant <12mts of age irrespective of CD4 % and number.

**Dosage and indications for prophylaxis against mycobacterium avium intracellulare complex (MAC)**

Indications:
CD4 number < 50 cells/µl and age > 6yrs.

CD4 cut off for other ages:
2-6 yrs is 75 cells/µl.
1-2 yrs is < 500 cells/µl
<12 mts is < 750 cells/µl.

Dosages: Azithromycin 5 mg/kg (maximum dose 250 mg) orally once daily, or 20 mg/kg once weekly.

Alternatively Rifabutin 5 mg/kg orally once daily (maximum dose 300 mg).

**IMMUNISATIONS AND PAEDIATRIC HIV INFECTION**

The HIV-infected child should receive ALL the childhood immunizations as soon as is age appropriate [DT(a)P, HiB, Meningitis C, Pneumococcal]. However exceptions apply and include:

1. For indeterminate infants, do not give BCG until the infant has three negative PCR’s.

2. Measles immunization (given as MMR) is recommended at the usual ages for infants/children with asymptomatic HIV infection and those with who are not severely immunocompromised (immunologic category 3 of the CDC classification).

3. Varicella vaccination is safe, effective and immunogenic, and may be given to HIV-infected children with CDC classification N1 or A1.

4. Influenza vaccine should be considered each autumn for all HIV-infected infants, children and adolescents over 6 months of age.

5. Oral Ty21a (typhoid) vaccine is a live-attenuated vaccine and should not be given to HIV-infected individuals; the parenterally administered Vi capsular polysaccharide vaccine is a suitable alternative for children over 2 years of age who are travelling to Africa.
THE PAEDIATRIC HIV CLASSIFICATION

Adapted from:
CDC. 1994 revised classification system for HIV in children less than 13 years of age. MMWR1994;43:RR-12


<table>
<thead>
<tr>
<th>Immunologic Categories</th>
<th>Clinical categories</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N: No signs/symptoms</td>
</tr>
<tr>
<td>1: No evidence of suppression</td>
<td>N1</td>
</tr>
<tr>
<td>2: Evidence of moderate suppression</td>
<td>N2</td>
</tr>
<tr>
<td>3: Severe suppression</td>
<td>N3</td>
</tr>
</tbody>
</table>

Immunologic categories based on age-specific CD4+ T-lymphocyte counts and percent of total lymphocytes

<table>
<thead>
<tr>
<th>Immunologic Category</th>
<th>Age of Child</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;12 mos</td>
</tr>
<tr>
<td></td>
<td>μL (%)</td>
</tr>
<tr>
<td>1: No evidence of suppression</td>
<td>&gt;1,500 (&gt;25)</td>
</tr>
<tr>
<td>2: Evidence of moderate suppression</td>
<td>750-1,499 (15-24)</td>
</tr>
<tr>
<td>3: Severe suppression</td>
<td>&lt;750 (&lt;15)</td>
</tr>
</tbody>
</table>
MANAGEMENT OF CLINICAL ILLNESS

(a) The infant/child with HIV and fever

Children with HIV infection attending clinic are told to come to the hospital if they become unwell. This is usually either because they have a fever, vomiting and diarrhoea, or a chest infection. Please see below for guidelines for the afebrile well child with diarrhoea and vomiting (D & V), and children with obvious chest infections. The following guidelines are for the child with a fever.

Note the stage of the child's illness. The more severely immunosuppressed the infant/child, the more likely minimal signs will be detected with serious pathology. Look in notes for recent letters and CD4 count. A CD4 < 10% or <300 cells/µl in an infant or child under 8 years indicates severe immunosuppression.

Take a good history of the acute illness and examine the child thoroughly. Get past history from notes; parents may be vague or reluctant historians of past illness. If the infant/child has had proven bacterial infection before, a recurrence of that infection may have occurred. Children with HIV have a considerably increased risk of bacterial infections. General principles are to treat earlier for longer courses, in big doses. Particularly look for:

A. ENT infections including sinusitis: obtain throat swabs - viral and bacterial. Treat with augmentin 400 duo (5ml bd for 1-12 yr olds) for 7-14 days. HIV-infected children have high rates of cervical lymphadenitis. If symptoms are mild: treat with oral augmentin. If symptoms are severe, the patient may require I/V antibiotics. Teicoplanin and ceftriaxone are preferred as the child may later be managed at home by community nurses. If the lesion is fluctuant involve the paediatric surgeons or ENT.

B. Skin infections and abscesses. Swab. Treat lesions with augmentin.

C. Oral candida and/or perhaps herpes simplex. Swab. Treat with oral fluconazole (3-4 mg/kg/day) and/or oral acyclovir.

D. UTI - treat with augmentin as above.

E. Sepsis - mainly pneumococcal - symptoms/signs masked in this group. If at all toxic or unwell obtain FBC, blood cultures and admit with I/V ceftiraxone (80 mg/kg/day). Add flucloxacillin if any skin infection/abscess are present. A high neutrophil count may be a useful guide but cannot always be expected

F. If prescribing oral/iv antibiotics administer prophylactic fluconazole to prevent candida (1-2 mg/kg/day OD, just whilst on antibiotics).

G. ABC or NVP hypersensitivity as discussed previously.
(b) The infant/child with advanced HIV and gastroenteritis (GE)

Think of serious bacterial infections eg. UTI, sepsis

**ENDEMIC CAUSES**
The cause of endemic GE during paediatric HIV infection varies with geographic location. In the UK pathogens can be isolated from the stools in around 50% of children admitted with GE without HIV (Table 1). Children with symptomatic HIV infection have all of these infections. Rota & adenovirus are the commonest causes of GE in the UK. Bacterial causes include salmonella, shigella, & campylobacter species, & adherent E.coli.

**GE FOREIGN TRAVEL**
*Malaria* must be looked for in a child recently returned from abroad with a fever and either vomiting or diarrhoea. It is particularly important to consider *typhoid* in a febrile child (2 x blood cultures and start on ceftriaxone). Other diagnoses to consider include amoebiasis, cholera, or helminth infection (if an eosinophilia is present).

**CHILDREN WITH ADVANCED HIV ARE DIFFERENT**
GI Pathogens include cytomegalovirus (CMV colitis), candida, Cryptosporidium arvum, Isopora belli, Microsporidia (especially Enteroctytozoan bieneusi), Giardia lamblia, Cyclospora and Mycobacteria avium complex and Yersinia entercolitica.

**PSEUDOMEMBRANOUS COLITIS (ANTIBIOTIC ASSOCIATED DIARRHOEA)**
Various antibiotics (especially ampicillin, erythromycin and co-trimoxazole) can produce a colonic overgrowth of toxin producing Clostridium difficile, with the local production of multiple plaque-like lesions on the mucosal surface. Mild colitis can rapidly result in toxic megacolon. The diagnosis can be confirmed on identification of C difficile toxin in the stool. Treatment involves replacement of the causative antibiotic with oral metronidazole or vancomycin.

**CLINICAL HISTORY**
Particular attention to: The presence of bilious vomiting, blood or mucous in diarrhoea, reduced urine output, altered level of consciousness, illness in family members, foreign travel, previous GI problems and current medications.

**EXAMINATION**
The most important points are to assess the state of dehydration and to identify other pathology mimicking or accompanying GE.

**DEHYDRATION**
This is a clinical diagnosis. A recent clinic weight and evaluating urine output and osmolarity may be useful in assessing fluid loss. Hypernatraemic dehydration can be very difficult to assess as signs of dehydration may be masked. Children with AIDS and GE lose bicarbonate resulting in accompanying metabolic acidosis, and may need oral/IV sodium bicarbonate supplementation.
NOT ALL CHILDREN WITH D & V HAVE GE
Particular care should be taken of the febrile child who looks toxic, the child with a tender or distended abdomen and the child with bloody diarrhoea. Diagnoses to consider include other infections (UTI, meningitis, pneumonia, sepsis, OM) and surgical conditions.

ART particularly the PIs (nelfinavir) can cause prolonged protracted diarrhoea.

MANAGEMENT - TRY TO RETURN TO A NORMAL DIET QUICKLY
It is critical to minimise the weight loss associated with acute infections. Involve the paediatric HIV dietician early.

ADMISSION CRITERIA
Admit all HIV-infected children with GE with accompanying: fever or if he/she needs IV therapy or in the presence of oliguria or with severe diarrhoea (eg >5 loose stools/day).

INVESTIGATIONS
All children with HIV and diarrhoea should be investigated. If febrile they should have blood cultures and an MSU as well as:

A) 1 fresh stool specimen labelled HIV for microbiology. Microscopy will identify trophozoites, cysts, and spores. Modified Ziehl-Nielsen staining can identify mycobacteria. Stool culture & agglutination is useful for E.coli, salmonella, shigella, and campylobacter species.

AND

B) 1 fresh stool labelled for virology for Electron microscopy (if available) /ELISA/culture.

It is important to write two forms and send 2 separate specimens are sent. Write the form yourself with decreased CD4 as clinical details.

Send 1 stool/day to virology and bacteriology for 3 days if diarrhoea persists.

If the diarrhoea persists, also consider: reducing substances, C.diff toxin, CMV buffy coat, blood cultures for MAC, endoscopy and biopsy.

Jejunal biopsy +/- colonoscopy should be considered in all culture negative persistent diarrhoea unresponsive to medical therapy. Biopsy material should be sent to histopathology (for CMV, fungi, acid fast bacilli [AFB]), microbiology (including M.TB/MAC) and viral culture.

TREATMENT
Rehydrate the patient.
If the child is febrile and toxic then empirically treat with IV ceftriaxone 50-80 mg/kg OD, and await blood culture results.
If the child is well and the diarrhoea persists over 3 days with negative stool cultures then treat with **oral metronidazole 7.5 mg/kg/dose tds**. Give at least one week’s course. If the diarrhoea persists with negative cultures, consider changing to ciprofloxacin. In an older child with chronic diarrhoea and AIDS, use an anti antimotility agent (Loperamide).

**SPECIFIC TREATMENTS**
CMV - Gancyclovir
MAI - Ciprofloxacin, Rifabutin, Clarithromycin
Isospora - Septrin
Microsporidia - Metronidazole, Albendazole
Cryptosporidia - Paromomycin and hyperimmune bovine colostrum

**NUTRITION**
Severe weight loss can occur very quickly so involve the dietician early.
If diarrhoea occurs > 5 days with weight loss, then consider Peptijunior.

Table 1

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>Viruses</th>
<th>Protozoa</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common:</td>
<td>Common:</td>
<td>Common:</td>
</tr>
<tr>
<td>Salmonella sp</td>
<td>Rotavirus</td>
<td><em>Giardia lamblia</em></td>
</tr>
<tr>
<td><em>Campylobacter jejuni</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less common:</td>
<td>Less common:</td>
<td>Less common:</td>
</tr>
<tr>
<td><em>E.Coli</em> (Enteropathogenic,</td>
<td>Adenovirus types 40/41</td>
<td>Cryptosporidium</td>
</tr>
<tr>
<td>Enterotoxic, &amp;</td>
<td>Norovirus or Small</td>
<td><em>Entamoeba histolytica</em></td>
</tr>
<tr>
<td>Enterohaemorrhagic)</td>
<td>Round Structured Virus</td>
<td><em>Isospora belli</em></td>
</tr>
<tr>
<td>Shigella sp.</td>
<td>Caliciviruses</td>
<td>Microsporidia</td>
</tr>
<tr>
<td><em>Yersinia Enterocolitica</em></td>
<td>Norwalk-like</td>
<td></td>
</tr>
<tr>
<td>Aeromonas/</td>
<td>Astroviruses</td>
<td></td>
</tr>
<tr>
<td>Plesiomonas</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(c) The infant/child with HIV and respiratory illness

**QUICK SUMMARY**
Check whether the child has been taking Septrin prophylaxis regularly. Check oxygen saturation, inquire about the rapidity of onset of respiratory symptoms. Does the infant/child look toxic and acutely unwell? Check for a neutrophilia.

If the infant/child does not appear acutely unwell, prescribe oral augmentin for 7 days. If the infant/child is acutely unwell, admit and treat with IV ceftriaxone + oral Azithromycin.
With a slower onset of symptoms and hypoxia in room air, add IV septrin (TMP-SMZ) to IV ceftriaxone and oral azithromycin.
THE AETIOLOGY OF RESPIRATORY DISEASE IN AN HIV INFECTED INFANT/CHILD

Many different pathological processes may lead to pulmonary disease, including pneumonia due to bacterial, viral, or opportunistic infections, lymphocytic interstitial pneumonitis (LIP) complex, and rarer non-infectious causes (table 1).

PNEUMOCYSTIS CARINII PNEUMONIA (PCP)

PCP is one of the commonest AIDS-defining diagnoses, occurring in about 40-50% of children reported. PCP is more common in children <1 year (72%) compared to those aged over 1 (38%). A recent UK study of vertically-infected children documented a median age at PCP diagnosis of 4.1 (1.4-27.3) months.

Clinical
The clinical features of PCP are tachypnoea, dyspnoea, cough and fever. The onset may be insidious over 1-2 weeks with slowly increasing tachypnoea. Coughing is not usually prominent until the full clinical picture develops with severe dyspnoea.

Physical findings are usually limited to fine crepitations. Fever is often low grade. Hypoxia is common, with oxygen saturations in room air between 80-90. A rapidly progressive course of disease leading to respiratory failure within days is commonly described.

The CXR may be normal or hyperinflated early in the disease, but there is usually rapid development of complete opacification with air bronchograms. The aoealar infiltrates progress peripherally with late apical sparing and small pleural effusions. Associated bullae, cysts and pneumothoraxes may be reported.

Diagnosis
In infants the diagnosis can sometimes be obtained from a naso-pharyngeal aspirate (NPA), but bronchoscopy with bronchoalveolar lavage (BAL) is now the optimum method for diagnosing PCP in children. At St. George's hospital all bronchoscopies are performed in the Paediatric Intensive Care Unit (PICU). BAL can be performed using an 8F nasogastric feeding tube in intubated children who may not tolerate bronchoscopy.

If BAL cannot be performed straight away, then start treatment pre-BAL. Positive results can be obtained up to 48 hours after starting treatment doses of septrin.

Microbiology must be informed prior to the BAL/NPA. The specimen is processed using a fluorescent antibody technique. As PCP is an AIDS defining diagnosis with an especially poor outlook, it is very important to make a definitive diagnosis even after commencing treatment.

Treatment
The recommended initial treatment of PCP is Trimethoprim 20 mg/kg/day and sulphamethoxazole 100 mg/kg/day, in 4 divided doses infused over 1 hour for 3 weeks. This can be given orally in the same doses for the third week if the child is off the ventilator and absorbing feeds.
It is not unusual for the child to clinically deteriorate for a few days after commencing therapy, and then significantly improve by one week. Beware of the slow onset of pneumothorax, and maintain a low threshold for repeat CXR if there is clinical deterioration.

Side effects from high-dose TMP-SMZ are not frequent, an erythematous rash being the commonest which responds to temporarily stopping the drug. An urticarial rash or signs of Stevens-Johnson syndrome, require immediate discontinuation of the drug indefinitely. GI disturbance & bone marrow suppression also occur.

If there is failure to respond to TMP-SMZ or an allergic reaction to the drug, treatment should be changed to slow I/V Pentamidine Isothionate (once daily, 4 mg/kg/day) for 3 weeks. Side effects include neutropenia, thrombocytopenia, hepatitis, renal impairment & early hypoglycaemia with late development of insulin dependent diabetes.

Adult studies have demonstrated a reduced morbidity in PCP with early use of corticosteroids. Although data are lacking in children, methyl prednisolone should be added when a diagnosis of PCP has been made. The recommended regime for I/V methyl prednisolone is 0.5 mg/kg/dose, four times a day for 7 days, twice daily for 7 days and once daily for 7 days. This can be changed to oral prednisolone in the same doses ( ie 2 mg/kg od for a week, then 1 mg/kg for a week, then 0.5 mg/kg for a week) when oral feeding starts.

Nutritional support is extremely important and a goal of 150-200 Cal/kg/day should be aimed for.

Failure to respond to TMP-SMZ alone should raise the possibility of a second treatable infection and repeat BAL or lung biopsy should be considered. CMV is frequently identified at BAL in PCP infection, but Ganciclovir should only be used in a child with PCP and CMV if the child is not responding to PCP therapy.

**LYMPHOCYTIC INTERSTITIAL PNEUMONITIS (LIP)**

The diagnosis of LIP is most frequently made on a routine CXR in an asymptomatic child in the 2nd year of life. Between 30-50% of vertically infected children develop LIP.

**Clinical**

The onset of LIP is usually slow, characterised by a chronic cough in children, often with evidence of generalised lymphadenopathy, bilateral parotid enlargement and hepatosplenomegaly. Many children are asymptomatic initially and the diagnosis is most frequently made on a routine CXR, or a CXR taken when the child presents with an acute lower respiratory tract infection (ALRTI). Having LIP leads to an increased frequency of ALRTI. *Streptococcus pneumoniae, Haemophilus influenzae* and *Staphylococcus aureus* are the commonest organisms.
Although a definitive diagnosis of LIP requires a lung biopsy, the presence of widespread reticulonodular shadowing (1-5 mm diameter) in a well child, with or without hilar lymphadenopathy, persisting on a CXR for greater than 2 months, which does not respond to antibiotics can be considered as presumptive evidence for LIP. It may not be possible to diagnose LIP with certainty by CXR alone. CMV pneumonitis, fungal infections, PCP and, in particular, tuberculosis should all be considered.

Management
Children presenting with ALRTI who are known to have LIP should have an FBC, CRP, blood cultures and if possible a sputum taken. Check for compliance with septrin prophylaxis. Initial treatment should be with IV ceftriaxone 80 mg/kg/day until there is a clinical response. If the child does not improve quickly (24 hours) add Azithromycin (10 mg/kg/OD for 5 days). If there is hypoxia or no clinical improvement in 48 hours then a bronchoscopy should be organised. The antibiotic course should be at least 10 days, as relapses, especially with pneumococcal infection are common. Augmentin, or a 5-day course of Azithromycin, is a good option if there are concerns about compliance. Always use the top dose for age.

**BACTERIAL INFECTIONS (NON-MYCOBACTERIAL)**
Serious bacterial infections are very common. The commonest clinically diagnosed infection is acute pneumonia and primary septicaemia. The frequency of bacterial infection increases with HIV disease progression. The increased risk of bacterial pneumonia for children with LIP has previously been discussed. Generally use intravenous antibiotics earlier, and give longer total treatment regimes (eg 10-14 days).

The commonest organisms identified in all the studies are encapsulated organisms, *Streptococcus pneumoniae* and *Haemophilus influenzae*. *Staphylococcus aureus* and gram negative infections, especially *Pseudomonas aeruginosa*, are seen more commonly in children with AIDS.

**Clinical**
The clinical presentation of acute bacterial pneumonia in children with early HIV infection is similar to non-infected children. The clinical signs may be less obvious in children with AIDS. It is always important to obtain blood cultures.

The importance of upper respiratory tract infections in children with HIV infection must be recognised. Ear infections and throat infections are very common. Sinusitis should be particularly sought for with clinical signs or sinus x-rays.

**Management** - See LIP section.
A child with clinical evidence of an ALRTI (fever, cough, raised respiratory rate, or chest signs, or new changes on the CXR) should be treated promptly, empirically with a broad spectrum of antibiotics (oral augmentin or IV ceftriaxone). The WCC can be useful, however, many children with serious bacterial infections do not have significantly raised neutrophil counts. The WCC should not therefore be relied upon, particularly in patients with advanced disease. The choice of oral or intravenous antibiotic depends on the child's clinical condition. As above add azithromycin and consider BAL early.
MYCOBACTERIAL DISEASE- MYCOBACTERIUM TUBERCULOSIS

Clinical
The diagnosis of tuberculosis disease in children remains difficult whether or not they are infected with HIV. The incubation period in children can be up to 6 months, and many children remain asymptomatic. Fever, weight loss and night sweats are unusual and symptoms range from a persistent cough to apathy and lethargy in disseminated disease. CXR signs include hilar lymphadenopathy, segmented or lobar disease, atelectasis, effusions or miliary shadowing. Extrapulmonary disease is rare.

Mantoux test interpretation in the HIV infected child is complex. Responses to skin testing are often impaired as a result of defective cell mediated immunity. Factors that need to be taken into account in interpreting a positive reaction are the stage of HIV disease, infectious contact, country of origin and age. If the child has had BCG as an infant, a reactive mantoux >10mm by 3-5 years may indicate possible infection with tuberculosis requiring further investigation. There is a paucity of studies describing the effect of HIV on Mantoux testing in children.

Culture is still the gold standard for TB diagnosis, allowing drug sensitivity patterns to be established but culture positive rates of only 30-50% are usual. Gastric lavage is more sensitive than broncho-alveolar lavage, and every attempt should be made to try and obtain 3 early morning gastric lavages if pulmonary TB is suspected.

Management
There are no controlled trials of tuberculosis treatment in HIV infected children. Current practice is to extend rifampicin and isoniazid to 9-12 months depending on clinical response and degree of immunosuppression. Give pyrazinamide and ethambutol for first 3 months only. In the absence of disease, chemoprophylaxis with isoniazid for 6/12 is indicated.

NON-TUBERCULOUS MYCOBACTERIAL DISEASE
Disseminated non-tuberculous mycobacterial disease (DNTM), is associated with severe immunosuppression and a CD4 count <50/mm3. 90% of cases are due to Mycobacterium avium intracellulare complex (MAC). Median survival of children from diagnosis is 6 months. The clinical features usually include prolonged fever, bone marrow suppression, weight loss and chronic GI symptoms. In patients with DNTM, MAC may be isolated from the lungs. Radiological presentation can occur with enlarged hilar lymph nodes. Treatment involves a complex multi-drug regime of ciprofloxacin, rifabutin & clarithromycin. If the clinical presentation is suspected then blood cultures for MAC should be taken using special radioactive bottles available from microbiology.
**CYTOMEGALOVIRUS**
Disseminated CMV infection is an important AIDS defining diagnosis in HIV-infected children. CMV can be asymptomatically shed in secretions and be cultured on BAL. Patients with disseminated CMV disease are viraemic and often have radiological evidence of CMV pneumonia, a rash and hepatitis. CMV viraemia should be sought for by sending CMV buffy coat to virology (also called DEAFF test) [1-2 ml in a Green Heparin bottle]. An early result is available after 48 hours. Patients with proven disseminated disease are treated with Ganciclovir (a nucleoside analogue). A 2-week induction phase of 5mg/kg every 12 hours intravenously, is followed by a prolonged maintenance phase of 5 mg/kg/day given 3-7 days/week.

**OTHER VIRAL INFECTIONS**
Respiratory syncitial virus (RSV) bronchiolitis may be complicated by *Pseudomonas aeruginosa* despite negative BAL’s. Ribavirin and ceftriaxone should be given to all infants known to be HIV positive who develop RSV bronchiolitis. Prolonged shedding of RSV is common.

**Table 1: Reported Causes of Respiratory Disease in Paediatric HIV Infection**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumocystis Carinii Pneumonia</td>
<td></td>
</tr>
<tr>
<td>Lymphocytic Interstitial Pneumonitis Complex</td>
<td></td>
</tr>
<tr>
<td>Non-tuberculous bacterial pneumonia: <em>Streptococcus pneumoniae</em>, <em>Haemophilus influenzae</em>, <em>Staphylococcus aureus</em>, <em>Pseudomonas aeruginosa</em>,</td>
<td></td>
</tr>
<tr>
<td>Atypical pneumonias: <em>Mycoplasma pneumoniae</em>, <em>Chlamydia pneumoniae</em></td>
<td></td>
</tr>
<tr>
<td>Mycobacteria: <em>Mycobacterium tuberculosis</em> and MAC</td>
<td></td>
</tr>
<tr>
<td>Viral Pneumonias: CMV, Measles, Adenovirus, Parainfluenzae, Influenzae, RSV, Varicella, HSV.</td>
<td></td>
</tr>
<tr>
<td>Fungal Infections: Aspergillosis, Histoplasmosis, Candida, Norcardia - usually asymptomatic or acute respiratory illness with cough, malaise, fever. CXR: small opacities or miliary mottling. Serology unreliable in immunocompromised. Need sputum to diagnose.</td>
<td></td>
</tr>
<tr>
<td>Congestive cardiac failure - cardiomyopathy/cor pulmonale</td>
<td></td>
</tr>
<tr>
<td>Malignancy - Kaposi’s Sarcoma, Leiomyoma, Leiomyosarcoma’s &amp; Lymphoma’s</td>
<td></td>
</tr>
</tbody>
</table>
GUIDELINES FOR HIV TESTING OF CHILDREN AND YOUNG PEOPLE

Introduction

TRANSMISSION OF HIV
The majority of children who have HIV acquire the virus from their mothers either in utero, at delivery or through breastfeeding (vertical transmission).

Children and young people may also acquire HIV horizontally:
- Through unprotected sexual intercourse with an infected person or through sexual abuse.
- Through sharing contaminated needles, syringes or other equipment during intravenous drug use.
- Following administration of infected blood or blood products or organ transplantation (rare in UK since screening of blood and organs).

Background to consent

Informed consent is required before doing an HIV test and this should be given voluntarily. Some of the issues around consent and children and young people are addressed below.

Young People over 16 years
Young people aged 16 or 17 years can consent to their own medical treatment (Family Reform Act 1969) but unlike adults, the refusal of treatment can be overridden by a person with parental responsibility or the court. This would be done on the basis that the welfare of the young person is paramount and only in exceptional circumstances and would require legal advice. If a young person of 16 or 17 gives consent, it is not necessary to get consent from a person with parental responsibility but it would be good practice to involve them unless the young person does not want this (DoH 2001).

Children and Young People < 16 years
A child or young person under 16 years old can give consent to treatment if they are "Gillick competent" now referred to as Fraser guidelines. This means that a child who has sufficient understanding to enable them to understand fully the nature and implications of having an HIV test will also have the capacity to give their own consent.

Young Person or Child without Capacity
For young children or those who lack the capacity to give informed consent, consent can be given by a person with parental responsibility or by the court. As most children with HIV will have acquired their illness from their mother, the involvement and consent of the mother should ideally be sought but it is good practice to involve both parents where possible. The Children Act (1989) promotes the participation of children in giving consent to procedures and their involvement in discussions on testing should be encouraged where appropriate.
For children looked after by social services, parental responsibility may be shared between the parents and local authority. Many local authorities have developed their own policies around testing of children and these should be referred to. They often involve consent having to be given by the Director or Assistant Director of social services and this should be obtained in writing. It is still important to consider the implications for the birth parents, particularly the mother as a positive test may mean that she also has HIV and it is important to consider how this will be handled. If a child is a ward of court only the court can make major decisions affecting the child.

When adults or organisations make decisions that affect children they must always think first about what would be best for the child (Article 3, UN Convention on the Rights of the Child 1989). [Routine HIV testing where children are being fostered or adopted is not appropriate and testing should only be done on the grounds of the child's health needs (2002 draft document DOH)]

For further guidance refer to the Department of Health Reference Guide to Consent for Examination or Treatment (2001) and St George's Hospital Policy on Obtaining Valid Consent for Treatment (2001) and the DOH draft document on Children in Need and Blood-borne Viruses (2002).

**Testing a Child for HIV**

Any Paediatrician should be able to pre and post test counsel a family for HIV and test the child. We would suggest you do this in liaison with the local Genito-urinary medicine health advisors. Further advice can be obtained from one of the Paediatric ID consultants or nurses (contact details are at the front of the manual).

As most children with HIV have acquired the virus from their mothers, testing a child has huge implications for the family, as a positive test is likely to mean the mother is positive and her partner and other children may also be infected. The pre-test discussion should ideally involve both parents and where appropriate the child or young person. Approximately 90% of the children with HIV in the UK are of Black African origin so it is important that the information given is culturally appropriate and interpreters are used where necessary.

**HIV testing is never done as an emergency and the family must be allowed time to consider the implications.** Surrogate testing by doing CD4 count should not be done. During the pre-test discussion, the reasons for wanting to test for HIV should be explained. The person's understanding of HIV and its transmission, should be checked and built upon, discussing the disease pattern and treatments. Advantages and disadvantages of having the test should be explored. Carers and the young person where appropriate, should be encouraged to discuss their concerns or potential difficulties of having a positive result. They should be helped to look at whom they would wish to tell, current social support and previous coping strategies. An appointment should be made for giving the results and contact numbers given to the family in case they would like further support or information whilst waiting for the result.
There may be occasions when the wishes of the carer are in conflict with those of the child or young person. If a parent refuses a test it is important to try and keep an open dialogue and to continue to work in partnership with them. It is necessary to differentiate between a sick child where doctors are concerned the child may have HIV and could benefit from treatment and older children who appear healthy. Cases where there may be conflict of interests such as refusal to test, or breast feeding a baby, will be discussed within the specialist multidisciplinary HIV paediatric team to decide if further action or legal advice is necessary. Verbal consent to the test is adequate but should be recorded in either the nursing or medical notes.

Confidentiality

During the pre-test and post-test discussion it should be explained that information about the family and HIV will remain confidential within the HIV team and that their consent will be obtained before this information is disclosed to others. This would only be breached in exceptional circumstances and where there is a real risk of serious harm to the patient or another individual (GMC Guidelines 1995). The Human Rights Act 1998, Article 8, the Right to Respect for Private and Family Life also supports the patient’s right to protect their confidentiality. Fear of transmission to others, for example at school, does not constitute a valid reason for disclosure.

Testing

For a child over 18 months old an HIV antibody test (clotted specimen) is adequate. If the child has recently been exposed to HIV, further testing at least 3 months after the exposure will be necessary.

In babies, testing for HIV is more complex. A baby born to a mother who has HIV will have maternal antibodies, which may still be in their blood until 18 months of age. The test required is a DNA Polymerase Chain Reaction (EDTA specimen) and this should be sent to the Health Protection Agency in Colindale. The testing of babies born to positive mothers is done at 0-2 days old, 6 weeks and 3 months old. An HIV antibody test will be done at 18 months of age. These babies would normally receive zidovudine syrup for the first 4 weeks and be attending the Family Clinic for follow up.

Giving the Results

The person who had the pre-test discussion with the family should, wherever possible, give the result. If the result is positive give the information in a clear and sympathetic way. Allow time for the young person or carers to react to the news. Listen to their response and help them talk through what it means and how they feel. Provide information on HIV but be aware that it may be difficult for anyone to take in this information whilst upset. It is important to identify who will support the family and help them to think about whom else they would like to know. Some level of anxiety or distress is an appropriate response a positive test result. The nurse should convey hopefulness without giving false reassurance. Contact numbers should always be given and a follow up appointment will be made in the Family Clinic. Parents are referred to the adult HIV team for their testing.