

Clinical Handbook

for Global Health Electives

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Introduction

“What I’ve experienced is that I can’t know the future. I can’t know if anything that I do will change what happens tomorrow. I can’t know with certainty, but what I do know is if I do nothing, nothing will change.”

–Dr. James Orbinski

Much has been written about Global Health since the first edition of this International Elective Student’s Handbook: Developing World Medicine was first published in 2006. While much has changed in the world, sadly, many things have remained the same. The first edition of this book looked at the top five diseases in terms of their mortality: malaria, HIV, TB, diarrhoeal diseases and acute respiratory infections. Many of these diseases have been all but eradicated from the developed world, yet still remain leading causes of death in low and middle income countries (LMIC).

Top Ten Causes of Death Globally (2010)⁷

1. Ischaemic heart disease
2. Stroke
3. COPD
4. Lower respiratory infections
5. Lung Cancer
6. HIV/AIDS
7. Diarrhea
8. Road Injuries
9. Diabetes
10. Tuberculosis

Today, low-income countries share many of the same burden of disease as in high income countries. Ischaemic heart disease is a leading cause of death, followed closely by stroke and COPD. Road injuries are an all too common cause of morbidity and mortality in low-income countries, and are the leading cause of death for travellers.

The disparities in health between high and low income countries become apparent when examining the list of top ten causes of death more closely. Four infectious diseases remain: lower respiratory infections, HIV/AIDS, diarrheal diseases and tuberculosis. Malaria for the first time fell out of the top ten, ranking eleventh. These conditions still exist in the isolated pockets of poverty in high income countries amongst our homeless and street involved, immigrant and refugee, indigenous populations and isolated communities of the North. More must be done at home and abroad to combat these conditions by addressing social determinants of health and ensuring access to essential medical care.

How This Handbook Will Help You

The purpose of this guide is to provide the elective student with some basic competencies for their international elective. As a young medical student or resident, your overwhelming desire may be to help your patients, but this may be more difficult than you anticipate secondary to lack of knowledge, skill or ability. Medical students and residents often bring boundless enthusiasm and an earnest desire to be of service while on their electives, hoping to give more than they will receive. These international elective experiences can be a powerful educator, opening your eyes to the day to day realities of life in the developing world.¹ The end result is frequently being trapped in a frustrating limbo of wanting to help, but not knowing where to start.

This handbook is aimed at students that are going to regions classically defined as “developing countries,” where hospitals are dominated by five broad diagnoses: Malaria, HIV Infections, Tuberculosis, Diarrheal Diseases and Acute Respiratory Infections.

Using the tutorial-favourite “DEEPICT” formula of understanding diseases, this brief guide will give you the most important pieces of information you’ll need to navigate aspects of treatment of your patients. Covering the vitals is the goal here, so while you won’t find detailed pathophysiology, you will find practical management principles you can use in the field.

Introduction cont'd

What is the Goal?

While our goal in creating this handbook is to offer materials to prepare you for your elective abroad, it must be emphasized how important the learning from our international colleagues is. Embark on your international health work with humility and a willingness to learn. It is a privilege see medicine practiced without the aid of CT scanners, delivered in makeshift classrooms-turned-clinics, and conceptualised in an environment where salaries are low, mortality rates are high, and the struggle to survive is formidable.

Good luck, and hope you find this helpful.

Safe travels. Always ask questions!

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Personal Protective Equipment

Part of ensuring your own personal safety is ensuring that you have the necessary equipment and supplies to perform your job safely. Below is a table of some recommendations for supplies for an international surgical elective, it could be adapted for other international electives.^{3,4}

Table 1-1: Examples of items to bring on an international elective

Personal items	Unblocked cell phone with flashlight Small high-intensity headlight (from a outdoor goods store) Diary/log Toiletries and appropriate clothing
Personal medical equipment	Scrubs (3–5 pairs) Boots Eye protection Masks Gloves Personal emergency medical kit DEET ointment or spray for malarial area and long sleeve shirts and long pants Sterile needles/syringes (in case of need for self)

Consider protection against arthropods such as mosquitoes, ticks, fleas and other insects which can transmit illness. Some vector borne illnesses are preventable through vaccine or chemoprophylaxis, while others are not. It is advisable to wear DEET containing insect repellents and long sleeved clothing, as well as use bed netting to help decrease arthropod exposure. Anopheles mosquitoes which transmit malaria are most active during dawn and dusk hours, while Aedes mosquitoes which transmit dengue and yellow fever are most active during the day. Thus, frequent re-application of repellent is ideal.

Travel Health

Prior to seeing a your physician or a travel health doctor we recommend looking at the PHAC (<http://www.phac-aspc.gc.ca>) or CDC (<http://wwwnc.cdc.gov/travel/>) travel health websites for information regarding disease burden in the location of your potential elective. By better informing yourself prior to your travel medicine consultation you will be better able to ask relevant questions. Be sure to discuss immunizations with your travel health provider. You can think of them broadly as: routine, required and recommended. Another consideration in many tropical locations is malarial chemoprophylaxis, taking into account local resistance patterns.

Introduction cont'd

Pre-Departure Training²

Pre departure training (PDT) is critically important to ensure a safe and meaningful global health elective. We strongly recommend discussion/reflection as part of PDT and encourage students to seek PDT at their own university/school. Often faculty/peer mentorship can also play an important preparation for a global health elective. Some questions to consider as you begin preparing for your elective are listed below:

- Where will you be on elective?
- What countries will you be traveling through?
- What languages are spoken?
- Will you take a vacation during your elective period?
- Will you be travelling alone or with other learners?
- What environmental concerns may impact on your trip (floods, drought, fires etc)?

If you are female:

- Are you pregnant?
- Are you planning on becoming pregnant in the next 3 months?
- Are you breastfeeding?

Travel Safety Tips

1. Look out for your own health and safety first.
2. Don't play with animals.
3. Use appropriate personal protective equipment.
4. Keep paper copies of important documents such as passport and medical insurance and/or email them to yourself.
5. Be aware of your surroundings.
6. Avoid large gatherings such as mass rallies or political protests
7. Ensure you have contact numbers for the Canadian Embassy or Consulate in the country you are in - Registration of Canadians Abroad (ROCA)⁶.
8. Learn the basic laws, customs and social norms of where you will be.
9. Check Travel Advisories from the Department of Foreign Affairs and International Trade and Public Health Agency of Canada: <http://travel.gc.ca/> and <http://www.phac-aspc.gc.ca>
10. Be respectful.

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Malaria

Pamela Kapend

General Information

The malaria parasite (Plasmodium) affects approximately 106 countries worldwide. According to the WHO, an estimated 3.3 billion people live in malaria-endemic regions. WHO approximated that in 2010 there were 216 million clinical episodes and 655,000 deaths attributed to malaria. The burden of malaria continues to weigh heavily on affected communities around the world and most importantly in sub-Saharan Africa where more than 80% of malaria-related deaths occur. Over 40% of these malarial deaths occur in the Democratic Republic of Congo and Nigeria. With an increase in preventative methods and treatment, the malaria mortality has dropped by approximately 25% globally.

Malaria Control

Several interventions for malaria controls must be considered and implemented in order to further reduce burden of disease.

- The MDGs (Millennium Development Goals) have defined malaria control as a reduction of 75% by year 2015
- Some control strategies include:
 - Improving diagnostic tools
 - Mosquito control
 - Improving personal protection
 - Improving disease tracking and parasite surveillance
 - Effective use of anti-malaria medications

Disease Transmission

Anopheles mosquitoes are prime carriers and host for the Plasmodium parasite.

Although both humans and Anopheles mosquitoes are known hosts for the parasite, only humans are afflicted by the disease.

For 10-18 days, the parasite-infested mosquito facilitates the growth and multiplication of the malaria pathogen. The mosquito then acts as a vector, spreading the disease. Once a human host is inoculated, the parasite infects liver cells where it matures, proliferates, and eventually releases toxins and Plasmodium parasites into the bloodstream.

The “blood stage” occurs when the plasmodium parasite infects individual red blood cells producing the clinical manifestations of malaria.

Malaria can also be transmitted from human to human hematogenously (e.g. blood transfusion, organ transplant, and contaminated needles).

Traveller’s Responsibility

Be informed

- It is imperative for travelers to gather information about the prevalence of malaria in the region they are entering. Individualized preventative methods can occur with an accurate knowledge of disease risk and the presence of drug resistant strains in a specific region.

Know your risk

- There are no risk-assessment tools nor quantifying methods hence the experience and judgment of a travel or tropical specialist is essential
 - Note that higher-risk travelers, like first- or second-generation immigrants returning to their countries of origin to visit friends and relatives (VFRs), should be particularly cautioned. VFR travelers tend to consider themselves at low risk or immune to the disease because they either grew up or frequently visited the malaria-endemic region.

Malaria cont'd

- Each individual must take into account their risk assessment including:
 - The destination country: the type of preventative measure depends on the potential risk of disease in a specific region.
 - Detailed itinerary: since malaria transmission isn't homogeneously spread, risk for each individual city/town/region must be considered.
 - The style and duration of travel: consider the type of activities and accommodations as well as duration of travel into each individual's risk assessment.
 - The season of travel: transmission intensity may vary based on the seasonal change.
 - The person's health state, e.g. pregnancy

Know your resources

- Be sure to visit the WHO, CDC or travelers.gc websites which provide accurate resources for any traveller.
- Traveller clinics also provide comprehensive assessments of each traveller's risk.

Classification

Uncomplicated: This is when the malaria infection is not life threatening and is easily treatable.

Complicated/severe: This is based on clinical presentation.

Patients presenting with any of the following clinical featured are considered to have disease:

- impaired consciousness/coma
- severe normocytic anemia [Hb < 70]
- renal failure
- acute respiratory distress syndrome
- hypotension
- disseminated intravascular coagulation (DIC)
- spontaneous bleeding
- acidosis
- hemoglobinuria
- jaundice
- repeated generalized convulsions
- parasitemia of >5%

Table 2-1: Malaria species and specifics

	Plasmodium vivax	Plasmodium malaria	Plasmodium falciparum	Plasmodium Ovale	Plasmodium Knowlesi
Prevalence	• most common in western pacific and the Americas	• Uncommon	• Ver common • Predominant in Africa, New guinea, and Hispaniola (DR and Haiti)	• Least common	• Rare however recent increase in incidence in Southeast Asia
Burden of disease	• Second largest	• Small	• Largest	• Small	• Small but increasing
Signs and symptoms	• Fever, malaise, weakness, gastrointestinal complaints (nausea, vomiting, diarrhea), neurologic complaints (dizziness, confusion, disorientation, coma), headache, back, pain, myalgia, chills, and/or cough				
Diagnosis	<ul style="list-style-type: none"> • Because malaria can present with non-specific signs and symptoms, it is imperative to obtain a complete history (including a travel history) in order to permit a rapid and accurate diagnosis • The diagnosis of malaria should also be considered in any patient with fever of unkwon origin and that is regardless of their travel history • Malaria is diagnosed via microscopic examination of thick and thin blood smear <ul style="list-style-type: none"> • Thick blood smear is more sensitive but more difficult to read • Thin smears help in parasite speciation • A negative smear makes the diagnosis of malaria unlikely <ul style="list-style-type: none"> • But some patients may be symptomatic with very low and undetectable initial smears of the parasite • In this case, 3 sets of bleed smears must be collected 12-24 hours apart • other tests: <ul style="list-style-type: none"> • Rapid diagnosis test (RDTs): antigen detection tests that can rapidly identify an infected patient however cannot confirm speciation or the parasitemia • Polymerase chain reaction (PCR): detects the parasite necleic acid. It's more sensitive and specific than microscopy <ul style="list-style-type: none"> • However this test can only be performed in reference laboratories and is hence reserved for specific reasons • Once the diagnosis is made, calculate the parasite density, which allows deciphering the degree of parasitemia 				

Malaria cont'd

Speciation

There are 5 main types of the Plasmodium species: *P. falciparum*, *P. knowlesi*, *P. vivax*, *P. ovale* and *P. malariae*.

- *P. falciparum* and *P. knowlesi* infections can cause severe disease or death.
- *P. vivax*, *P. ovale*, or *P. malariae* usually cause milder manifestations.
- The *P. vivax* and *P. ovale* species possess a dormant form known as hypnozoite which can remain in the liver and cause relapsing infections from months to several years after inoculation.

Precautions and Treatment

Personal protective measures:

- Are found to be efficacious in low-risk travelers and in combination with pharmacotherapy.
- Each traveller should be instructed to use insect repellent, wear long sleeves and long pants (especially at night), sleep in a mosquito-free setting or use an insecticide-treated bed-net.

Antimalarial drugs:

- Drug prophylaxis is recommended in highly endemic regions and or high-risk patients
- atovaquone/proguanil (Malarone), chloroquine, doxycycline, mefloquine and primaquine can all be used as prophylaxis.
- Medication selection will vary with each patient profile and preference as well as cost and drug availability.
- Important to note:
 - Malarone can be started 1–2 days before traveling.
 - Chloroquine is safe in pregnancy and is taken weekly but beware of increasing resistance.
 - Mefloquine is also taken weekly but is contraindicated in patients with seizure disorder or a history of psychosis.
 - Primaquine cannot be used in G6PD deficiency.

Treatment:

Treatment of malaria depends on:

- Disease severity,
- The species causing the infection,
- The region in which the infection was acquired and its drug-resistance status,
- Drug allergies or drug Interactions with other medications taken by the patient, and
- Patient's health status including pregnancy, chronic illnesses and comorbid conditions.

Uncomplicated Malaria

A) For Chloroquine-sensitive *P. falciparum* or Species Not Identified (I.e areas like Central America west of the Panama Canal, Haiti, the Dominican Republic, and most of the Middle East).

- Treat with PO chloroquine
- Or PO hydroxychloroquine

B) For Chloroquine-resistant *P. falciparum* or Species Not Identified

Four treatments are available:

- Atovaquone-proguanil (Malarone) or Artemether-lumefantrine (Coartem) which are safe for non-pregnant adults and children.
- Or a combination of Quinine sulfate plus doxycycline, tetracycline, or clindamycin can be used.
- Or Mefloquine

Note: This drug is associated with rare but severe neuropsychiatric reactions when used at treatment doses hence should be kept as last option.

Malaria cont'd

C) *P. malariae*, *P. knowlesi*, *P. vivax* and *P. ovale*

- Treat with PO chloroquine (or hydroxychloroquine).
- There is no widespread evidence of chloroquine resistance for *P. malariae* and *P. knowlesi*.
 - Patients with *P. vivax* infections from Papua New Guinea or Indonesia are resistant to chloroquine.
- They are treated with Quinine sulfate plus doxycycline or tetracycline, or, atovaquone-proguanil, or mefloquine.
- Treat the hypnozoites (caused by *P. vivax* and *ovale*) with Primaquine for 14 days.
Note: Patients must be screened for G6PD deficiency before starting this medication.

D) Treatment of Severe Malaria

- Oral antimalarial drugs are not recommended for the initial treatment of severe malaria.
- Patient should receive parenteral treatment with quinidine gluconate.
 - This drug should be administered in an acute care setting (e.g. ICU).
 - Obtain baseline EKG as the drug is cardiotoxic.
 - Glucose must be monitored closely to avoid quinidine-related hyperinsulinemic hypoglycemia.
 - Some adverse effects include ventricular arrhythmia, hypotension, hypoglycemia, and prolongation of the QTc interval.
- Once the patient's parasite density is < 1%, start oral treatment.
 - May use oral quinine at the same dose as for uncomplicated malaria treatment.
 - Quinidine/quinine therapy should be combined with doxycycline, tetracycline, or clindamycin.
 - Or atovaquone-proguanil or artemether-lumefantrine may be used instead of an oral quinine-based regimen.

E) In Pregnancy:

- Pregnancy is associated with decreased immune response hence a decreased ability to clear malaria infections.
- Pregnant women are 3 times more likely to develop severe illness than their non-pregnant counterparts.
- Malaria infection in pregnancy is associated with high mortality and morbidity and can lead to premature delivery, congenital infection, miscarriage, low birth weight and perinatal death.
- For uncomplicated malaria caused by *P. malariae*, *P. vivax* and *P. ovale* or chloroquine-sensitive *P. falciparum*:
 - Treat with chloroquine.
- For uncomplicated malaria caused by chloroquine-resistant *P. falciparum*:
 - Treat with mefloquine or a combination of quinine sulfate plus clindamycin.

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HIV Infections

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General Information

The human immunodeficiency virus (HIV) is a single-stranded RNA retrovirus. The virus' main target is CD4+ T-cells (also known as helper T-cells), causing severe immune dysregulation. There are two different types of HIV: HIV-1 and HIV-2. Of the two, HIV-1 is predominant. There are also many subtypes and circulating recombinant forms. Greater than 75% of HIV in developed countries is HIV-1, group M, subtype B; however, there is much more diversity in developing countries compared to Western countries. The highest degree of viral subtype diversity exists in Central and Western Africa.¹

In 2012 there were 34.0 million people worldwide living with HIV and 2.5 million new infections each year. There are 1.7 million deaths every year due to AIDS.² In North America, approximately 1 out of every 4 persons infected with HIV is unaware they are HIV positive. The rate of undiagnosed HIV may be higher in resource-limited settings with reduced HIV testing efforts.

Table 3-1: Transmission

	Transmission risk/event (%)	Risk reduction techniques
Female-to-male	0.4 (vaginal)*	barriers (i.e. condoms), circumcision
Male-to-female	0.3 (vaginal)*	barriers, tenofovir vaginal gel
Men who have sex with men	1.4 (anal)*	barriers
Mother-to-child	25-35*	C-section, neonatal ART, avoidance of artificial rupture of membranes & instrumentation
Injection drug use	>80 (shared needles)	needle exchange programs, 'safe injection sites'
Iatrogenic	variable	routine PCR testing of human tissue products, aseptic technique

*Antiretroviral therapy (ART) and suppression of viral replication significantly reduces the risk of transmission by >90%

Mother-to-child transmission

- Approximately 15% of new HIV infections are in children.
- Identification of HIV-infected mothers and engagement in HIV care reduces the risk of transmission to <2%.
- Routine opt-out screening for all pregnant women is now the standard of care in most areas. It is best practice to test for HIV as early into the pregnancy as possible.
- Breastfeeding reduces neonatal mortality in resource-limited settings, despite risk of HIV-transmission.
 - Breastfeeding should be encouraged, in conjunction with ART.
- Efavirenz is the only antiretroviral absolutely contraindicated in pregnancy.
- Historic WHO perinatal treatment guidelines for pregnant women in resource-limited settings:
 - a) AZT from 28 weeks of pregnancy or as soon as possible thereafter.
 - b) AZT and NVP (intravenous) intrapartum.
 - c) AZT and NVP for 7-42 days of life in neonate.
 - d) AZT and 3TC for >7 days postpartum in mother.
- New, preferred alternative: "Plan B+"
 - Initiation of lifelong combination ART in all pregnant women, in addition to b) and c) above.

Diagnosis

- Seroconversion (ie. anti-HIV antibody formation) almost always occurs within 3 months of infection.
- Window period: time between infection and seroconversion.
- Standard serologic testing for antibodies is falsely negative during the window period.
- Point-of-care capillary blood testing is most often used in developing countries. Positive results should be confirmed, if possible, with a Western blot.
- If the HIV test is negative but there is still high suspicion of infection, you may proceed with one of the following options:
 - a) repeat test in 3 months.
 - b) test for p24 antigen.
 - c) test with PCR (HIV viral load).

HIV Infections cont'd

Natural History

Acute infection

- 0–6 months
- Rapid decline in CD4 count and high viral loads
 - After acute infection, there is a subsequent lowering of viral load to new set point, and increase in CD4 count.
- Seeding of lymphoid organs and other viral reservoirs
- Acute seroconversion illness:
 - A flu-like illness occurring in approximately 50% of persons newly infected with HIV.
 - Common symptoms include fevers, myalgias, diffuse lymphadenopathy, maculopapular rash and pharyngitis.
 - Less common symptoms include meningitis, encephalitis, Guillain-Barre syndrome and myelopathy.

Latent phase

- Highly variable, generally 3–11 years
- Slow steady decline in CD4 count
- Persistent generalized lymphadenopathy in 35–60% of asymptomatic patients

Symptomatic phase (AIDS):

- If the CD4 count is less than 500, constitutional symptoms may include mucocutaneous lesions, bacterial infections, TB and/or lymphoma
- If the CD4 count is less than 200, the patient is at high risk for AIDS-defining opportunistic infections.

Table 3-2: Opportunistic infections

	<350	<200	<100	<50
Microsporidia & Cryptosporidia diarrhea	X			
Oral Candida (Oral Thrush)	X			
Kaposi Sarcoma	X			
Non-Hodgkins Lymphoma	X			
Oral Hairy Leukoplakia				
Pneumocystis jiroveci Pneumonia (PCP)				
Histoplasmosis		X		
Varicella zoster (VZV), Multidermatomal/Disseminated		X (<150)		
Toxoplasmosis encephalitis		X		
Cryptococcal Meningitis			X	
Burkitt's Lymphoma			X	
Mycobacterium avium complex (MAC), Disseminated			X	
Cytomegalovirus, Retinopathy/				X
Pleuritis/Colitis				X
Progressive Multifocal Leukoencephalopathy (PML)				X
Invasive Aspergillosis				X
Primary CNS Lymphoma				X

HIV Infections cont'd

Chronic HIV infection

Chronic HIV infection causes long-term vascular and neurological damage through constant immune activation:

- Vascular disease
 - Coronary artery disease
 - Peripheral vascular disease
 - Cerebrovascular disease
 - Chronic venous insufficiency
- Distal sensory polyneuropathy
 - Affects 1 out of every 10 persons infected with HIV
 - Occurs in a glove-and-sock distribution and can be very painful
- HIV-associated neurocognitive impairment (HAND)
 - 5–10% prevalence
 - Can lead to HIV-related dementia (AIDS dementia)

Idiosyncratic manifestations to HIV include:

- Kidney disease
 - 1 in 5 has microalbuminuria
 - HIV-associated nephropathy presents with proteinuria
- Pulmonary arterial hypertension
 - Affects 1 in 200
 - Manifests as syncope, chest pain and/ or shortness of breath on exertion

HIV-related complications exacerbated by ART include:

- HIV-associated lipodystrophy
 - Includes hypertriglyceridemia, high cholesterol, hyperinsulinemia and/ or hyperglycemia
 - See fat redistribution with truncal obesity, peripheral wasting, dorsal fat pad, facial wasting
 - Associated with ART
- Osteoporosis
 - Occurs in up to 10% of female patients
 - Only complication of HIV infection made worse by ART

Management Principles

Risk behaviour reduction: safe sex counseling, treatment of drug abuse, perinatal testing and counselling.

Address underlying social barriers to care (social determinants of health), ie. poverty, intimate partner violence, incarceration.

Laboratory evaluation:

Initial:

- Flow cytometry (CD4 count), HIV viral load, viral resistance profile (if available)
- CBC, LFTs, Cr, lipid profile, fasting glucose,
- Toxoplasma IgG, Syphilis serology, HBsAg, Anti-HBsAg, anti-HCV, CMV IgG, TB skin test (>5 mm is positive), CXR

Every 3–6 months:

- At minimum, monitor CD4 count, CBC, Cr, LFTs. Monitor viral load if available
- Other testing as clinically indicated

HIV Infections cont'd

Vaccinations

- Seasonal influenza, TdAP, Hepatitis B, HPV (<27 years old - both sexes), PCV-13 followed by PPV-23
- Live vaccines including Varicella (all children and adults if no history of primary chickenpox/shingles), VZV (a high-dose Varicella vaccine for shingles; adults only), and MMR indicated exclusively in patients with CD4 count >200 cells/mm³

Prophylaxis of opportunistic infections

- Pneumocystis jirovecii: trimethoprim/sulfamethoxazole (TMP/SMX) DS 1 tab 3x/wk
 - Indications: CD4 <200 cells/mm³ or oral thrush or prior PCP
 - Alternatives: dapsone 100 mg daily, atovaquone 1500 mg daily
- Mycobacterium-avium complex:* azithromycin 1200 mg weekly or 600 mg twice weekly
 - Indication: CD4 <50 cells/mm³
 - Alternatives: clarithromycin 500 mg BID, rifabutin 300 mg daily
- Toxoplasma gondii:* trimethoprim/sulfamethoxazole (TMP/SMX) 1 tab 3x/wk
 - CD4 <100 cells/mm³ and Toxoplasma IgG antibody positive
 - *Prior documented disease requires multi-drug regimen
- CMV, HSV, Cryptococcus, Histoplasma capsulatum, Coccidioides immitis, Penicillium marneffeii, Salmonella spp., and Bartonella all have specific prophylaxis guidelines in the setting of a previous infection or exposure and low CD4 count (see reference 6).

Guidelines for initiating ART

- Choice is personal and depends on patient readiness/ability for adherence.
- In resource-rich settings, ART should be initiated early in the course of disease.
- ART is absolutely indicated if CD4 <500 cells/mm³, any opportunistic infection/cancer is present or the patient is pregnant.
- In resource-limited settings, ART initiation depends on local capacity.
 - Indicated if CD4 <350 cells/mm³, any opportunistic infections/cancers, and during pregnancy.
- Highly-active ART, or triple therapy usually consists of two nucleoside analogue reverse transcriptase inhibitors (NRTIs) with one 'backbone' from a different class.

Immune reconstitution inflammatory syndrome:

- An overwhelming immune response to a latent pathogen or other antigen with recovery of the immune system, ranges from mild to life-threatening.
- 10-30% of patients.
- Common triggers include Mycobacterium tuberculosis, Cryptococcus spp., Toxoplasma, and other viral/fungal infections.
- Risk elevated with CD4 <50 cells/mm³ and precipitous decrease in HIV viral load.

HIV Infections cont'd

Table 3-3: Common antiretrovirals

Drug	Short-form	Adverse effects
Nucleoside reverse transcriptase inhibitors (NRTIs)	Class effects:	GI upset, headache, myelosuppression, mitochondrial toxicity
Zidovudine	AZT	Anemia, lactic acidosis, lipodystrophy
Didanosine	ddI	Pancreatitis, neuropathy, LFT abnormalities, lactic acidosis
Stavudine	d4T	Neuropathy, pancreatitis, lactic acidosis, lipodystrophy, hyperlipidemia
Lamivudine	3TC	HBV flare in co-infected patients stopping the medication
Emtricitabine	FTC	Skin discoloration, hepatotoxicity with HBV
Abacavir	ABC	Hypersensitivity (always test HLA-B57*01; fatal), fever, rash, GI upset
Tenofovir	TDF	Renal toxicity, HBV flare if stopped
Non-nucleoside reverse transcriptase inhibitors (NNRTIs)		
Nevirapine	NVP	Hepatotoxicity, rash, *long half-life & high risk of resistance if HAART is stopped
Efavirenz	EFV	Dysphoria, hepatotoxicity, odd dreams, mood changes, lipid abnormalities, teratogenic
Protease Inhibitors (PIs)	Class effects:	Diarrhea, lipodystrophy, insulin resistance
Ritonovir*	<i>r</i>	Hepatitis
Lopinavir (as Kaletra/Aluvia with <i>r</i>)		PR & QT prolongation, pancreatitis
Saquinavir (as Invirase with <i>r</i>)		PR & QT prolongation
Indinavir		Nephrolithiasis, hepatitis
Atazanavir		PR prolongation, rash, hyperbilirubinemia
Darunavir		Rash, hepatitis
Integrase inhibitors**		
Raltegravir (requires BID Rx)		CK elevation, rhabdomyolysis, diarrhea, nausea
Elvitegravir	EVG	Diarrhea
Entry inhibitors		
Maraviroc		
Efuvirtide (salvage therapy)		
Combinations		
Truvada	TDF+FTC	
Atripla	TDF+FTC+EFV	
Combivir	AZT+3TC	
Trizivir	AZT+3TC+ABC	
Triomune	d4T+3TC+NVP	

*Most often used to "boost" bioavailability of other PIs by inhibiting the enzyme that breaks down these PIs
 **Lowers the viral load much quicker than other classes

Further reading:

1. AIDSinfo. A service of the U.S. Department of Health and Human Services: <http://aidsinfo.nih.gov>
2. HIV Insite. A complete, concise HIV resource: <http://hivinsite.ucsf.edu>
3. WHO HIV/AIDS. The World Health Organization website with up to date guidelines for resource-limited settings: <http://www.who.int/hiv/en/>

HIV Infections cont'd

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Diarrheal Diseases

Andrew Bresnahan

General Information

Diarrhea, a preventable and treatable clinical manifestation of disease, is defined as the frequent passage of stools that are looser and more frequent than normal (>200 g/dl for adults on western diets).^{1,2} Diarrhea presenting for less than two weeks is considered acute, while presentation for 2-4 weeks duration is considered persistent, and greater than 4 weeks is considered chronic. True diarrhea is distinguished from other conditions with similar presentations: pseudodiarrhea, the frequent passage of small volumes of stool that is often associated with irritable bowel syndrome, and rectal incontinence, the involuntary discharge of rectal contents often associated with neuromuscular disorders or structural problems. The global incidence of diarrhea is >1 billion/year. It is the second leading cause of death for children under five years of age, killing 1.5 million children and 2-3 million people each year.³

Classification

A first step in approaching diarrhea is to classify its presentation; this can help us understand its etiology and pathophysiology, and help guide our investigations and treatment. Acute diarrhea can be classified as either inflammatory or non-inflammatory, while chronic diarrhea can be classified as: infectious, secretory, steatorrhea, or osmotic in character.

Acute inflammatory diarrhea involves irritation of the intestinal mucosal, often at the small intestine or colon.² Etiologies include a range of infectious agents, including bacteria (Shigella, Salmonella, Campylobacter, Yersinia, E.coli, C. difficile), and protozoa (E. histolytica, Strongyloides), along with other causes such as NSAIDS, IBD, and ischemia.⁶ Mechanisms vary by agent, and can include toxin-induced secretory diarrhea or inflammation-induced exudative diarrhea. Typical clinical presentations are small volume and high frequency stools, blood in stool (dysentery), cramping, urgency with or without tenesmus, and fever +/- shock.⁴ Attention to the incubation period (for infectious causes) and the frequency and severity of vomiting, abdominal pain, fever, and diarrhea may be helpful for diagnosis.

In acute non-inflammatory diarrhea, the mucosa is intact and the insult is usually located at the small intestine. Etiologies include infection by bacterial agents (S. aureus, C. perfringens, B. cereus, E. Coli, Salmonella enteritidis, Vibrio cholera), protozoa (Giardia lamblia), or viruses (Rotavirus, Norwalk, CMV), as well as drug effects (antibiotics, laxatives, antacids). Again, mechanisms vary by agent. Clinical presentations may include large volumes of watery stool with no or little blood, and abdominal pain, +/- shock.⁴

Chronic inflammatory diarrhea is most often caused by neoplasia, IBD, or ischemic bowel, but may also be caused by infectious agents. As with acute inflammatory diarrhea, chronic inflammatory diarrhea is characterized by enterocyte damage, villus atrophy, and crypt hyperplasia[5]. Clinical manifestations can include pain, fever, bleeding, and other markers of inflammation.

Chronic secretory diarrhea occurs with an increase in active gastric secretion or when inhibition of absorption causes an imbalance in the fluid and electrolyte transport mechanisms that draws ions and water across the mucosa into the lumen. Secretory diarrhea can be caused by medications, bowel resection, mucosal disease, enterocolic fistula, hormones, congenital defects in ion absorption, or toxins from infection. Cholera toxin, for example, inhibits the absorptive Na⁺ transport system in intestinal villi cells, while activating the Cl⁻ transport system in crypt cells, resulting in a net accumulation of NaCl in the intestinal lumen. To maintain osmolality, isotonic fluid flows into the lumen. When the volume of fluid exceeds the ability of the gut to reabsorb it, diarrhea results.⁶

Chronic osmotic diarrhea involves ingestion of poorly absorbable osmotically active solutes that draw sufficient fluid into the lumen to overwhelm the reabsorptive capacity of the colon.² Etiologies include the ingestion of osmotic laxatives, and carbohydrate malabsorption, including lactase deficiency.⁴

Chronic steatorrhea is caused by the malabsorption of fats, and leads to the production of oily, floating, foul-smelling stools. With steatorrhea, the osmotic effects of fatty acids cause increased fecal volume, liquidity, and frequency. There are two etiologies of chronic steatorrhea: maldigestion from pancreatic insufficiency or problems with bile production or secretion; and malabsorption from disease of the mucosa, celiac disease and Crohn's disease. However, this form of diarrhea can also occur from secondary causes such as bowel resections, mesenteric ischemia, and bacterial overgrowth.²

Diarrheal Diseases cont'd

Investigations

As the classifications above suggest, investigation of diarrhea should be guided by rational history taking, physical examination, and thoughtful consideration of the patient's social, economic, and ecological context.

Indications for investigation of acute diarrhea include "profuse diarrhea with dehydration, grossly bloody stools, fever 38.5°C, duration >48 hours without improvement, recent antibiotic use, new community outbreaks, associated severe abdominal pain in patients >50 years, and elderly (70 years) or immunocompromised patients".² For patients suspected of acute infectious diarrhea, appropriate investigations may include culture and sensitivity, and ova and parasite testing.⁷ Structural examination by flexible sigmoidoscopy, colonoscopy, CT scan, or other diagnostic imaging may also be helpful for detecting non-infectious sources acute diarrhea.²

Lab tests helpful for the investigation of chronic diarrhea may include those listed above for acute diarrhea, along with blood tests including CBC, chemistry, CRP, TSH, and celiac serology; upper GI endoscopy with duodenal biopsy, among other reasonable investigations.⁴

Treatment and Prevention

Fluid and electrolyte replacement are key to the treatment all types of acute diarrhea. Oral rehydration therapy is appropriate in most cases, though IV treatment may be necessary in cases of extreme dehydration. Anti-diarrheals, including antimotility agents, absorbents, and modifiers of fluid transport, may be helpful for stopping diarrhea. Antibiotics may be indicated with prolonged fever, bleeding, or for certain pathogens. It is important antibiotics are used with caution to prevent drug resistance or dangerous side effects. With chronic diarrhea, treatment modality is highly dependent on etiology.² For both chronic and acute diarrhea, collaboration with public health authorities should be considered where appropriate to limit and prevent outbreaks of infectious disease.

Because diarrhea is a sign of underlying disease, prevention strategies should be tailored to etiology. For infectious causes, special consideration should be given to agent, reservoir, mode of transmission, incubation period, and period of communicability.⁸ Prevention strategies are at their best when they include public policies to advance health equity and improve the social determinants of health.⁹ Primary prevention strategies include the universal provision of safe food and drinking water, sanitation and hygiene, and vaccination campaigns where appropriate.¹ Concurrent disinfection, control of patient contact, and contact investigations may also be appropriate, especially in epidemic settings. Finally, surveillance and other public health intelligence practices can also improve the successful design and delivery of timely prevention campaigns.

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Tuberculosis

Shannon Bucking

“Wherever the art of medicine is loved, there is also a love of humanity.”

–Hippocrates

Current Global Situation and Trends

Global Burden of Tuberculosis

Despite a 36% decrease in global prevalence since 1990, tuberculosis (TB) continues to be the second leading cause of death from an infectious disease worldwide. In 2011, 8.7 million people became sick due to TB and 1.4 million people died.

Geographical Distribution

In geographical terms, the highest burden of TB is found in Africa and Asia. This includes 59% of cases in Asia, 26% in Africa, 8% in the Eastern Mediterranean, 5% in Europe and 3% in the Americas.

Table 5-1: Incident cases of TB by country (2011)

Country	India	China	South Africa	Indonesia	Pakistan
Incident Cases	2.5 million	1.1 million	0.6 million	0.5 million	0.5 million

Burden of TB in Children

An estimated 490 000 new cases of TB and 64 000 deaths due to TB per year occur in children under 15 years. However, estimates are variable as diagnosis of TB is more difficult in children than in adults. Children are also at risk of becoming orphans: almost 10 million children were orphaned in 2009 due to the loss of a parent to TB. Screening for TB in children is a challenging issue as unnecessary chest X-rays lead to radiation exposure, which may increase the risk of malignancy later in life.

HIV/TB Co-infection

Of the 34 million people living with HIV, 33% are infected with latent TB. HIV positive individuals are up to 34 times more likely to develop active TB than those who are HIV negative. In 2011, there were 1.1 million new TB cases in the HIV positive population.

This issue appears to be particularly challenging in Africa, which accounts for 79% of TB cases among people living with HIV worldwide.

Furthermore, TB is the leading cause of death among people living with HIV. Current guidelines recommend that HIV positive individuals be prescribed antiretroviral therapy, regardless of CD4 count.

Multi-Drug Resistant (MDR) TB

MDR-TB is defined as TB that is resistant to isoniazid and rifampicin, the first-line drugs used in the treatment of TB. Drug resistance may develop when a patient is unable to complete their first course of medications. Barriers include access to care, financial considerations and medication side effects. Current World Health Organization guidelines recommend treatment of MDR-TB with second-line TB drugs for twenty months.

Tuberculosis cont'd

General Information

Definition

Tuberculosis is an infectious disease caused by *Mycobacterium tuberculosis*, a bacteria in the genus *mycobacterium*. Distinction must be made between infection with TB and disease. Infection simply means the presence of organisms, but there may be no clinically significant disease. This is also referred to as latent infection.

TB is acquired through airborne transmission of infected droplet particles from an individual with active TB coughing or sneezing. Poverty and crowded living conditions may lead to tuberculosis infection as a result of many people living in close proximity to one another. Susceptible people (children, elderly, immunosuppressed) are at particular risk of transmission.

Pulmonary TB involves the lung parenchyma, whereas **extra-pulmonary TB** can include the pleura, lymph nodes, abdomen, genitourinary tract, skin, joints, bones, and meninges.

Pathogenesis

Primary TB is the form of the disease that develops in a previously unexposed, unsensitized person, whereas secondary TB is the form that arises in a previously sensitized host. Primary TB does not usually present with symptoms in healthy individuals, though occasionally fever and pleural effusion may be present.

If infected, a patient may have a positive Mantoux (PPD) skin test, caused by delayed hypersensitivity, indicating T-cell mediated immunity to mycobacterial antigens. However, false-negative reactions are common.

Secondary TB may develop years to decades after primary TB. This usually occurs when the immune response is depressed in the host, such as in the setting of HIV, malignancy or long-term use of immunosuppressant drugs. Secondary TB may be due to reactivation of the latent infection, or it may be an exogenous reinfection, after exposure to another contagious individual.

The patient with secondary TB often develops prominent lung lesions, especially in the apices of both lungs. The mycobacteria enter the airspaces and trigger a tissue response to protect themselves, causing cavitation. These cavitations can then erode into the bronchioles and bronchi, increasing the risk of transmission to others.

Clinical Features

Signs & Symptoms

As a result of the release of inflammatory markers, the patient may develop systemic symptoms, including cough, weight loss, malaise, anorexia and night sweats. Sputum is usually present, and increases in amount with disease progression. It may be purulent in late stages. Hemoptysis may also be present. Fever is also a clinical feature of TB and presents with a classic pattern: patients are often afebrile in the morning and then peak in late afternoon or evening, with fever subsiding throughout the night.

Other clinical features that may or may not be present are dyspnea, pleuritic chest pain and ulceration of mucous membranes. Complications of TB include: hemoptysis, pneumothorax, bronchiectasis, extensive pulmonary destruction, malignancy, and chronic pulmonary aspergillosis.

Tuberculosis cont'd

Diagnosis

According to the World Health Organization (WHO), a definite case of TB is defined as a patient with mycobacterium tuberculosis complex identified from a clinical specimen, via culture or molecular assay. A patient with pulmonary symptoms suggestive of TB and a sputum sample that is acid-fast bacilli (AFB) positive is also considered a definitive case in a setting with inadequate lab facilities.

Sputum Smear

Sputum smear microscopy is the most common method to diagnose TB worldwide. A sputum sample is provided by the patient, and a smear of the sample is examined under the microscope. *M. tuberculosis* are slender, aerobic rods that grow in straight or branching chains. Their cell wall contains mycolic acid, which retains stains, the basis for the term "acid-fast bacilli".

Chest Radiograph

Pulmonary TB is characterized by the presence of: nodular or alveolar infiltrates with cavitation, pleural effusion, pulmonary nodules, hilar or mediastinal adenopathy, and semi-calcified well-defined solitary coin lesion, termed tuberculoma on chest X-ray.

Treatment

The goal of treatment is to eradicate infection with *M. tuberculosis* and prevent relapse. Directly observed therapy has been supported by strong evidence and used worldwide, in order to promote adherence and prevent the development of MDR-TB.

Treatment of active TB consists of four medications: isoniazid (INH), rifampicin (RIF), ethambutol and pyrazinamide. Current guidelines recommend treatment with the four drugs during the initial phase for 2 months, and then INH and RIF during a continuation phase for another 4-7 months, depending on the patient's initial severity of disease. The WHO recommends a repeat sputum smear at 2 months after beginning treatment to assess efficacy.

Adverse effects of medications include: hepatotoxicity, ocular toxicity, rash and drug fever.

Vaccination

The Bacille Calmette-Guerin (BCG) vaccine, which has been used in many parts of the world for over a century, is the most frequently administered vaccine. It is made from an attenuated strain of bovine tuberculosis, is administered to neonates, and prevents severe, disseminated TB. However, the BCG vaccine is currently recommended only in countries with a high TB burden.

Recent attempts to develop another TB vaccine have focused on optimizing any pre-existing immunity granted by the BCG vaccine. However, results have been inconclusive, and further research is needed.

Global Health Policy

In 2010, the success rate for treatment of new TB cases was 85%, according to the WHO. This may in part be due to international cooperation through the Stop TB Strategy, which aims to dramatically reduce the global burden of TB by 2015 in line with the Millennium Development Goals. Specific objectives include: achieving universal access to high-quality care for all people with TB, reduce the human suffering and socioeconomic burden associated with TB, protect vulnerable populations, support the development of new tools, and protect human rights in TB prevention, care and control. Estimates indicate that we are on target to meet a component of MDG 6: to halt and begin to reverse the incidence of TB by 2015. However, there is still much work to be done.

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Malnutrition

Alia Dharamsi

Malnutrition is a broad term that encompasses both states of undernourishment and overnourishment. Undernourishment can result from either a limited intake of food or poor gastrointestinal absorption; overnourishment results from an intake above the body's needs. The number of overnourished individuals is now approximately equal to undernourished individuals in the world. However, overnourishment remains a disease of the wealthy, whereas undernourishment is a disease of the millions of people living in poverty internationally. For the purposes of this handbook, the focus will be on undernourishment as this is what most individuals working abroad will encounter as they embark on international service and humanitarian endeavours.

Global Malnutrition¹

- 12% (870 million) of the world's population is undernourished.
- In 1990 the Millennium Development Goals (MDG) were created: MDG 1 was to "halve the proportion of hungry people in developing countries by 2015, to 11.6%".
 - Globally, the number of hungry people in developing countries has decreased from 23.3% in 1990 .
- 98% of the world's hungry live in developing countries.
 - Almost 15% of the population of resource-poor countries are undernourished.
 - Asia and the Pacific have 563 million hungry people, although these numbers are declining.
 - The number of hungry people continues to rise in sub-Saharan Africa.
- Women account for 60% of the world's hungry.

Childhood Malnutrition¹

- Children under the age of 5 are most susceptible to death and illness from malnutrition.
 - In this age range, irreversible physical and cognitive delay can occur from undernutrition.
- 2.6 million children under 5 die each year due to undernutrition.
- 1/6 children (100 million) in developing countries is underweight.
- 1/4 children in the world are stunted; in some developing countries this number can be as high as 1/3.

Malnutrition cont'd

Understanding the Language of Malnutrition^{2,3}

Malnutrition is when food intake or absorption are inadequate to sustain a person's normal physiological function, including growth, learning, physical work, immune function, pregnancy and lactation.

Undernutrition occurs when intake does not provide enough calories to meet normal physiologic needs.

Protein-energy malnutrition refers to a form of undernutrition where protein intake is insufficient; in this case caloric intake may be adequate but the diet is low in protein. Children are most susceptible to protein-energy malnutrition because of their increased protein needs during growth.

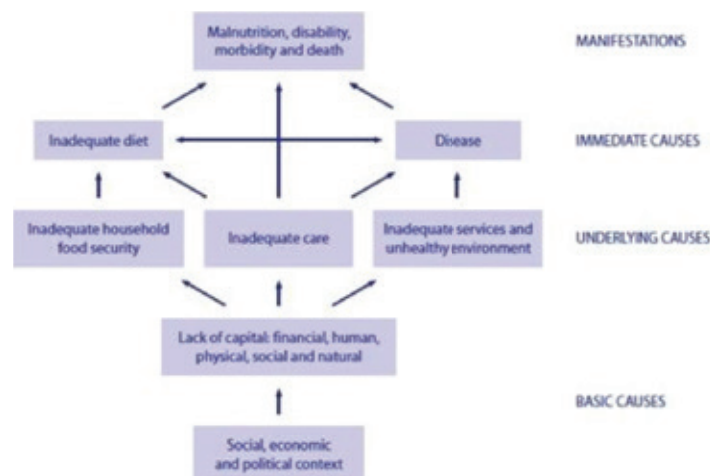
Anthropometry (body measurements) are used along with clinical assessment to further qualify undernutrition in children into a range of conditions including:

- **Underweight:** low weight-for-age as compared to a reference population of well nourished and healthy children.
 - **Chronic Malnutrition/Stunting:** height-for-age <15th percentile. Stunting is an indicator of chronic malnutrition. Stunting demonstrates the effect of chronically poor nutrition on a child's growth over time. Approximately 1/3 of the world's children are stunted. When travelling abroad you may see children who appear to be of healthy size and weight. However when you ask them their age you may be surprised to find out they are 2, 3, or even 5 years older than their height leads you to believe.
 - **Acute Malnutrition/Wasting:** weight-for-height <3rd percentile. Wasting is a result of substantial weight loss, usually associated with a recent/severe process like starvation and/or disease. 55 million children suffer from acute malnutrition globally. Acute malnutrition can be measured with weight/height and also by MUAC: Mid Upper Arm Circumference (a band with standardized measurements around the upper arm).
- **Acute malnutrition is classified into:**
 - Moderate acute malnutrition (MAM)
Weight-for-height 70-79th percentile
 - Severe acute malnutrition (SAM) presents with in two ways
 - a) **Marasmus:** Weight-for-height <70th percentile
 - severe malnutrition, mainly due to energy (calorie) deficiency.
 - **Clinical picture:** tissue and muscle wasting, loose gluteal folds, severe loss of adipose tissue from thighs and buttocks, very hungry
 - b) **Kwashiorkor:** Weight-for-height 60-80th percentile (and edema)
 - severe malnutrition primarily due to insufficient protein intake
 - **Clinical picture:** pedal edema, distended abdomen, thinning hair, loss of teeth, dermatitis, enlarged and fatty liver, irritable
 - Any child with edema is automatically classified as severe malnutrition

Causes of Malnutrition⁴

Malnutrition is caused by an interplay of factors that affect the health of individuals in a given area. Immediate factors act on the individual, underlying factors acting at a household level, and basic factors act on society in general.

Figure 6-1: A simplified model for understanding causes of malnutrition⁴



Malnutrition cont'd

Immediate causes of malnutrition include inadequate intake (relative to need) and disease. Malnutrition increases susceptibility to infection, which can exacerbate malnutrition: this leads to a spiral of malnutrition-infection. During times of crisis/emergency, children are likely to die from diarrheal illness, respiratory illness, measles and malaria due not only to poor sanitation and crowding (as seen in refugee camps and slums) but also due to underlying malnutrition, which impedes their immune system's ability to protect against disease.

Management of Malnutrition⁵

Management of Moderate Acute Malnutrition (MAM)

- Supplemental feeding programs (SFPs) can not only improve the nutritional status of those with MAM, but also prevent them from developing SAM. Supplemental feeding programs typically target those at high risk including children 6 months - 5 years, pregnant women, and breastfeeding mothers. SFPs are typically started in emergency situations, or when ration distribution systems are being started in an area (e.g. refugee camps).
- SFPs can target specific individuals who have MAM ("targeted" SFPs), or all members of an at-risk group ("blanket" SFPs). Decisions to implement a targeted to blanket SFP usually depends on available resources.
- Take-home supplement rations (ie: dry rations they can prepare themselves) are preferable to SFPs that require individuals to come to a center to eat prepared food.
- Supplements should be energy dense, micronutrient rich, and culturally appropriate.

Management of Severe Acute Malnutrition (SAM)⁶

- SAM is a severe, life threatening condition. Individuals with SAM but no other medical conditions can be managed as outpatients through health posts/clinics. Individuals with SAM and other medical complications should be treated as inpatients as they require close monitoring.
- "Ready to use therapeutic foods" (RUTF) are used to treat SAM, and are an energy-dense, micronutrient-enriched paste that can be eaten straight from the package and require no preparation. "Plumpy'nut" is a peanut based product that has been widely used for community-treatment of SAM. It is very high in calories and protein leading to rapid weight gain.

Micronutrient Malnutrition^{7,8}

Micronutrients are essential inorganic compounds needed in small amounts for normal enzymatic, hormonal, and physiological function. On a global public health scale, iodine, Vitamin A and iron deficiency present a major threat to the well-being of children and pregnant women.

Iodine Deficiency⁹

Iodine deficiency is the most prevalent cause of brain damage and impaired cognitive development in children. Iodine deficiency during pregnancy can result in miscarriage or stillbirth or cause cretinism (irreversible mental retardation). Iodine deficiency can result in decreased intellectual function, and impact a person's ability to function in school and at work. Iodine deficiency is easily preventable with the iodization of salt. Iodization programs have resulted in a halving of the number of countries where iodine deficiency is a health concern in the population.

Vitamin A Deficiency¹⁰

Vitamin A deficiency has significant effects on the immune system and vision. Vitamin A deficiency is the leading cause of preventable blindness in children. As well, lack of Vitamin A leads to an increased susceptibility to infection (ie: diarrheal illnesses and measles), and an increased probability of death from infection. The effect of vitamin A deficiency is also seen in pregnant women, who are at an increased risk of mortality from infection and may present with night blindness. Current efforts to combat Vitamin A deficiency include vitamin A supplementation, encouraging increased growth and consumption of Vitamin A rich foods, as well as food fortification.

Malnutrition cont'd

Iron Deficiency¹¹

Two billion people around the world are anemic, mostly due to iron deficiency. In some countries 1/2 pregnant women are anemic and 40% of children in preschool are anemic. Anemia can be exacerbated by infections with malaria, HIV/AIDS, parasite infection (e.g. schistosomiasis), and tuberculosis. The effects of anemia may be subtle, but they reduce the ability of people to learn in school or to work. This leads to economic consequences impeding national growth. To counter iron-deficiency anemia, consumption of iron rich foods, food fortification and iron supplementation can be implemented. In addition, it is important to control infection, improve sanitation, and immunize against preventable illnesses. Other vitamins and minerals like folate, B12 and vitamin A are also necessary to ensure appropriate production of red blood cells and defense against infection.

Micronutrient supplementation strategies¹²

Micronutrients can be supplied through diet, by increasing the diversity of food intake, or by fortification of food. Encouraging agricultural development, home gardening, and improving access to markets and diet diversity, all increase the quality of nutrient intake. Micronutrient supplements can also be given to families to add vitamins and minerals to existing foods when dietary nutrient intake is limited. “Sprinkles” is a supplement that was developed by a Canadian pediatrician as a way of fortifying foods in the home with necessary micronutrients. Individual sachets are about the size of a packet of sugar and they can be added to any food without changing the texture or flavor. In addition, it is possible to supplement with single nutrients via tablets or oral suspensions.

The Link Between Maternal and Child Health: First 1000 days¹³

It has been understood for decades that there is a very intimate link between the health of a pregnant mother and the health of her infant. The 1000 days between conception and a child's 2nd birthday are crucial to ensure a healthy future for baby. Providing appropriate nutrition to the mother during pregnancy and during the first 2 years of life of the child can have significantly positive impacts on a child's ability to learn and grow, as well as increase survival and health long-term. The First 1000 Days initiative focuses on ensuring mothers and young children have appropriate amounts of vitamins and minerals, educating mothers about nutritional practices like healthy foods and breast feeding, and providing treatment/prevention of malnourishment with therapeutic foods.

As You Move Forward

As you travel you will see the effects of malnourishment and undernourishment around you. Key points to take away from this chapter include the factors contributing to malnutrition, the malnutrition-infection cycle, the various ways undernutrition can be measured, and the importance of micronutrients in development. It is impossible to delve into the various interventions and programs that are in place working tirelessly to improve the health and nutritional status of people worldwide. The goal of this chapter was to introduce you to common vocabulary used to describe undernutrition. It is hoped that you feel better equipped to join the international conversation on nutrition and health as we all strive to ensure that everyone has access to enough macro- and micronutrients to survive and thrive.

Malnutrition cont'd

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Respiratory Infections Adeel Sherazi

General Information

Acute respiratory infections (ARIs) are a leading cause of morbidity and mortality with a death rate of a staggering 4 million people per year. Pneumonia, influenza and tuberculosis are amongst the most serious of the ARIs.

In developing countries, pneumonia is mainly a disease of children and young adults and accounts for about 28% of all pediatric mortalities. Nearly three-quarters of the 156 million yearly incidences of pneumonia occur in just 15 countries, mostly in South Asia and sub-Saharan Africa. Within these countries, up to half of the patients presenting to an outpatient hospital department will have an ARI.

Pneumonia is treatable with antibiotics, but in many areas of the world poor availability of appropriate therapeutic agents and limited diagnostic tools contribute to the severity of infection. Other factors contributing to infection include overcrowding, poor sanitation, seasonal trends and smoke from cooking fires. Additionally, malnourished infants are at a significantly greater risk of contracting pneumonia as compared to infants of normal weight.

The etiology of pneumonia is bacterial or viral infections that damage the lower respiratory tract. The result is that alveoli become filled with fluid and pus secondary to inflammation, which limits gas exchange and makes breathing difficult.

Classification

There are a number of ways to classify pneumonia and this can influence the approach to treatment. Table 7-1 gives several examples that are commonly used.

Clinical Features and Assessment

In many cases, the lack of appropriate diagnostic resources may limit how well one can diagnose pneumonia and determine the correct etiology. However, there are several key features on clinical presentation listed below that should be noted. In addition, the CURB-65 Criteria is a useful and easy to remember clinical prediction rule for identifying increased mortality in a patient with pneumonia.

Symptoms that Could Indicate Pneumonia

- Specific Symptoms
 - New cough with or without sputum, or a change in sputum colour
 - Chest discomfort

Table 7-1: Classification of respiratory infections

Primary vs. Secondary	Clinical Classification
Primary Previously healthy patient Triggered by an especially virulent organism such as <i>S. aureus</i> or <i>S. pneumoniae</i> Lobar lung involvement	Community-Acquired Pneumonia 90% of cases by person-to-person transfer Other cases because of environmental agents or transfer from animals
Secondary Patient has predisposing condition. These are most commonly immunocompromise (ex AIDS), bronchial obstruction (ex asthma or a neoplasm), decreased ventilation (ex secondary to surgery or pain) Triggered by organisms of high or low virulence Typically a patchy bronchopneumonia	Hospital-Acquired (Nosocomial) Pneumonia Pneumonia that occurs more than 48 hours after admission Usually secondary to aspiration, for example following vomiting or due to impaired consciousness Can be passed from inhalation of bacteria from contaminated instruments
Etiological Agent (in order of prevalence) Bacteria <i>Streptococcus pneumoniae</i> <i>Haemophilus influenzae</i> <i>Mycobacterium tuberculosis</i> <i>Staphylococcus aureus</i> (especially virulent and common in hospitals) <i>Pneumocystis jirovecii</i> (most commonly in HIV-infected patients) Gram-negative bacteria	Recurrent Pneumonia More than two episodes of pneumonia due to: 1) Localized respiratory disease (bronchiectasis, bronchial obstruction) 2) Generalized respiratory disease (COPD, impaired respiratory defences) 3) Non-respiratory problem (recurrent aspiration, immunosuppression) 4) Acquired immunodeficiency (AIDS)
Viruses Respiratory syncytial virus (RSV) Influenza virus A and B Human paramyxovirus (hPIV's)	
Fungi (rare) <i>Pneumocystis pneumonia</i> (immunocompromised patients) Histoplasmosis	

Respiratory Infections cont'd

- Non-Specific Symptoms (more likely in children and older adults)
 - Constitutional: Rigors, sweats, fatigue, chills, myalgias
 - Abdominal Pain
 - Headaches

Strong Indicators of Pneumonia in Children

- Fever of greater than 38°C
- Peripheral cyanosis
- Dyspnea. This is more severe if there is a tracheal tug, nasal flaring or use of accessory muscles.
- Knowledge of missing immunizations

CURB-65 Guidelines to Indicate Severity (Look for 2 or more signs)

- Confusion
- Uremia (blood urea nitrogen ≥ 20 mg/dL)
- Respiratory rate > 30 breaths/min
- Blood pressure ≤ 90 mm Hg systolic or < 60 mm Hg diastolic
- Age ≥ 65 yr (often present with comorbidities such as CHF and renal disease)

Assessment

- History
 - Learn about the onset, character and timing of all symptoms.
 - Find out about any past ARIs and the treatment.
 - Identify risk factors such as smoking, travel and past antibiotic use.
 - Ask about living conditions and past hospitalizations.
- Physical Exam
 - Inspection: Check for fever, chills, shortness of breath, cough (with or without sputum), increased respiratory rate, signs of distress.
 - Palpation: Assess for sharp stabbing (i.e. pleuritic) pain localized in areas of the chest that is worse with deep inspiration and may change with position.
 - Percussion: Note any dullness in areas of consolidation along with altered transmission of breath sounds with egophony and increased tactile fremitus.
 - Auscultation: Listen for inspiratory crackles, crepitations and pleural rub.

Diagnostic Tests

There may be limited resources for investigations so be aware that the decision to treat may have to be made on clinical grounds alone. It is important to rely on the history and the physical examination and be confident in one's clinical gestalt.

- Pulse Oximetry
 - A good test to do on initial assessment. Patients with pneumonia may show decreased oxygen saturation and it is especially dangerous with $\text{SaO}_2 \leq 92\%$.
- Chest X-ray
 - Look for opacifications – these may be lobe-specific or distributed randomly among both lungs. Bilateral, interstitial distribution is an indication of a viral origin.
- Culture of sputum
 - Hard to assess and a high risk of contamination. If other signs are pointing to a pneumonia, avoid this step.

Respiratory Infections cont'd

Differential Diagnosis

- Acute Bronchitis (Inflammation of the bronchi)
 - May present with low-grade fever, postnasal drip and absence of x-ray findings.
 - Does not require antibiotic therapy unless there is a coexisting medical condition or purulent sputum.
- COPD (Airflow limitation due to inflammation, mucus and alveolar damage)
 - Patients will typically have a cough and prominent wheezing along with signs of respiratory distress such as pursed lip breathing.
 - Chest will be hyperresonant and there can be absence of x-ray findings.
 - May present with edema secondary to cor pulmonale.
- Acute Respiratory Distress Syndrome/ARDS (Inflammatory condition of the lung)
 - Sudden onset of respiratory failure secondary to pulmonary edema.
 - Chest x-ray with prominent, diffuse, bilateral infiltrates and may have total whiteout.
 - Usually occurs secondary to sepsis, severe trauma and drug overdose, among others.
- Influenza (Viral infection)
 - Most commonly presents with chills, sore throat, fever, myalgias, rhinorrhea, cough, weakness and/or a headache.
 - Chest x-ray is usually clear.
- Severe Acute Respiratory Syndrome/SARS (Viral respiratory disease)
 - Develops quickly and begins with a fever and mild symptoms of headache and myalgia.
 - Progresses over several days to the lower respiratory tract and causes dry cough, shortness of breath, and pulmonary infiltrates.
- Others
 - Tuberculosis (discussed earlier)
 - Congestive Heart Failure
 - Pulmonary Embolism
 - Pleural complications leading to effusions, empyema and abscesses
 - Sarcoidosis: non-productive cough and manifestations in other organ systems

Treatment and Prevention

The focus is to protect, prevent and treat pneumonia or other ARIs when they are present. Means of protection include limiting exposure to smoke or infectious individuals. Additionally, proper nutrition and breastfeeding are key methods of protection as they maintain the body's immune function. The mainstay of prevention is vaccinations, including the following: influenza, pneumococcus, measles, and whooping cough (pertussis). An important consideration with vaccinations is that strains of infectious agents vary in different parts of the world and antigens for regionally prevalent strains may be absent in vaccines that are typically marketed for industrialized countries.

Treatment:

1. Oxygen—if possible, monitor O₂ saturations
2. Antimicrobials
3. Analgesia for pleuritic pain
4. Fluids for dehydration, watching for adequate urine output (1.5 L/24hrs)
5. Bed rest—sitting up rather than lying flat to avoid aspiration risk
6. Treat empirically for sepsis if very ill

Respiratory Infections cont'd

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Maternal Mortality

Anastasia Blake

“An estimated 6,000 mothers die in childbirth every year [in Uganda]. The cause? Lack of skilled attendants and emergency care. More often than not, if mothers do arrive at a care facility, they’re already half dead....It takes about four hours for a mother to bleed to death after delivery. But when you deliver at home and live two hours from the nearest clinic, time is not in your favour.”

–Dr. Jean Chamberlain Froese

General Information

“Every two minutes a woman dies from potentially avoidable or preventable problems in pregnancy or childbirth.”(WHO). This dire situation has recently gained some much-needed attention from global health activists and has been recognized by the United Nations Development Programme in the form of Millennium Development Goal Number 5 (MDG5): “[to reduce] by three-fourths the maternal mortality ratio and achieve universal access to reproductive health”. The international efforts initiated to address MDG5 have led to a decline in global maternal mortality of 3.1% per year between 1990 and 2010, while in some Low- or Middle-Income Countries (LMICs) the maternal mortality rates have not changed. This is far from the target of 5.5% per year required to achieve MDG5 by 2015.

Nearly 99% of all maternal deaths occur in developing countries, with more than half of these deaths occurring in sub-Saharan Africa and almost one-third in South Asia. Antepartum and intrapartum complications are the leading cause of death among adolescent females in most developing countries. Furthermore, women in developing countries have many more pregnancies throughout their lifetime, increasing their lifetime risk of pregnancy-related death from 1:3800 to 1:150.

There are four major pregnancy-related complications that are responsible for approximately 80% of maternal deaths worldwide: postpartum hemorrhage, infection, hypertensive disorders of pregnancy and unsafe abortion. Yet death from these complications can be prevented with improved access to antenatal care, skilled birth attendants and postpartum care. However, a multitude of barriers continue to prevent women from receiving or seeking out the care they need, including: poverty, cultural practices, geographic isolation, inadequate services, and lack of education about when to seek care. In short, much more needs to be done to improve access to these essential services in the developing world.

General Approach

The Integrated Management of Pregnancy and Childbirth (IMPAC) manual “Managing Complications in Pregnancy and Childbirth” recommends that any pregnant woman with red flag symptoms including bloody show with palpable contractions, ruptured membranes, weakness, syncope, severe headache, blurred vision, vomiting, fever and respiratory distress should be rapidly assessed with the following approach:

1. **Airway and Breathing:** examine for cyanosis, respiratory distress, pallor, wheezing/rales - consider severe anemia, heart failure, pneumonia, asthma
 2. **Circulation:** examine for cool/clammy skin, HR > 110 and BP < 90 - shock
 3. **Vaginal Bleeding:** assess gestational age vs recently given birth, history of retained placenta, amount of bleeding, examine abdomen +/- vulva, uterus
 - a) If < 22 weeks GA, consider abortion, ectopic, molar pregnancy
 - b) If > 22 weeks GA, consider abruption, uterine rupture, previa
 - c) If postpartum, consider atony, lacerations, retained placenta, inverted uterus
 - d) DO NOT DO A VAGINAL EXAM IF > 22 WEEKS GA WITH VAGINAL BLEEDING AND PLACENTAL LOCATION UNKNOWN (if ultrasound not available proceed with speculum exam with caution).
 4. **Unconscious, Convulsing or Febrile:** assess GA, BP (especially dBP >95), temperature (>38oC), symptoms of infection (neck stiffness, shallow breathing, acute abdomen, purulent vaginal discharge, tender breasts) - consider eclampsia, malaria, epilepsy, tetanus, endometritis, septic abortion, mastitis, meningitis
- Abdominal Pain:** assess GA, location of pain, unilateral/bilateral, vital signs
- a) If < 22 weeks GA, consider ectopic pregnancy, spontaneous abortion, or other causes for abdominal pain (ovarian torsion, appendicitis, pyelonephritis, etc.)
 - b) If > 22 weeks GA, consider preterm/term labour, chorioamnionitis, abruption, sepsis, uterine rupture

Maternal Mortality cont'd

Clinical Features

- Normal Labour and Childbirth: Labour is regular, frequent uterine contractions WITH cervical change (both dilation and effacement)
 - Abnormal fetal heart rate is indicated by FHR <100 or >160 and presence of meconium in amniotic fluid
 - Diagnosis of active labour confirmed by serial vaginal exams demonstrating progressive cervical effacement and dilation
 - Presenting part determined by abdominal palpation and cervical exam
- Malposition/Malpresentation: any position other than OA or any presentation other than vertex (ex. face, brow, frank (extended) breech, footling breech, complete (flexed) breech) Planned vaginal delivery of complete or frank breech may be preferable than C/S depending on local resources especially if ECV not available or indicated.
- Shoulder Dystocia: failure of shoulders to deliver spontaneously by usual methods, dystocia can be accompanied by the appearance and retraction of fetal head (turtle sign), fetal head remains tightly applied to the vulva and traction fails to deliver the shoulders
- Antepartum/Postpartum Hemorrhage: vaginal bleeding during or immediately after pregnancy +/- signs of hemorrhagic shock, abdominal pain or cramping, visible trauma to vulva/cervix/perineum
- Postpartum Infection: fever, lower abdominal pain, prolonged bleeding, fever, foul-smelling or purulent discharge, cervical motion tenderness, nausea, vomiting, +/- rebound tenderness, signs of shock
- Hypertensive Disorders of Pregnancy: elevated BP (especially dBp >95), headache, blurred vision, oliguria, epigastric or RUQ pain, altered LOC, seizures, proteinuria, pulmonary edema, hyperreflexia
- Gestational Hypertension: onset of hypertension between 20 weeks GA and 48-hours post-delivery
 - Pre-eclampsia: HTN + proteinuria
 - Eclampsia: HTN + proteinuria + tonic-clonic seizures

Differential Diagnosis

Vaginal Bleeding in Pregnancy:

- Early (<22 weeks): ectopic, abortion (incomplete, inevitable, complete), molar pregnancy
- Late (>22 weeks): abruption, previa, ruptured uterus
- Postpartum: 4 T's "tone, tissue, trauma, thrombin"

Fever in Pregnancy/Postpartum: septic abortion, chorioamnionitis, endometritis, pelvic abscess, other infection (pyelonephritis, pneumonia, malaria, typhoid), DVT/PE

Altered Level of Consciousness in Pregnancy: eclampsia, epilepsy, severe malaria, meningitis, encephalitis

Abdominal Pain in Pregnancy: ectopic, abortion, abruption, torsion of ovarian cyst, pre-term labour/labour, infection, other abdominal pathology (appendicitis, cholecystitis, pyelonephritis, etc.)

Treatment and Prevention

Normal Labour:

- Encourage pushing once fully dilated; as head delivers, maintain head flexion with fingers of one hand and protect perineum with other, encourage small pushes
- Feel for a nuchal cord and attempt to slip it over the baby's head; if the cord is tight around the neck, doubly clamp and cut it
- Allow anterior shoulder to reconstitute and deliver while supporting perineum. No traction often necessary. If traction required, deliver the anterior shoulder by gentle downward traction on the head followed by gentle upward traction to deliver the posterior shoulder
- Dry the baby and place it on the mother's abdomen, delayed cord clamping now suggested due to benefits with increasing neonatal hemoglobin
- Active Management of the Third Stage of Labour: oxytocin 10 Units IM immediately after delivery, then apply steady cord traction with counter-traction above the pubic bone

Maternal Mortality cont'd

Malposition/Malpresentation:

- Monitor closely as increased risk of uterine rupture and obstructed labour
- OP (occiput posterior) position: spontaneous rotation to OA (occiput anterior) will occur in 90% of cases; if fetal distress or signs of obstruction, deliver by caesarean section
- **Brow:** unusual for spontaneous conversion, deliver by caesarean section
- **Face:** mentum-anterior may deliver spontaneously; if mentum-posterior, deliver by caesarean section
- **Breech:** vaginal delivery is possible with complete and frank breech presentations, but emergency caesarean section should be available if needed
 - Do not rupture membranes artificially; if membranes rupture spontaneously, examine cervix immediately to exclude cord prolapse
 - If signs of abnormal heart rate or dystocia, deliver by caesarean section
 - If fetal surveillance normal, encourage maternal pushing and allow delivery to occur spontaneously until body has delivered
 - Have an assistant apply suprapubic pressure to maintain head in flexion and use a modified Mauriceau-Smellie-Veit (MSV) maneuver to flex the head
 - MSV maneuver: place one hand on baby's back with a finger pushing down on the occiput, place the other hand beneath the baby with forearm supporting it and two fingers pushing on the maxillae to flex head

Shoulder Dystocia:

- "ALARMER" mnemonic for management:
 - Ask for help
 - Lower head of bed and Legs hyperflexed (McRoberts' maneuver)
 - Anterior disimpaction (suprapubic pressure)
 - Rotate of posterior shoulder (apply posterior pressure on anterior shoulder and anterior pressure on posterior shoulder to rotate 180°)
 - Manual delivery of posterior arm (grasp humerus of posterior arm, flex arm and sweep across chest)
 - Episiotomy (to make more space for hands to accomplish maneuvers)
 - Roll over on all fours (and re-attempt previous maneuvers)

Antepartum Hemorrhage:

- ABCs, rapid IV infusion, assess clotting status, transfuse as necessary, monitor fetal status and deliver rapidly if deterioration or massive hemorrhage
- If bleeding slows and no fetal distress, manage expectantly with close observation in hospital

Postpartum Hemorrhage:

- Prevention with active management of the third stage of labour (AMTSL)
- ABCs and treat shock as above; palpate uterine fundus to assess tone, massage uterus to expel blood clots, check for lacerations of vagina/cervix/perineum, examine placenta for missing fragments and manually explore uterus
 - TONE: bimanual massage, uterotonics
 - TRAUMA: repair lacerations, if uterine inversion → reposition immediately prior to giving uterotonics
 - TISSUE: remove remaining placental tissue manually
 - THROMBIN: assess for coagulopathy if possible, manage with transfusion of blood products as available

Postpartum infection:

- Antibiotics: ampicillin 2 g IV q6h + gentamycin 5 mg/kg IV q24h + metronidazole 500 mg IV q8h
- If retained placenta possible, perform manual exploration of uterus
- If no improvement with IV antibiotics or worsening condition, laparotomy +/- hysterectomy

Maternal Mortality cont'd

Hypertensive Disorders of Pregnancy:

- Prevention of complications by close monitoring of BP, reflexes, urine, fetal condition
- If signs of fetal compromise, expedite delivery
- If convulsions: manage ABCs, position on left side, IV, foley to monitor urine output
 - Magnesium sulfate (loading dose: 4 g of 20% magnesium sulfate IV over 5 minutes, followed by 5 g 50% magnesium sulfate IM in each buttock), monitor for signs of toxicity
 - If magnesium not available, use Diazepam 10 mg IV over 2 minutes
 - If dBP >110 mmHg, give antihypertensive drugs (Hydralazine 5 mg IV over 5 minutes or Labetolol 10 mg IV)
 - Delivery within 24 hours of onset

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Pediatric Presentations

Matthew Carwana

Common Definitions in Infant and Child Health

- Infant Mortality: Number of deaths of children less than one year per 1000 live births
- Neonatal Mortality: Number of deaths of children within 28 days postpartum per 1000 live births
- Perinatal Mortality: Number of stillbirths (>22 weeks gestational age) and deaths in the first week of life per 1000 live births
- Under-5 Mortality: Rate of death between birth and 5 years per 1000 live births

Figure 9-1: Under-5 Mortality, 2010 (Source: The Lancet)



Care of the Neonate

General Information

- Nearly 40% of all under-five child deaths are neonates (first 28 days of life)
- 3/4 of newborn deaths occur during the first week of life (35-45% within the first 24 hours)
- In LMICs as many as half of all mothers and newborns do not receive skilled care during and immediately after birth
- 2/3 of newborn deaths can be prevented with skilled intervention at birth and during the first week of life

Clinical Features

- Red flags for high-risk newborn:
 - Mother without antenatal care
 - Mother with significant medical history (HIV positive, drug use, pregnancy induced hypertension etc.)
 - Infant born prematurely (<37 weeks GA)
 - Infant small for gestational age
 - Intrapartum complications (maternal fever, premature rupture of membranes, fetal distress, prolonged labour, meconium in fluid)

Signs of a seriously ill newborn: inability to breastfeed, convulsions, drowsy/unconscious, respiratory rate <20 or apnea (>15 seconds), tachypnea (>60 breaths/min), grunting, severe chest indrawing, grey appearance, central cyanosis, deep jaundice, severe abdominal distension

Differential Diagnosis

Broad differential for ill-appearing newborn, can be broken down into several categories:

- Respiratory – respiratory distress syndrome, transient tachypnea of the newborn, pneumothorax, meconium aspiration syndrome
- Cardiac – congenital heart defects
- Infection – sepsis, meningitis/encephalitis
- Metabolic/congenital – hypoglycemia, hypothyroidism, congenital syndromes, metabolic disorders
- Trauma – delivery-related (ie. brachial plexus injury), non-accidental trauma

Treatment

Routine care for all newborns:

- Dry baby with clean towel, replace wet cloths, place the baby skin-to-skin with mother as soon as possible
- Cover the baby to prevent heat loss (particularly the head)
- Encourage initiation of breastfeeding within the first hour, let baby feed on demand if able to suck
- Give vitamin K injection IM, antiseptic ointment or antibiotic eye drops to both eyes soon after delivery

Pediatric Presentations cont'd

Emergency Management of Seriously Ill Newborn

- O₂ via nasal prongs or nasal catheter
- Bag-mask ventilation with oxygen (if available), consider CPAP (if available)
- Obtain IV access, start ampicillin and gentamicin (or available broad spectrum antibiotics by local protocol) and maintenance fluids (replacement as necessary typically with dextrose containing fluid, usually D10W)
- If drowsy, unconscious or convulsing: check capillary blood glucose
 - Glucose <1.1 mmol/L (<19.82 mg/dL) – give glucose IV immediately
 - Glucose 1.1 - 2.2 mmol/L (19.82- 39.64 mg/dL) – feed immediately, increase feed frequency, recheck in 2-6 hours
 - Glucose check unavailable – assume hypoglycemia, give dextrose by IV
- Work-up (as available) – full septic work-up (CBC, blood culture, urine culture, CSF analysis, CXR), oxygen saturations

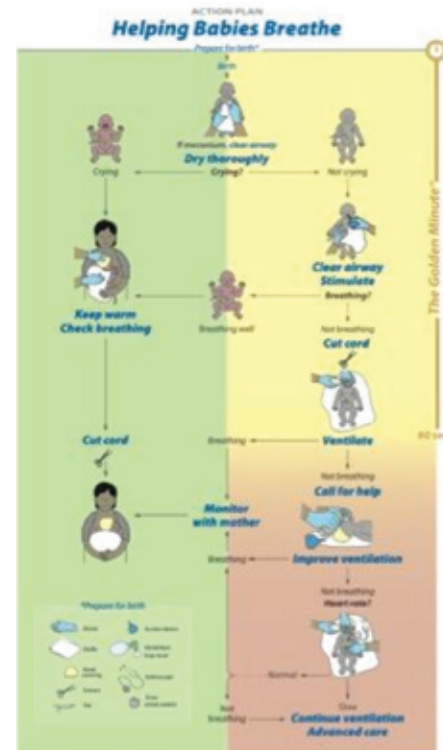


Figure 9-2: Helping Babies Breathe—American Academy of Pediatrics program for low-income settings

Supportive Care for Sick Neonate

- Thermal environment – keep infant dry and well wrapped, use bonnet or cap to reduce heat loss, keep room at least 25°C, ensure infant is maintaining core temperature.
 - Kangaroo mother care has equal efficacy to incubator or external heating device.
- Encourage breast feeding if available; withhold oral feeds in bowel obstruction, necrotizing enterocolitis, acute phase of lethargy or unconscious infant.
 - Increased fluids if infant under a radiant heater.
 - Monitor rate of IV fluids very closely.

Care of Infants >28 Days and Older Children

General Information

- Children get many of the same infections and disease processes as adults described in the previous chapters...but they are NOT just little adults! Unique physiology and risk factors makes them more susceptible to certain infections (respiratory, AOM, UTI).
- Common: malaria, respiratory infections, diarrheal diseases, TB, HIV.
- Don't forget about relatively rarer neglected infections, especially those known to be prevalent in the community you are working: Dengue, protozoal infections, strongyloidiasis etc.
- Malnutrition is a serious, underlying contributor to childhood illness and mortality in low-income countries.

Pediatric Presentations cont'd

Table 9-1: Normal vital signs by age in children¹¹

Age	Heart Rate (beats/min)	Blood Pressure (mm Hg)	Respiratory Rate (breaths/min)
Premature	120-170	55-75/35-45	40-70
0-3 mo	100-150	65-85/45-55	35-55
3-6 mo	90-120	70-90/50-65	30-45
6-12 mo	80-120	80-100/55-65	25-40
1-3 yr	70-110	90-105/55-70	20-30
3-6 yr	65-110	95-110/60-75	20-25
6-12 yr	60-95	100-120/60/75	14-22
12+ yr	55-85	110-135/65/85	12-18

Remember that children compensate their blood pressure better than adults, so a low BP is a very late, serious sign of shock in a child.

Emergency Management of Children

IV Fluids for Shock:

- Infuse 20 ml/kg as quickly as possible of normal saline or Ringer's lactate.
- Reassess after first infusion; if no improvement, repeat 20 ml/kg as rapidly as possible.

IV Glucose for Hypoglycemia:

- Check blood glucose; give infusion if < 2.5 mmol/L (< 45 mg/dL) in well nourished or < 3 mmol/L (< 54 mg/dL) in malnourished child, or if rapid testing not available.
- Give 5 mL/kg D10W IV push (or 2 mL/kg D25W IV).
- Re-check in 30 minutes; if still low, repeat 5 mL/kg D10W IV push.

Dehydration and fluids in children

WHO guidelines in LMIC typically teach to first attempt to use oral rehydration solution (ORS) in the setting of dehydration. Second they suggest IV fluids with deficit replacement for moderate dehydration. Third line is resuscitation by giving fluids every four hours as many locations won't have IV pumps. This differs from traditional teaching in Canada and the US where resources are more plentiful.

Replacement fluids: 4-2-1 rule

- Give 4 ml/hr for each of first 10 kg, 2 ml/hr for each of next 10 kg, then 1 ml/hr for every kg over 20
- E.g. 31 kg child requirements = $(4 \times 10) + (2 \times 10) + (1 \times 1) = 71$ ml/hr solution of choice

Table 9-2: Estimating amount of fluid loss as a percentage of body weight in children²

	Mild	Moderate	Severe
Infant	5%	10%	15%
Child	3%	6%	9%

Approach to replacement of fluids in the severely dehydrated child:

1. If the child is acutely ill/septic – resuscitate with fluid bolus first!
2. Assess degree of dehydration (see chart in Malnutrition chapter).
3. Calculate approximate volume of fluid loss based on child's body weight.
4. Choose best fluid based on child's age and presenting illness, type of dehydration (generally isotonic solutions like D5NS are often preferred in children with significant illness).
5. Replace 50% of fluid over 8 hrs, then the remaining 50% of fluid over the next 16 hrs.
6. Add maintenance fluids (4-2-1) to replacement fluids.
7. Replace ongoing losses – vomiting, diarrhea etc.

For children using Oral Rehydration Solution (ORS) rehydration – aim for volume of 5 ml/kg/hour.

Pediatric Presentations cont'd

Malnutrition in children

Malnutrition is a critical contributor to childhood illness and underlies more than 40% of the childhood mortality in low-income settings (UN World Food Program - Hunger Stats).

Appropriate management of malnutrition requires much more than refeeding; must consider hypoglycemia, hypothermia, and high risk of infection, as well as electrolyte balance.

Figure 9-3: WHO approach to management of a child with malnutrition²

	Stabilization		Rehabilitation	
	Days 1–2	Days 3–7	Weeks 2–6	
1. Hypoglycaemia		→		
2. Hypothermia		→		
3. Dehydration		→		
4. Electrolytes		→	→	→
5. Infection		→	→	
6. Micronutrients	no iron	→	→	with iron →
7. Initiate feeding		→	→	
8. Catch-up growth				→
9. Sensory stimulation		→	→	→
10. Prepare for follow-up				→

Burns

- Very common in many low-income countries where cooking is done in the home over open flame
- Note that the “rule of 9s” used in adult burns does not apply to children; relative surface area changes based on development; can use a specific BSA chart or estimate based on the child’s palm being approximately 1% of total body surface area
- Full thickness burns: black or white, usually dry, no feeling, non-blanchable; partial thickness burns: pink or red, blistering or weeping, painful

Basic management of burns in children:

1. **Admit:** burns > 10% Body Surface Area (BSA), burns involving face, hands, feet, perineum, joints, burns that are circumferential and those that cannot be managed as an outpatient.
2. **Ensure ABCs** (particular focus on A—smoke inhalation, severe facial burn requiring intubation) .
3. **Fluids:** Usually Ringers Lactate is used where available. Resuscitate with maintenance, adding 4 mL/kg for every 1% of surface burned, give fluids ½ over first 8 hours, ½ over remaining 16 hours , monitor vitals and urine output.
4. **Prevent infection:** clean intact skin, debride skin that is not intact, use what ever topical antibiotic/antiseptic is available (silver nitrate, silver sulfadiazine, gentian violet, betadine, mashed papaya); clean and dress wound daily.
5. **Treat secondary infection if present:** oral amoxicillin 15 mg/kg PO and cloxacillin.
6. **Pain control:** paracetamol (acetaminophen) 15 mg q6H PO or IV analgesics such as morphine for severe pain.
7. **Nutrition:** high calorie diet as soon as possible in first 24 hours, ensure adequate protein, vitamin and iron (children with extensive burns require 1.5 x calories and 2-3 x protein).

Convulsions

- Common presenting symptom in seriously ill child—cerebral malaria, meningitis, secondary to hypoglycemia or febrile seizures (in young children 6 months to 6 years).
- Manage airway first.
- Give available anticonvulsant (diazepam or lorazepam, phenobarbital).
- Give IV glucose 5 mL/kg D10W.
- Treat underlying cause.

Pediatric Presentations cont'd

Prevention of Mother-to-Child Transmission of HIV (PMTCT)

Transmission of HIV from mother to baby can occur during pregnancy, labour and delivery, or through breastfeeding. In order to make decisions about PMTCT, you must know a woman's HIV status – important to encourage voluntary HIV testing in all pregnant women.

Two key approaches to prevention:

1. Lifelong antiretroviral therapy (ART) – for HIV infected women in need to treatment for their own health, which is also safe and effective in reducing MTCT
 - Eligible = WHO clinical stage 3 or 4, or CD4 cell count <350 cells/mm3

Table 9-3: Maternal ART and infant ARV prophylaxis⁴

Maternal ART + infant ARV prophylaxis	
Mother	
Maternal antepartum daily ART, starting as soon as possible irrespective of gestational age, and continued during pregnancy, delivery and thereafter. Recommended regimens include: AZT + 3TC + NVP or AZT + 3TC + EFV* or TDF + 3TC (or FTC) + NVP or TDF + 3TC (or TFC) + EFV*	
Infant	
Daily NVP or twice-daily AZT from birth until 4–6 weeks of age (irrespective of the model of infant feeding).	

2. Antiretroviral prophylaxis – to prevent MTCT during pregnancy, delivery, and breastfeeding for HIV-infected women not in need of treatment

Considerations for delivery:

- C-section desirable if possible
- If unable to perform C-section, ensure mother receives ART during delivery

Feeding the newborn of HIV-positive mother:

Best Option: Formula feeding only (no risk of virus transmission)

Second Option: Breastfeeding only (mothers should be on ART while breastfeeding)

Worst Option: Mix of breast and formula feeding (formula can irritate GI tract decreasing barriers and allowing HIV virus to enter infant bloodstream)

Table 9-4: Options for ARV prophylaxis⁴

Maternal ART + infant ARV prophylaxis (Option A)		Maternal triple ART (OptionB)
Mother		Mother
Antepartum twice-daily, AZT starting from as early as 14 weeks of gestation and continuing during pregnancy. At onset of labour, sd-NVP and initiation of twice daily AZT + 3TC for 7 days postpartum. (Note: If maternal AZT was provided for more than 4 weeks antenatally, omission of the sd-NVP and AZT + 3TC tail can be considered; in this case, continue maternal AZT during labour and stop at delivery.)		Triple ARV prophylaxis starting from as early as 14 weeks of gestation and continue until delivery, or, if breastfeeding, continued until 1 week after all infant exposure to breast milk has ended. Recommended regimens include: AZT + 3TC + LPV or AZT + 3TC + ABC or TDF + 3TC EFV or TDF + 3TC (or TFC) + EFV
Infant		Infant
<i>For breastfeeding infants</i>	<i>Infants receiving replacement feeding only</i>	<i>Irrespective of mode of infant feeding</i>
Daily NVP from birth for a minimum of 4–6 weeks, and until 1 week after all exposure to breast milk has ended.	Daily NVP from birth for a minimum of 4–6 weeks, and until 1 week after all exposure to breast milk has ended.	Daily NVP or twice-daily AZT from birth until 4–6 weeks of age.

Pediatric Presentations cont'd

Books and PDFs to Bring Along on an International Pediatrics Elective

- WHO Pocket Book of Hospital Care for Children
- Oxford Handbook of Tropical Medicine
- WHO Clinical Guidelines – HIV, TB, Malaria

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Trauma

Paul Jones

General Information

Road traffic accidents are the tenth leading cause of death in low income countries according to the World Health Organization (WHO). A recent study found that 32% of fatal injuries were traffic accident related in Sierra Leone.¹ They found that falls were the most common nonfatal injuries and that injuries were most likely to extremities (70%) and face, head and neck (25%).¹

Burns are also a common injury which often require medical attention. The most common causes of burn injury were contact with a hot liquid or object, and the most common body region affected was the upper extremities, followed by burns to the head/face/neck. About 60% of burns affect individuals above 18 years and the victims are more frequently male.^{1,11}

The third most common body region affected by trauma is the abdomen.² It is also more commonly affected by penetrating injuries such as stab wounds.³ Animal bites and gunshot wounds are relatively uncommon in most locations, but gunshot wounds are more common in places experiencing armed conflict or have high prevalence of narco-trafficking and gang activity.

General Approach

Advanced Trauma Life Support (ATLS)⁶ teaches three general principles when caring for traumatically injured patients:

1. Treat the greatest threat to life first.
2. The lack of a definitive diagnosis should never impede the application of an indicated treatment.
3. A detailed history is not essential to begin the evaluation of a patient with acute injuries.

They recommend the “ABCDE” mnemonic to order your evaluation and interventions:

- Airway with cervical spine protection
- Breathing
- Circulation, stop the bleeding
- Disability or neurologic status
- Exposure (undress) and Environment (temperature control)

Clinical Features

Hemorrhage (external and internal) and burns are both at risk of hypovolemic shock. Early signs of shock include tachycardia, tachypnea, decreased capillary refill and cool extremities. Later signs can include hypotension, altered level of consciousness and decreased urine output. Most bleeding can be controlled with well aimed direct pressure (Dr. David Johnson, Medical Director of Wilderness Medical Associates). In extreme cases such as traumatic amputations, tourniquets have been shown to be effective.

ATLS Approach⁶

- Preparation
- Triage
- Primary Survey (ABCDEs)
- Resuscitation
- Adjuncts to primary survey and resuscitation
- Consider need for patient transfer
- Secondary survey (head-to-toe evaluation and patient AMPLE history)
- Adjuncts to the secondary survey
- Continue postresuscitation monitoring and reevaluation
- Definitive Care

Trauma cont'd

When diagnostic tests are limited you must rely on your physical exam and clinical judgement. In order to assess for shock you need to make full use of the available information. You must consider pulse, respirations, blood pressure, level of consciousness (LOC) and temperature. If a blood pressure cuff is unavailable you can crudely estimate systolic blood pressure based on the most distally palpable pulse (Radial >80 mmHg; Femoral >70 mmHg; Carotid > 60 mmHg). LOC, extremity colour, temperature and capillary refill are all sensitive indicators of perfusion. LOC can be recorded with either the crude AVPU system or the more precise Glasgow Coma Scale (GCS).

AVPU

- Alert
- Responds to verbal stimuli
- Responds to painful stimuli
- Unresponsive

Glasgow Coma Scale (GCS)

- Eyes Open (Spontaneous = 4; To Voice = 3; To pain = 2; No response = 1)
- Best Verbal (Answers questions appropriately = 5; Confused, disoriented = 4; Inappropriate words = 3; Incomprehensible sounds = 2; No verbal response = 1)
- Best Motor (Obeys commands = 6; Localizes to pain = 5; Withdraws from pain = 4; Decorticate (flexion) = 3; Decerebrate (extension) = 2; No response = 1)

(GCS = 13-15 mild injury; GCS = 9-12 moderate injury GCS = <= 8 severe injury “Less than 8, intubate”)

In the ATLS approach to patients with traumatic injuries Airway is the first step in the algorithm while simultaneously protecting the cervical spine. The American College of Surgeons Committee on Trauma says, “Assume a cervical spine injury in any patient with multisystem trauma, especially those with an altered level of consciousness or blunt injury above the clavicle.” So how do you clear a C-spine or decide who to image? You can make use of the Canadian C-Spine Rule in alert patient with a GCS of 15.⁵ High risk features necessitate imaging to clear the cervical spine, these include: age > 65, dangerous mechanism* and paresthesia in extremities.

Dangerous Mechanism = Need imaging

- Fall from elevation > 3 feet/ 5 stairs
- Axial load to head e.g. diving
- MVC high speed (>100 km/hr), rollover, ejection
- Motorized Recreational Vehicles
- Bicycle Collision

Low risk features listed below allow for safe clinical assessment of range of motion.

- Simple rear-end MVC* (excludes pushed into oncoming traffic, hit by bus/truck, rollover, hit by high speed vehicle)
- Sitting position in ED
- Ambulatory at any time
- Delayed onset of neck pain
- Absence of midline c-spine tenderness

If the patient is able to actively rotate the neck 45 degrees each way then imaging is not required.

For extremity injuries it is recommended that you check distal circulation, sensation, as well as normal joint movement. Extremity injuries can be broadly classified as stable or unstable. For dislocated or grossly deformed extremities or digits you can utilize a simple approach of “hand stable, splint stable, cast stable”. Always document circulation and sensation pre and post reduction. Open fractures will require antibiotics, tetanus vaccination and surgical management for irrigation and debridement.⁴

Trauma cont'd

AMPLE History

- Allergies
- Medications currently used
- Past Illness/Pregnancy
- Last Meal
- Events/Environment related to the injury

Investigations

- Serial Physical Examinations * In some context this may be the only investigative tool you have.
- Xrays (C-spine, chest, pelvis for blunt trauma)
- ECG
- CBC/INR/PTT/Cross and Type/BetaHCG/Lytes/Cr/BUN/Glucose/Tox Screen
- FAST Ultrasound/CT/DPL/Laparotomy* These are advanced modalities most medical students and residents will not be able to perform or accurately interpret.

Differential Diagnosis

Shock: inadequate organ and tissue perfusion with oxygenated blood (brain, kidney, extremities - check LOC, urine output and peripheral pulses/limb temperature)

Head Injury: decreased LOC, deteriorating resp pattern, Cushing reflex (high BP, low HR, irregular resp) lateralizing CNS signs, seizure, papilledema (late), nausea, vomiting, headache

Tension Pneumothorax: resp distress, tachycardia, distended neck veins, cyanosis, asymmetry of chest wall motion, tracheal deviation away from pneumothorax, hyperresonance on percussion, unilateral absence of breath sounds

Open Pneumothorax: gunshot wound or other penetrating wound, unequal breath sounds

Hemothorax: pallor, flat neck veins, shock, hypotension, absent breath sounds, hypotension, unilateral dullness

Cardiac Tamponade: penetrating wounds (usually), Beck's triad (hypotension, distended neck veins, muffled heart sounds) tachycardia, tachypnea

Pelvic Fracture: localized swelling, tenderness, deformity of lower extremity, pelvic instability, hypotension

Compartment Syndrome: pulse discrepancies, pallor, paresthesia, paralysis, pain out of proportion, polar (cold)

(Above adapted from references: ^{6, 7, 8})

Treatment

Airway/Breathing/C-Spine Stabilization/Circulation/Oxygenation/Monitor Vitals/Control Hemorrhage/Give Fluids/Pain Control/Definitive Care.

*Foley Contraindication—blood at urethral meatus, scrotal hematoma, high riding prostate on DRE

*NG Tube Contraindication—Significant midface trauma or basal skull fracture

In trauma associated hemorrhage consider the role of tranexamic acid. The CRASH-2 trial has shown that TXA (Tranexamic acid) reduces mortality from hemorrhage by about one sixth.⁹ If this widely practicable intervention was used worldwide in the treatment of bleeding trauma patients, it could prevent over 70,000 deaths each year.¹⁰ The study protocol was Tranexamic acid 1000 mg IV over 10 minutes and then 1000 mg IV over the next 8 hours.⁹

Trauma cont'd

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Indigenous Populations Anastasia Blake

General Information

There are some 370 million Indigenous peoples worldwide. Despite a myriad of cultures, Indigenous peoples share many challenges that have contributed to significant health disparities when compared with their non-Indigenous counterparts. In both poor and industrialized nations, Indigenous peoples are over-represented in the lower socio-economic class. Determinants of health for Indigenous populations include: poverty, malnutrition, overcrowding, poor sanitation, environmental contamination, climate change, limited access to health care resources, effects of colonization, and changing from traditional to modern lifestyles. As a result of these inequities, Indigenous populations have higher infant and child mortality rates, higher maternal morbidity and mortality rates, heavy infectious disease burdens, shorter life expectancies, higher rates of tobacco/alcohol/drug use, increased risk of interpersonal violence/homicide/suicide, increased prevalence of diseases caused by environmental contamination, and increased prevalence of “lifestyle diseases” including diabetes, obesity, hypertension, cardiovascular disease and chronic renal disease.

In Canada, Indigenous peoples recognized by the government include First Nations, Inuit and Métis, yet there are many other Indigenous groups that do not fall into one of these categories. According to the 2001 national census, Aboriginal peoples make up 4.4% of the total Canadian population; however it is likely that these numbers underestimate the actual size of the Aboriginal population as many First Nations on-reserve communities did not participate in the census. The Aboriginal population is also growing at a rate much higher than the rest of Canada, with the mean age of Aboriginal peoples being 24.7 years compared to 33.7 years for the non-Aboriginal population. As J. Reading (2012), Director at the Centre for Aboriginal Health Research at the University of Victoria notes, “It is no secret that Aboriginal peoples in Canada, no matter where they live, face unique health challenges. They experience higher rates of diabetes, heart disease, tuberculosis, HIV/AIDS and many other diseases. Infant mortality rates are higher and life expectancy is lower than in the general population. The Aboriginal suicide rate is two-to-three times higher than the non-Aboriginal rate for Canada and, within the youth age group, this rate is estimated to be five-to-six times higher.”

In 2012, the Health Council of Canada highlighted another important factor that has negatively affected the health of Aboriginal populations: “many Aboriginal people don’t trust – and therefore don’t use – mainstream health care services because they don’t feel safe from stereotyping and racism, and because the Western approach to health care can feel alienating and intimidating”. This mistrust of the health care system is founded on a long and painful history of colonization and forced cultural assimilation, and has contributed to advanced diseases with poorer outcomes as well as missed opportunities for preventative care and screening.

General Approach

The determinants of health discussed in the previous section highlight many areas where health care providers can work to address the health disparities faced by Indigenous populations such as advocating for improved access to basic sanitation, adequate housing, education, healthcare and food security. Perhaps an even more important task that health care providers can take part in is helping to create an environment that is perceived to be free of bias and fosters the trust of Aboriginal patients. Cultural competency and cultural safety are two concepts that have been developed to aid those working with Indigenous populations in this task.

Cultural competency can be viewed as the act of creating a healthcare environment free of racism and stereotypes. With respect to Aboriginal patients, this includes recognizing the intergenerational effects of colonization and cultural assimilation and adapting care to reflect the distinct needs of this population. Cultural competency involves seeking to understand what Aboriginal culture means to the patient and what unique needs they may have as a result and advocating to ensure those needs are met. Furthermore, it involves consideration of Indigenous perspectives on the meaning of health, including the balance between mind, body, spirit, and emotion, between individuals within communities, and between communities and the natural environment, and incorporating those views into an approach to health care. Lastly, cultural competency emphasizes the importance of collaboration with Aboriginal communities in making decisions that affect them.

Cultural safety applies this knowledge of the role that social, historical and political factors have played in determining the health of Indigenous peoples to the intrinsic power differentials of the mainstream health care system. “Healthcare providers need to be aware that the impact of the long history of discrimination and racism directed at First Nations, Inuit, and Métis people is still felt today. Aboriginal people – particularly those who were affected by the residential

Indigenous Populations cont'd

school experience – may have a heightened sensitivity to practices that are a routine part of hospital life.” (Health Care Council, 2012). For example, complicated medical jargon, short appointment times, emergency triaging processes, and “Western” medical treatments may all be perceived as discriminating against Aboriginal people, even when they are not. Lastly, and most importantly, cultural safety is defined by the recipient of the health care services; it occurs when Aboriginal people report feeling safe using the health care system.

Treatment and Prevention

Utilizing the framework described above, many interventions have been implemented to improve the health of Indigenous populations at the international, national, community and individual levels. International efforts such as the WHO’s International Decade of the World’s Indigenous Peoples, the Global Indigenous Health program at the University of Victoria, and the International Network of Indigenous Health Knowledge and Development have allowed countries to share Indigenous health research and strategies to address common problems. In Canada, collaboration and partnership between First Nations, Inuit and Métis organizations has led to the development of many unique programs that, due to jurisdictional constraints, could not otherwise exist. Furthermore, multiple programs have been developed to increase the cultural awareness and competency of health care workers including: an online Indigenous Cultural Competency Training Program for health care workers in British Columbia; a First Nations, Inuit and Métis Health Core Competencies Curriculum Framework for Undergraduate Medical Education in Canada; and an action-based guide to cultural competency by the Society of Obstetricians and Gynecologists of Canada.

Many other key areas have been identified where targeted approaches could significantly improve the health of Indigenous peoples globally. These include:

Health of Mothers and Children:

- Provision of adequate prenatal care and safe birthing facilities
- Access to family planning methods
- Reduction in high risk behaviours in pregnancy including smoking, alcohol use, recreational drug use
- Encouragement of breastfeeding, nutritious weaning practices, dental hygiene
- Routine monitoring of child growth and development

Nutritional Deficiencies:

- Access to nutritious and affordable food
- Provision of nutritional supplements (ex. folic acid, iron)

Infectious Diseases:

- Ensuring access to adequate housing, clean water, basic sanitation, waste removal
- Implementation of immunization programs
- Early diagnosis and treatment of infectious diseases

Addiction:

- Access to treatment programs for drug and alcohol abuse
- Encouragement of smoking cessation and provision of culturally appropriate resources

Urbanization/Lifestyle Diseases:

- Screening for lifestyle diseases
- Encouragement of healthy food choices
- Engagement of Indigenous communities in creating treatment programs and community-based wellness programs (Adapted from Gracey, 2009)

Indigenous Populations cont'd

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Ethics

Paul Jones

“Primum non nocere.” – “First, do no harm.”

–Anonymous

Much has been written about biomedical ethics, often in dense philosophical writings. By now you have probably become familiar with the Georgetown Mantra of: beneficence, non-maleficence, autonomy and justice.¹ The field of biomedical ethics often talks about ethical theories on the “macro-scale” and ethical dilemmas on the “micro-scale”. To the common individual, little thought is typically given to the “macro-scale” philosophical theories which underpin professional codes of conduct and laws.

Travelling to developing countries is often a great applied introduction to ethics on both a micro and macro level. You can be confronted with questions of rights, justice, non-maleficence and resource allocation, to name just a few.

If you haven’t already, think about these questions:

- Who should have access to health care?
- Who should pay for it?
- What is the role of government in health care?
- How can a patient provide informed consent when communicating across language and cultural barriers, through the assistance of interpreters?
- How do we ensure basic medical/surgical care and access to essential life saving medicines?
- What if there isn’t enough oxygen or power for an entire surgical case?
- Who shall live?

Resource Allocation

In low resourced settings, rationing of scarce resources is a daily reality, which health care professionals and patients alike must confront. Questions are raised about equity and egalitarianism. Often health professionals default to a purely utilitarian perspective seeking to provide the most good to the greatest number, invoking some form of triage. The concept of triage originates from the French word *trier* meaning to separate out and in medical use typically means to assign degrees of urgency to patients with differing illnesses or injuries. Fundamentally, triage questions raise concerns about justice, impartiality and fairness. Countering the utilitarian perspective is philosopher John Rawls who proposes a differing theory of justice with what he called the “difference principle”.¹ This requires preferential treatment for the most disadvantaged of individuals, regardless of the social costs this principle can entail. In impoverished settings or times of conflict, rationing can be extreme, forcing clinicians to confront questions of who shall live, and where finite medical resources should be directed. For example:

- Is it just to declare medical intervention for certain patients “futile” or to not perform a resuscitation, as doing so would tie up precious resources, which could be redirected to care for greater numbers of individuals who have a higher chance of survival?
- How do we address fundamental questions of inequality and ensure that we conduct ourselves in an ethical manner?
- How do we cope with barriers to access to life sustaining medicines or surgical procedures?

By engaging in these tough questions while maintaining civility, respect and the dignity of the patient, we can attempt to do the most good.

Supervision

A critical component of any good medical elective is appropriate and effective supervision since “inadequately supervised students risk doing more harm than good”.

Medical schools have an obligation to their students and communities where their students work, to ensure that no student undertakes an elective in a low-resource setting with the purpose of being allowed to practice beyond that which would be acceptable at their home institution, based on their level of training.

Ethics cont'd

(Preparing Medical Students for Electives in Low-Resource Settings)

Part of being accountable as a medical trainee is ensuring that you have adequate knowledge, skill and ability to perform the tasks your supervisor sets before you. If you feel unable or unwilling to do something expected of you on your medical elective such as an invasive procedure, it is imperative that you voice this concern to your direct supervisor. If this does not adequately resolve things, you can also contact the Undergraduate International Electives Office or Chair of Global Health in your respective department or faculty.

Informed consent is central to the medical encounter. It is important that medical trainees wear proper identification, introduce themselves and ask permission to conduct any history or physical examination. When communicating across language barriers, interpreters may be required. For consent to be informed, the patient must have knowledge of the possible consequences of action or inaction. They must know and understand the risks and benefits of the planned care or procedure. Patients must always have the right to refuse care.

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Appendix

i) Normal Laboratory Values

Normal Lab Values

Hemoglobin	Creatinine 0.13–0.22 mmol per kilogram body weight, daily
Male 13.5–17.7 g/dL	5-hydroxyindole acetic acid (5HIAA)
Female 11.5–16.5 g/dL	<75 µmol daily; amounts lower in females than males
Mean corpuscular haemoglobin (MCH)	Protein (quantitative) <0.15 g per 24 hours
27–32 pg	Sodium 60–80 mmol per 24 hours
Mean corpuscular hemoglobin concentration (MCHC) 32–36 g/dL	
Mean corpuscular volume (MCV) 80–96 fL	
Packed cell volume (PCV)	
Male 0.40–0.54 L/L	
Female 0.37–0.47 L/L	
White blood count (WBC) 4–11 10 ⁹ /L	
Basophil granulocytes <0.01–0.1 10 ⁹ /L	
Eosinophil granulocytes 0.04–0.4 10 ⁹ /L	
Lymphocytes 1.5–4.0 10 ⁹ /L	
Monocytes	
0.2–0.8 10 ⁹ /L	
Neutrophil granulocytes 2.0–7.5 10 ⁹ /L	
Total blood volume 60–80 ml/kg	
Plasma volume 40–50 ml/kg	
Platelet count 150–400 10 ⁹ /L	
Serum B12 160–925 ng/L (150–675 pmol/L)	
Serum folate 2.9–18 µg/L (3.6–63 nmol/L)	
Red cell folate 149–640 µg/L	
Red cell mass	
Male 25–35 ml/kg	
Female 20–30 ml/kg	
Reticulocyte count 0.5–2.5% of red cells (50–100 10 ⁹ /L)	
Erythrocyte sedimentation rate (ESR)	
<20 mm in 1 hour	
Plasma viscosity 1.5–1.72 mPa.s	
Coagulation	
Bleeding time (Ivy method) 3–9 min	
Activated partial thromboplastin time (APTT)	
23–31 s	
Prothrombin time 12–16 s	
International Normalized Ratio (INR)	
1.0–1.3	
D-dimer <500 ng/ml	
Blood gases (arterial)	
PaCO ₂ 4.8–6.1 kPa (36–46 mmHg)	
PaO ₂ 10–13.3 kPa (75–100 mmHg)	
[H ⁺] 35–45 nmol/L	
pH 7.35–7.45	
Bicarbonate 22–26 mmol/L	
Urine values	
Calcium 7.5 mmol daily or less (<300 mg daily)	
Copper 0.2–1.0 µmol daily	

Appendix cont'd

Biochemistry (serum/plasma)

Alanine aminotransferase (ALT) 5–40 U/L	Female 7–32 U/L
Albumin 35–50 g/L	Glycosylated (glycated) haemoglobin (HbA1c) 3.7–5.1%
Alkaline phosphatase 39–117 U/L	Hydroxybutyric dehydrogenase (HBD) 72–182 U/L
Amylase 25–125 U/L	Immunoglobulins (11 years and over)
Angiotensin-converting enzyme 10–70 U/L	IgA 0.8–4 g/L
1-antitrypsin 1.1–2.1 g/L	IgG 5.5–16.5 g/L
Aspartate aminotransferase (AST) 12–40 U/L	IgM 0.4–2.0 g/L
Bicarbonate 22–30 mmol/L	Iron 13–32 µmol/L (50–150 µg/dL)
Bilirubin <17 µmol/L (0.3–1.5 mg/dL)	Iron binding capacity (total) (TIBC) 42–80 µmol/L (250–410 µg/dL)
Caeruloplasmin 0.20–0.61/L	Lactate dehydrogenase 240–480 U/L
Calcium 2.20–2.67 mmol/L (8.5–10.5 mg/dL)	Magnesium 0.7–1.1 mmol/L
Chloride 98–106 mmol/L	
Complement	
C3 0.75–1.65 g/L	
C4 0.20–0.60 g/L	
Copper 11–20 µmol/L (100–200 mg/dL)	
C-reactive protein <10 mg/L	
Creatinine 79–118 µmol/L (0.6–1.5 mg/dL)	
Creatine kinase (CPK)	
Female 24–170 U/L	
Male 24–195 U/L	
CK-MB fraction <25 U/L (<60% of total activity)	
Ferritin	
Female	
6–110 µg/L	
Male	
20–260 µg/L	
Post menopausal 12–230 µg/L	
-fetoprotein <10 k U/L	
Glucose (fasting) 4.5–5.6 mmol/L (70–110 mg/dL)	
Fructosamine up to 285 µmol/L	
-glutamyl transpeptidase (-GT)	
Male 11–58 U/L	

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