The MDM DIAGNOSIS AND TREATMENT MANUAL represents the views of the MDM and was produced after careful consideration of the scientific and medical knowledge and the evidence available at the time of their publication. The MDM is not responsible in the event of any contradiction, discrepancy and/or ambiguity between the MDM DIAGNOSIS AND TREATMENT MANUAL and any other official recommendations or guidelines issued by the relevant public health authorities, in particular in relation to good use of healthcare or therapeutic strategies. THE MDM Health professionals are encouraged to take the MDM DIAGNOSIS AND TREATMENT MANUAL fully into account when exercising their clinical judgment, as well as in the determination and the implementation of preventive, diagnostic or therapeutic medical strategies; however, the MDM DIAGNOSIS AND TREATMENT MANUAL do not override, in any way whatsoever, the individual responsibility of health professionals to make appropriate and accurate decisions in consideration of each patient’s health condition and in consultation with that patient and, where appropriate and/or necessary, the patient’s caregiver. Nor do the MDM DIAGNOSIS AND TREATMENT MANUAL exempt health professionals from taking into full and careful consideration the relevant official updated recommendations or guidelines issued by the competent public health authorities, in order to manage each patient’s case in light of the scientifically accepted data pursuant to their respective ethical and professional obligations. It is also the health professional’s responsibility to verify the applicable rules and regulations relating to drugs and medical devices at the time of prescription.

This manual constitutes a bibliographic review.
DOCTORS OF THE WORLD

Diagnosis and treatment manual
Protocol Key:

- Specific Protocol
- EMT/EMT-B Licensed Attendant and above may perform these steps
- AEMT/EMT-I, Paramedic/EMT-P Licensed Attendant and above may perform these steps
- Paramedic/MD

Definition of a patient:

A patient is any individual that meets at least one of the following criteria:

1) A person who has a complaint or mechanism suggestive of potential illness or injury;

2) A person who has obvious evidence of illness or injury; or

3) A person identified by an informed 2nd or 3rd party caller as requiring evaluation for potential illness or injury.

Pediatric patient considerations:

For patients <18 years old, use the Pediatric Patient Destination protocol.

Pediatric treatment protocols are to be used on children who have not yet experienced puberty. Signs of puberty include chest or underarm hair on males, and any breast development in females.
TERMS AND CONVENTIONS

ACE Angiotensin-converting enzyme
AED means Automated External Defibrillator
AMS means Altered Mental Status
ANA Antinuclear antibody
AOM Acute otitis media
ARBs angiotensin II receptor blockers
ASA means Acetylsalicylic Acid
BDP Beclometasone dipropionate
BG means Blood Glucose
BP means Blood Pressure
BUD Budesonide
CCC means Continuous Cardiac Compressions
CHF means Congestive Heart Failure
CBC Complete blood count
COPD means Chronic Obstructive Pulmonary Disease
CN cranial nerve
CNS central nervous system
CP means Chest Pain
CPR means Cardiopulmonary Resuscitation
CVA means Cardiovascular Accident
DM Diabetes mellitus
GDM Gestational diabetes mellitus
DPI Dry powder inhaler
ECG means Electrocardiogram
ED emergency department
EMS emergency medical system
ETA means Estimated Time of Arrival
FEV1 Forced expiratory volume in 1 second
FVC Forced vital capacity
GDM Gestational diabetes mellitus
HEENT means Head, Ears, Eyes, Nose, Throat
Hib Haemophilus influenzae type b
HR means Heart Rate
HSV herpes simplex virus
ICS Inhaled corticosteroids
IM means Intramuscular
IV means Intravenous
IVP means Intravenous Push
LABA Long-acting beta2-agonists
MEE middle ear effusion
MI means Myocardial Infarction
NS means Normal Saline
NSAID nonsteroidal anti-inflammatory drug
NV means Nausea/Vomiting
O2 Oxygen
OCS Oral corticosteroids
OGTT Oral glucose tolerance test
OM Otitis media
OME Otitis media with effusion
ORT oral rehydration therapy
PCI means Percutaneous Coronary Intervention
PCR means Patient Care Record/Report
PEF Peak expiratory flow
PEM protein-energy malnutrition
pMDI Pressurized metered dose inhaler
PO means By Mouth
PRN means As Needed
q means Every
ROSC means Return of Spontaneous Circulation
RR means Respiratory Rate
SABA Short-acting beta2-agonists
SBI serious bacterial infections
SE Status epilepticus
SL means Sublingual
SOB means Shortness of Breath S/S means Signs/Symptoms
SVT means Supraventricular Tachycardia
TBSA total body surface area
TM tympanic membrane
URI Upper respiratory tract infection
UTI urinary tract infection
VF means Ventricular Fibrillation
VS means Vital Signs
VT means Ventricular Tachycardia
WPW means Wolff-Parkinson-White Syndrome
SHOCK

Shock is a medical emergency in which the organs and tissues of the body are not receiving an adequate flow of blood. This deprives the organs and tissues of oxygen (carried in the blood) and allows the buildup of waste products. Shock can result in serious damage or even death.

Aetiology and pathophysiology

Distributive shock results from excessive vasodilation and the impaired distribution of blood flow. Septic shock is the most common form of distributive shock and is characterized by considerable mortality (treated, around 30%; untreated, probably >80%).

Other causes of distributive shock include systemic inflammatory response syndrome (SIRS) due to noninfectious inflammatory conditions such as burns and pancreatitis; toxic shock syndrome (TSS); anaphylaxis; reactions to drugs or toxins, including insect bites, transfusion reaction, and heavy metal poisoning; addisonian crisis; hepatic insufficiency; and neurogenic shock due to brain or spinal cord injury.

Types of shock

- Distributive shock (vasodilation), which is a hyperdynamic process
- Cardiogenic shock (pump failure)
- Hypovolemic shock (intravascular volume loss)
- Obstructive shock (physical obstruction of blood circulation and inadequate blood oxygenation)

Systemic inflammatory response syndrome

The American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference Committee defined SIRS as the presence of at least 2 of the following 4 criteria:

- Core temperature of higher than 38°C or lower than 36°C
- Heart rate of more than 90 beats per minute
- Respiratory rate of more than 20 breaths per minute
- The clinical suspicion of systemic inflammatory response syndrome by an experienced clinician is of utmost importance.

Patients with shock frequently present with tachycardia, tachypnea, hypotension, altered mental status changes, and oliguria.

Physical Examination

Cardinal features of distributive shock include the following:

- Change in mental status
• Heart rate - Greater than 90 beats per minute (note that heart rate elevation is not evident if the patient is on a beta blocker)

• Hypotension - Systolic blood pressure less than 90 mm Hg or a reduction of 40 mm Hg from baseline

• Respiratory rate - Greater than 20 breaths per minute

• Extremities - Frequently warm, with bounding pulses and increased pulse pressure (systolic minus diastolic blood pressure) in early shock; late shock may present as critical organ dysfunction

• Hyperthermia - Core body temperature greater than 38.3°C

• Hypothermia - Core body temperature less than 36°C

• Pulse oximetry - Relative hypoxemia

• Decreased urine output

Clinical symptoms of the underlying infections found in distributive shock include the following:

• Pneumonia - Dullness to percussion, rhonchi, crackles, bronchial breath sounds

• Urinary tract infection - Costovertebral angle tenderness, suprapubic tenderness, dysuria and polyuria

• Intra-abdominal infection or acute abdomen - Focal or diffuse tenderness to palpation, diminished or absent bowel sounds, rebound tenderness

• Gangrene or soft-tissue infection - Pain out of proportion to lesion, skin discoloration and ulceration, desquamating rash, areas of subcutaneous necrosis

Anaphylaxis is characterized by the following clinical symptoms:

• Respiratory distress

• Wheezing

• Urticarial rash

• Angioedema

TSS is characterized by the following clinical symptoms:

• High fever

• Diffuse rash with desquamation on the palms and soles over a subsequent 1-2 weeks

• Hypotension (may be orthostatic) and evidence of involvement of 3 other organ systems

Streptococcal TSS more frequently presents with focal soft-tissue inflammation and is less commonly associated with diffuse rash. Occasionally, it can progress explosively within hours.
Adrenal insufficiency is characterized by the following clinical symptoms:

- Hyperpigmentation of skin, oral, vaginal, and anal mucosal membranes may be present in chronic adrenal insufficiency.
- In acute or acute-on-chronic adrenal insufficiency brought on by physiologic stress, hypotension may be the only physical sign.

Treatment & Management

Symptomatic and aetiological treatment must take place simultaneously.

In all cases:

Emergency: immediate attention to the patient.

Warm the patient, lay him flat, elevate legs (except in respiratory distress, acute pulmonary oedema).

Insert a peripheral IV line using a large calibre catheter (16G in adults). If no IV access, use intraosseous route.

Oxygen therapy, assisted ventilation in the event of respiratory distress.

Assisted ventilation and external cardiac compression in the event of cardiac arrest.

Intensive monitoring: consciousness, pulse, BP, CRT, respiratory rate, hourly urinary output (insert a urinary catheter) and skin mottling.

Management according to the cause

Haemorrhage

Control bleeding.

Priority: restore vascular volume as quickly as possible: Insert 2 peripheral IV lines

Ringer Lactate or 0.9% sodium chloride: replace 3 times the estimated losses

Severe acute dehydration due to bacterial/viral gastroenteritis

Urgently restore circulating volume using IV bolus therapy:

Ringer Lactate or 0.9% sodium chloride:

Children < 2 months: 10 ml/kg over 15 minutes. Repeat (up to 3 times) if signs of shock persist.

Children 2-59 months: 20 ml/kg over 15 minutes. Repeat (up to 3 times) if signs of shock persist.

Children ≥ 5 years and adults: 30 mg/kg over 30 minutes. Repeat once if signs of shock persist.

Then, replace the remaining volume deficit using continuous infusion until signs of dehydration resolve (typically 70 ml/kg over 3 hours).
Closely monitor the patient; be careful to avoid fluid overload in young children and elderly patients).

**Note:** in severely malnourished children the IV rate is different than those for healthy children

**Severe anaphylactic reaction**

Determine the causal agent and remove it, e.g. stop ongoing injections or infusions, but if in place, maintain the IV line.

Administer **epinephrine (adrenaline)** IM, into the antero-lateral tight, in the event of hypotension, pharyngolaryngeal oedema, or breathing difficulties:

Use *undiluted* solution (1:1000 = 1 mg/ml) and a 1 ml syringe graduated in 0.01 ml:

- **Children under 6 years:** 0.15 ml
- **Children from 6 to 12 years:** 0.3 ml
- **Children over 12 years and adults:** 0.5 ml

In children, if 1 ml syringe is not available, use a *diluted* solution, i.e. add 1 mg epinephrine to 9 ml of 0.9% sodium chloride to obtain a 0.1 mg/ml solution (1:10 000):

- **Children under 6 years:** 1.5 ml
- **Children from 6 to 12 years:** 3 ml

At the same time, administer rapidly **Ringer lactate** or **0.9% sodium chloride**: 1 litre in adults (maximum rate); 20 ml/kg in children, to be repeated if necessary.

If there is no clinical improvement, repeat IM epinephrine every 5 to 15 minutes.

In shock persists after 3 IM injections, administration of IV epinephrine at a constant rate by a syringe pump is necessary:

Use a *diluted* solution, i.e. add 1 mg epinephrine (1:1000) to 9 ml of 0.9% sodium chloride to obtain a 0.1 mg/ml solution (1:10 000):

- **Children:** 0.1 to 1 microgram/kg/minute
- **Adults:** 0.05 to 0.5 microgram/kg/minute

Corticosteroids have no effect in the acute phase. However, they must be given once the patient is stabilized to prevent recurrence in the short term:

**hydrocortisone hemisuccinate** IV or IM

- **Children:** 1 to 5 mg/kg/24 hours in 2 or 3 injections
- **Adults:** 200 mg every 4 hours

In patients with bronchospasm, epinephrine is usually effective. If the spasm persists give 10 puffs of inhaled **salbutamol**.
**Septic shock**

Vascular fluid replacement with Ringer Lactate or 0.9 % sodium chloride.

Use of vasoconstrictors:

**dopamine** IV at a constant rate by syringe pump: 10 to 20 micrograms/kg/minute or, if not available **epinephrine** IV at a constant rate by syringe pump:

Use a *diluted* solution, i.e. add 1 mg epinephrine (1:1000) to 9 ml of 0.9% sodium chloride to obtain a 0.1 mg/ml solution (1:10 000). Start with 0.1 microgram/kg/minute.

Increase the dose progressively until a clinical improvement is seen.

Look for the origin of the infection (abscess; ENT, pulmonary, digestive, gynaecological or urological infection etc.).
SEIZURES

Involuntary movements of cerebral origin (stiffness followed by clonic movements), accompanied by a loss of consciousness, and often urinary incontinence (generalized tonic-clonic seizures). It is important to distinguish seizures from ‘pseudo-seizures’ (e.g. in hysteria or tetany) during which consciousness may appear altered but is not lost.

Priorities: stop the seizures and determine the cause.

In pregnant women, eclamptic seizures require specific medical and obstetrical care

Initial treatment

During a seizure

Protect from trauma, maintain airway, place patient in ‘recovery position’, loosen clothing.

Most seizures are quickly self-limited. Immediate administration of an anticonvulsant is not systematic. If generalized seizure lasts more than 3 minutes, use diazepam to stop it: diazepam

Children: 0.5 mg/kg preferably rectally without exceeding 10 mg. IV administration is possible (0.3 mg/kg over 2 or 3 minutes), only if means of ventilation are available (Ambu bag and mask).

Adults: 10 mg slowly IV (or rectally).

In all cases:

• Dilute 10 mg (2 ml) of diazepam in 8 ml of 5% glucose or 0.9% sodium chloride.

• If convulsion continues, repeat dose once after 5 minutes.

• In infants and elderly patients, monitor respiration and blood pressure.

• If convulsion continues after the second dose, treat as status epilepticus.

The patient is no longer seizing

Look for the cause of the seizure and evaluate the risk of recurrence.

Keep diazepam and glucose available in case the patient starts seizing again.
STATUS EPILEPTICUS

Status epilepticus (SE) is a common, life-threatening neurologic disorder that is essentially an acute, prolonged epileptic crisis. SE can represent an exacerbation of a preexisting seizure disorder, the initial manifestation of a seizure disorder, or an insult other than a seizure disorder. In patients with known epilepsy, the most common cause is a change in medication. Most seizures terminate spontaneously.

By clinical history, nonmotor simple partial status epilepticus involves subjective sensory disturbances, including the following:

- Focal or unilateral paresthesias or numbness
- Focal visual changes, usually characterized by flashing lights
- Focal visual obscuration or focal colorful hallucinations
- Olfactory or gustatory hallucinations
- Atypical rising abdominal sensations

Diagnosis

Examination for status epilepticus includes the following:

- Generalized convulsive status epilepticus: Typical rhythmic tonic-clonic activity, impaired consciousness; rarely, may present as persistent tonic seizure
- Status epilepticus due to the use of illicit, or street, drugs: needle-track marks
- Status epilepticus due to possible mass lesion or brain infection: Papilledema, lateralized neurologic features
- Subtle or transformed status epilepticus: Any patient without improving level of consciousness within 20-30 minutes of cessation of generalized seizure activity
- Associated injuries in patients with seizures: May include tongue lacerations (typically lateral), shoulder dislocations, head trauma, facial trauma

Complications

Complications of status epilepticus are many. Systemic complications include the following:

- Hyperthermia
- Acidosis
- Hypotension
- Respiratory failure
- Rhabdomyolysis
- Aspiration
Management

Aggressive treatment is necessary for status epileptics. Clinicians should not wait for blood level results before administering a loading dose of phenytoin, regardless of whether the patient is already taking phenytoin.

Pharmacotherapy

Most patients with status epilepticus who are treated aggressively with a benzodiazepine, fosphenytoin, and/or phenobarbital experience complete cessation of their seizures. If status epilepticus does not stop, general anesthesia is indicated.

Medications used in the treatment of status epilepticus include the following:

- Benzodiazepines (eg, lorazepam, diazepam, midazolam): First-line agents
- Anticonvulsant agents (eg, phenytoin, fosphenytoin)
- Barbiturates (eg, phenobarbital, pentobarbital)
- Anesthetics (eg, propofol)

Supportive therapy

Supportive care in patients with status epilepticus includes the following:

- Maintenance of vital signs
- Airway, breathing, circulation (eg, hemodynamic/cardiac monitoring)
- Respiratory support, with intubation and/or mechanical ventilation if necessary
- Periodic neurologic assessments

Protect from trauma, loosen clothing, maintain airway and administer oxygen as required.

Insert an IV line.

Both generalized tonic-clonic status epilepticus (SE) and subtle SE must be treated aggressively. Maintenance of vital signs, including respiratory function, is of major importance. Any indication of respiratory insufficiency should be addressed by intubation. Early treatment measures are performed in concert with diagnostic studies. The treating physician should not wait for a blood level to return from a laboratory test before giving the patient a loading dose of phenytoin. The same protocol should be followed regardless of whether the patient is already taking phenytoin. Assume that the patient is noncompliant because this is the most common cause of SE in patients with known epilepsy.

Prehospital Care

Supportive care, including ABCs, must be addressed in the prehospital setting. If the seizure fails to stop within 4-5 minutes or if the patient is continuing to seize at the time of emergency EMS personnel arrival, prompt administration of anticonvulsants may be necessary.
Because of the refrigeration requirements and the infrequent use of most anticonvulsants, diazepam (Valium) is often the only anticonvulsant available in the prehospital setting. Diazepam may be administered IV or per rectum.

Establish intravenous access, ideally in a large vein. Intravenous administration is the preferred route for anticonvulsant administration because it allows therapeutic levels to be attained more rapidly. Begin cardiac and other hemodynamic monitoring.

Administer a 50-mL bolus of 50% dextrose IV and 100 mg of thiamine. If seizure activity does not terminate within 4-5 minutes, start anticonvulsant medication. If EMS history has already defined SE, treatment should begin immediately. In some settings where drug intoxication might be likely, consider also adding naloxone at 0.4-2.0 mg IV to the dextrose bag.

Administer diazepam (0.15 mg/kg) or lorazepam (0.1 mg/kg) IV over 5 minutes, followed by fosphenytoin. Fosphenytoin is given in a dose of 15-20 mg phenytoin equivalents [PE]/kg, at a rate not to exceed 150 mg PE/min. The dose of phenytoin is 18-20 mg/kg, at a rate not to exceed 50 mg/min. Never mix phenytoin with a 5% dextrose solution; put it in a normal saline solution to minimize the risk of crystal precipitation.
## Diagnostic work-up flowchart

- Check ABCs
- Insert IV
- STAT laboratory studies:
  - Electrolytes, calcium, magnesium
  - CBC
  - Liver and renal FX test
  - Toxicology screen
  - Anticonvulsant levels
  - Arterial blood gas
- Insert urinary catheter
- Urinalysis, urine toxicology
- Cardiac O2 saturation, monitors
- Consider the following during general and neurologic exam:
  - Trauma
  - Infection
  - Stroke
  - Drug ingestion
  - As indicated:
    - Chest x-ray
    - CT scan or MRI
    - Lumbar puncture
    - Blood cultures
    - Blood toxicology screen
- Treat underlying disorder(s)
- Admit to hospital

## Treatment flowchart

- Start an IV line, administer a 50-mL bolus of 50% dextrose IV and 100 mg of thiamine, then start the anticonvulsant. In some settings where drug intoxication might be likely, consider also adding naloxone at 0.4-2.0 mg IV to the dextrose bag.
- Administer diazepam (0.15 mg/kg) or lorazepam (0.1 mg/kg) IV over 5 minutes, followed preferably by fosphenytoin (15-20 mg phenytoin equivalents PE/kg at a rate not to exceed 150 mg PE/min) or phenytoin (18-20 mg/kg at a rate not to exceed 50 mg/min). Never mix phenytoin with a 5% dextrose solution: put it in a normal saline solution to minimize the risk of crystal precipitation.
- Intubate if necessary, and control hyperthermia.
- If seizures continue after 20 minutes, give additional fosphenytoin (10 mg PE/kg IV) or phenytoin (10 mg/kg IV). Aim for a total serum phenytoin level of about 22-25 μg/mL.
- If seizures continue after 20 minutes, give phenobarbital (15 mg/kg IV).
- If seizures continue, consider administering general anesthesia.
FEVER

Fever is the temporary increase in the body's temperature in response to a disease or illness.

A child has a fever when the temperature is at or above one of these levels:

- 38°C measured in the bottom (rectally)
- 37.5°C measured in the mouth (orally)
- 37.2°C measured under the arm (axillary)

An adult probably has a fever when the temperature is above 37.2 - 37.5°C, depending on the time of day.

A symptom is something the patient reports and feels, while a sign is something other people, including a doctor may detect. For example, a headache may be a symptom while a rash may be a sign.

When somebody has a fever, signs and symptoms are linked to what is known as sickness behavior, and may include:

A person is said to have a fever if the temperature in the mouth is over 37.7°C (99.9F). Temperature can also be measured in the rectum (anus), under the arm or inside the ear.

- Feeling cold when nobody else does
- Shivering
- Anorexia
- Dehydration
- Depression
- Hyperalgesia
- Lethargy
- Problems concentrating
- Sleepiness
- Sweating

In a febrile patient, first look for signs of serious illness, then try to establish a diagnosis.
FEVER WITHOUT A FOCUS

Infants or young children who have a fever with no obvious source of infection.

Fever is defined as a rectal temperature that exceeds 38°C. Direct the initial evaluation of these patients toward identifying or ruling out SBI, most commonly urinary tract infections. Meningitis, pneumonia, UTI, and bacteremia are serious etiologies of fever in infants and young children.

History

Obtaining an accurate history from the parent or caregiver is important when assessing fever without a focus; the history obtained should include the following information:

- Fever history: What was child's temperature prior to presentation and how was temperature measured? Consider fever documented at home by a reliable parent or caregiver the same as fever found upon presentation. Accept parental reports of maximum temperature.
- Fever at presentation: If the physician believes the infant has been excessively bundled, and if a repeat temperature taken 15-30 minutes after unbundling is normal, the infant should be considered afebrile. Always remember that normal or low temperature does not preclude serious, even life-threatening, infectious disease.
- Current level of activity or lethargy
- Activity level prior to fever onset (ie, active, lethargic)
- Current eating and drinking pattern
- Eating and drinking pattern prior to fever onset
- Appearance: Fever sometimes makes a child appear rather ill
- Vomiting or diarrhea
- Ill contacts
- Medical history
- Immunization history (especially recent immunizations)
- Urinary output: Inquire as to the number of wet diapers

Physical

While performing a complete physical examination, pay particular attention to assessing hydration status and identifying the source of infection. Physical examination of every febrile child should include the following:

Record vital signs.

- Temperature: Rectal temperature is the standard. Temperature obtained via tympanic, axillary, or oral methods may not truly reflect the patient’s temperature.
- Pulse rate
- Respiratory rate
- Blood pressure

Measure pulse oximetry levels.

- Pulse oximetry may be a more sensitive predictor of pulmonary infection than respiratory rate in patients of all ages, but especially in infants and young children.
- Pulse oximetry is mandatory for any child with abnormal lung examination findings, respiratory symptoms, or abnormal respiratory rate, although keep in mind that the respiratory rate increases when children are febrile.

Record an accurate weight on every chart.

- All pharmacologic and procedural treatments are based on the weight in kilograms.
- In urgent situations, estimating methods (eg, Broselow tape, weight based on age) may be used.

During the examination, concentrate on identifying any of the following:

- Toxic appearance, which suggests possible signs of lethargy, poor perfusion, hypoventilation or hyperventilation, or cyanosis (ie, shock)
- A focus of infection that is the apparent cause of the fever
- Minor foci (eg, otitis media, pharyngitis, sinusitis, skin or soft tissue infection)
- Identifiable viral infection (eg, bronchiolitis, croup, gingivostomatitis, viral gastroenteritis, varicella, hand-foot-and-mouth disease)
- Petechial or purpuric rashes, often associated with bacteremia
- Purpura, which is associated more often with meningococcemia than is the presence of petechiae alone

For all patients aged 2-36 months, management decisions are based on the degree of toxicity and the identification of serious bacterial infection.
The Yale Observation Scale is a reliable method for determining degree of illness.

<table>
<thead>
<tr>
<th>Observation Items</th>
<th>1 (Normal)</th>
<th>3 (Moderate Impairment)</th>
<th>5 (Severe Impairment)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quality of cry</td>
<td>Strong with normal tone or contentment without crying</td>
<td>Whimpering or sobbing</td>
<td>Weak cry, moaning, or high-pitched cry</td>
</tr>
<tr>
<td>Reaction to parent stimulation</td>
<td>Brief crying that stops or contentment without crying</td>
<td>Intermittent crying</td>
<td>Continual crying or limited response</td>
</tr>
<tr>
<td>Color</td>
<td>Pink</td>
<td>Acrocyanotic or pale extremities</td>
<td>Pale or cyanotic or mottled or ashen</td>
</tr>
<tr>
<td>State variation</td>
<td>If awake, stays awake; if asleep, wakes up quickly upon stimulation</td>
<td>Eyes closed briefly while awake or awake with</td>
<td>Falls asleep or will not arouse</td>
</tr>
<tr>
<td></td>
<td></td>
<td>prolonged stimulation</td>
<td></td>
</tr>
<tr>
<td>Hydration</td>
<td>Skin normal, eyes normal, and mucous membranes moist</td>
<td>Skin and eyes normal and mouth slightly dry</td>
<td>Skin doughy or tented, dry mucous membranes, and/or sunken eyes</td>
</tr>
<tr>
<td>Response (eg, talk, smile) to social overtures</td>
<td>Smiling or alert (&lt; 2 mo)</td>
<td>Briefly smiling or alert briefly (&lt; 2 mo)</td>
<td>Unsmiling anxious face or dull, expressionless, or not alert (&lt; 2 mo)</td>
</tr>
</tbody>
</table>

**Medical Care**

For children with fever without a focus who appear ill, conduct a complete evaluation to identify occult sources of infection. Follow the evaluation with empiric antibiotic treatment and admit the patient to a hospital for further monitoring and treatment pending culture results.

Patients aged 2-36 months may not require admission if they meet the following criteria:

- Patient was healthy prior to onset of fever.
- Patient is fully immunized.
- Patient has no significant risk factors.
- Patient appears nontoxic and otherwise healthy.
- Patient's parents (or caregivers) appear reliable and have access to transportation if the child's symptoms should worsen.

Treatment recommendations for children with fever without a focus are based on the child's appearance, age, and temperature.
For children who do not appear toxic, treatment recommendations are as follows:

- Schedule a follow-up appointment within 24-48 hours and instruct parents to return with the child sooner if the condition worsens.

- Hospital admission is indicated for children whose condition worsens or whose evaluation findings suggest a serious infection.

The need to consult with specialists depends on the specialty of the physician who initially evaluated the patient and the ultimate source of fever. Typically, general pediatricians easily manage febrile infants on both an inpatient and outpatient follow-up basis.

**Medication**

**Symptomatic treatment**

Undress the patient.

**Antipyretics:**

**Paracetamol PO**

Children: 60 mg/kg/day in 3 or 4 divided doses

Adults: 3 to 4 g/day in 3 or 4 divided doses

or

**ASA PO** (to be avoided in children under 16 years)

Adults: 1 to 3 g/day in 3 or 4 divided doses

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<table>
<thead>
<tr>
<th>AGE</th>
<th>2 MONTHS</th>
<th>1 YEAR</th>
<th>5 YEARS</th>
<th>15 YEARS</th>
<th>ADULT</th>
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</thead>
<tbody>
<tr>
<td>Weight</td>
<td>4 kg</td>
<td>8 kg</td>
<td>15 kg</td>
<td>35 kg</td>
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<tr>
<td><strong>Paracetamol</strong></td>
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<tr>
<td>120 mg/5 ml oral solution</td>
<td>2 ml x 3</td>
<td>3 to 6 ml x 3</td>
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<tr>
<td>100 mg tablet</td>
<td>1/2 tab x 3</td>
<td>3/4 to 1 1/2 tab x 3</td>
<td>1 1/2 to 3 tab x 3</td>
<td>-</td>
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<tr>
<td>500 mg tablet</td>
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<td><strong>A.S.A.</strong></td>
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<tr>
<td>300 mg tablet</td>
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<td></td>
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<td></td>
<td>2 tab x 3</td>
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<tr>
<td>500 mg tablet</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1 tab x 3</td>
</tr>
</tbody>
</table>
Ibuprofen PO

Children under 3 months do not administer

Children over 3 months: 30 mg/kg/day in 3 divided doses

Children over 15 years around 35 kg 200 mg tablet 1 to 2 tab x 3

Adults: 1200 to 1800 mg/day in 3 to 4 divided doses

**RED FLAG - Caution**

- Paracetamol is the drug of choice for pregnant and breast-feeding women.

- Acetylsalicylic acid is not recommended during the first 5 months of pregnancy, contra-indicated from the beginning of the 6th month, and to be avoided in breastfeeding-women.

- Ibuprofen is not recommended during the first 5 months of pregnancy and contraindicated from the beginning of the 6th month. It can be administered to breastfeeding women as short-term treatment.
PAIN in ADULTS

A.- TYPES OF PAIN

A.1. NOCICEPTIVE PAIN

Usual form of pain, acute or chronic. Nervous system without damage.

B.1.1 Somatic pain: Comes from the skin and deep tissues and is easier to locate than visceral pain: musculoskeletal pain, backpain, headache, arthritis.

B.2.2 Visceral pain: Comes from the internal organs. Usually dull and vague, and may be harder to pinpoint: bladder pain, prostate pain, irritable bowel syndrome, endometriosis, dysmenorrhea, nephritic colic.

A.2 NEUROPATHIC PAIN

Less frequent. Nerves have damage. Recurrent acute attacks of pain such as paresthesia or burning, usually with dysesthesia.

Some forms of pain can be a combination of nociceptive and neuropathic, e.g. some teeth pain.

B.- TREATMENT

Oral forms should be used whenever possible

The combination of different drugs (multimodal analgesia) is adequate (e.g. Paracetamol and NSAIDs; Paracetamol and codeine)

Do not use simultaneously weak and strong opioids

The dosage and duration of treatment are guided by the assessment of type, intensity and location of the pain
B.1. NOCICEPTIVE PAIN

WHO PAIN LADDER
(TREATMENT of NOCICEPTIVE SOMATIC PAIN)

<table>
<thead>
<tr>
<th>LEVEL</th>
<th>RATING</th>
<th>TREATMENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>MILD</td>
<td>1-2</td>
<td>Paracetamol /ASA/ NSAIDs</td>
</tr>
<tr>
<td>MODERATE</td>
<td>3-6</td>
<td>As Mild Pain + Weak Opioids (Codeine, Tramadol)</td>
</tr>
<tr>
<td>SEVERE</td>
<td>7-10</td>
<td>As Mild Pain + Strong Opioids (Morphine, Fentanyl)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>GROUP</th>
<th>DRUG</th>
<th>USUAL DOSAGE (Oral)</th>
<th>MAX p.d</th>
<th>OTHER CONSIDERATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>ANALGESICS</td>
<td>Paracetamol</td>
<td>500mg/4-6 h</td>
<td>3.000 mg</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Metamizol</td>
<td>575mg/6-8h</td>
<td>3.500 mg</td>
<td></td>
</tr>
<tr>
<td>SALICILATES</td>
<td>AcetilSalicylic Acid (ASA)</td>
<td>500mg/4-6h</td>
<td>3.500 mg</td>
<td></td>
</tr>
<tr>
<td>NSAIDs</td>
<td>Ibuprofen</td>
<td>400mg/8h</td>
<td>1.200 mg</td>
<td></td>
</tr>
<tr>
<td></td>
<td>600mg/12h</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>200mg/4h</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Naproxen</td>
<td>500mg/12h</td>
<td>1.000 mg</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Diclofenac</td>
<td>50mg/8h</td>
<td>150 mg</td>
<td></td>
</tr>
<tr>
<td></td>
<td>75mg/12-24 h (Retard)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Weak Opioids</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Codeine</td>
<td>10-30mg/6-8 h</td>
<td>240 mg</td>
<td>*Nausea and Vomiting are frequent at the beginning of treatment: metoclopramide 5-10mg/8h.</td>
</tr>
<tr>
<td></td>
<td>Tramadol</td>
<td>25-50mg/6-8 h</td>
<td>400 mg</td>
<td>*Dosage dependent drowsiness</td>
</tr>
<tr>
<td></td>
<td>Strong Opioids</td>
<td></td>
<td></td>
<td>*Constipation is common. Add a laxative if treatment is &gt;48h: Lactulose 15-45 ml/p.d., Bisacodyl 10-15 mg/p.d.</td>
</tr>
<tr>
<td></td>
<td>Morphine</td>
<td>Fast: 5-10mg/4h</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Retard: 20-150mg/12h</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Fentanyl</td>
<td>Transdermal: 12-100mcg/h</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sublingual: 100-800mcg/4h</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Visceral pain can be treated with the same criteria and drugs than somatic pain. Antispasmodics can also be useful.

<table>
<thead>
<tr>
<th>GROUP</th>
<th>DRUG</th>
<th>USUAL (Oral)</th>
<th>DOSAGE</th>
<th>MAX p.d</th>
<th>OTHER CONSIDERATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>ANTISPASMODIC</td>
<td>Hioscine Butylbromide</td>
<td>20mg/6h</td>
<td>120mg</td>
<td></td>
<td>Side effects: Dry mouth, constipation, nausea</td>
</tr>
</tbody>
</table>

B.2. NEUROPATHIC PAIN

<table>
<thead>
<tr>
<th>GROUP</th>
<th>DRUG</th>
<th>USUAL (Oral)</th>
<th>DOSAGE</th>
<th>MAX p.d</th>
<th>OTHER CONSIDERATIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>ANTIDEPRESSANTS</td>
<td>Amitryptiline</td>
<td>10-25 mg/p.d, at night. Increase doses depending on effect</td>
<td>150mg</td>
<td></td>
<td>Side effects: Dry mouth, constipation.</td>
</tr>
</tbody>
</table>

C. - SPECIFIC CLINICAL SCENARIOS

C.1. Mechanic musculoskeletal pain

Paracetamol or/and NSAIDs.

Ointment liniment and heat are useful

C.2 Acute backpain with neuralgia

Consider first acute i.m treatment (Diclofenac 75 mg) followed by oral NSAIDs.

Do not prescribe benzodiazepines (e.g. diazepam)

C.3. Osteoarthritis Pain

Paracetamol or/and NSAIDs.

Do not offer glucosamine or chondroitin products.

Topical NAIDs have proven to be effective in knee and shoulder pain.

C.4. Nephritic Colic:

Diclofenac 75 mg i.m. followed by oral NSAIDs and SPASMOLITICS.

If nausea or vomiting, metoclopramide 10mg i.m followed by oral intake if needed.

C.5. Dysmenorrhea: Naproxen
Clinical Validation of FLACC: Preverbal Patient Pain Scale

To examine the difference across time for the FLACC scores, a one-way ANOVA was performed using a within factor of measurement time (three time points). Pre-analgesic FLACC scores (Time 1) ranged from 1-10. Onset of analgesia FLACC scores (Time 2) and peak analgesia FLACC scores (Time 3) ranged from 0-10.

Observational evaluation scale - Children 2 months-5 years

<table>
<thead>
<tr>
<th>Categories</th>
<th>Scoring</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Face</td>
<td>No particular expression or smile</td>
</tr>
<tr>
<td>Legs</td>
<td>Normal position or relaxed</td>
</tr>
<tr>
<td>Activity</td>
<td>Lying quietly, normal position, moves easily</td>
</tr>
<tr>
<td>Cry</td>
<td>No cry (awake or asleep)</td>
</tr>
<tr>
<td>Consolability</td>
<td>Content, relaxed</td>
</tr>
</tbody>
</table>

Note: Each of the five categories Face (F), Legs (L), Activity (A), Cry (C), and Consolability (C) is scored from 0-2, which results in a total score between 0 and 10.


Each category is scored from 0 to 2, giving a final score between 0 and 10.

0 to 3: mild pain, 4 to 7: moderate pain, 7 to 10: severe pain

Observational evaluation scale - Children under 2 months NFCS scale (Neonatal Facial Coding System)

| Table 1. Neonatal Facial Coding System<sup>AE</sup> |
|-----------------------------------------------|-----------------|-----------------|
| 0 points                                     | 1 point         |
| Brow bulge                                   | Absent          | Present         |
| Eye squeeze                                  | Absent          | Present         |
| Deepened nasolabial furrow                   | Absent          | Present         |
| Open lips                                    | Absent          | Present         |
| Stretched mouth                              | Absent          | Present         |
| Lip purse                                    | Absent          | Present         |
| Taut tongue                                  | Absent          | Present         |
| Chin quiver                                  | Absent          | Present         |

A score of 2 or more signifies significant pain, requiring analgesic treatment
Figure illustrates some of the common facial actions corresponding to pain in infants.
Malnutrition is defined by the World Health Organization as a "cellular imbalance between the supply of nutrients and energy and the body's demand for them to ensure growth, maintenance, and specific functions. Many diseases are directly or indirectly caused by a lack of essential nutrients in the diet.

**Signs and symptoms**
Clinical signs and symptoms of PEM include the following:
- Poor weight gain
- Slowing of linear growth
- Behavioral changes: Irritability, apathy, decreased social responsiveness, anxiety, and attention deficits

The most common and clinically significant micronutrient deficiencies and their consequences include the following:
- Iron: Fatigue, anemia, decreased cognitive function, headache, glossitis, and nail changes
- Iodine: Goiter, developmental delay, and mental retardation
- Vitamin D: Poor growth, rickets, and hypocalcemia
- Vitamin A: Night blindness, xerophthalmia, poor growth, and hair changes
- Folate: Glossitis, anemia (megaloblastic), and neural tube defects (in fetuses of women without folate supplementation)
- Zinc: Anemia, dwarfism, hepatosplenomegaly, hyperpigmentation and hypogonadism, acrodermatitis enteropathica, diminished immune response, and poor wound healing

**Physical examination**
Physical findings that are associated with PEM include the following:
- Decreased subcutaneous tissue: Areas that are most affected are the legs, arms, buttocks, and face
- Edema: Areas that are most affected are the distal extremities and anasarca (generalized edema)
- Oral changes: Cheilosis, angular stomatitis, and papillary atrophy
- Abdominal findings: Abdominal distention secondary to poor abdominal musculature and hepatomegaly secondary to fatty infiltration
- Skin changes: Dry, peeling skin with raw, exposed areas; hyperpigmented plaques over areas of trauma
- Nail changes: Fissured or ridged nails
- Hair changes: Thin, sparse, brittle hair that is easily pulled out and that turns a dull brown or reddish color
Diagnosis

Initial diagnostic laboratory studies include the following:

- Complete blood count
- Sedimentation rate
- Serum electrolytes
- Urinalysis
- Culture

Management

Children with chronic malnutrition may require caloric intakes of more than 120-150 kcal/kg/day to achieve appropriate weight gain. Most children with mild malnutrition respond to increased oral caloric intake and supplementation with vitamin, iron, and folate supplements. The requirement for increased protein is met typically by increasing the food intake.

In moderate to severe cases of malnutrition, enteral supplementation via tube feedings may be necessary.
Clinical Note:

If a patient presents to a general practice during an acute coronary syndrome, it may be a non-clinical member of staff who is their first point of contact. It is therefore important that all staff members are aware of the practice protocol for managing patients with unexplained chest pain, and know how to initiate the protocol.

Staff members should know which room in the practice is the most appropriate to locate patients requiring urgent attention. Ideally this room will have an examination bed or couch with clear access on all sides and an ECG machine on hand, as well as convenient access for ambulance staff and equipment. All staff should be alerted to the location and status of the patient, who should not be left unattended.

If the patient cannot be transported to hospital immediately

If there will be a significant delay, i.e. more than two hours, in transporting patients with an acute coronary syndrome to hospital then it is appropriate to discuss the patient with an emergency medicine consultant or a cardiologist who may suggest additional interventions, if the required medicines are available. Patients presenting in rural areas are most likely to be considered for these additional treatments.
Include the following information where possible:

- Time of onset of symptoms and duration
- Previous and current ECGs
- Current blood pressure, heart rate and oxygen saturation levels
- A list of any medicines given acutely, including time and dose
- Co-morbidities
- All medicines currently prescribed as well as any over-the-counter products
- Allergies
- Any relevant person details such as an advanced care plan
- Any relevant family history

Check immediately if chest pain is current, or when the last episode was, particularly if in the last 12 hours. Check if the chest pain may be cardiac. Consider:

- history of the pain
- any cardiovascular risk factors
- history of ischaemic heart disease
- any previous treatment previous investigations for chest pain.

Initially assess people for any of the following symptoms, which may indicate an acute coronary syndrome:

pain in the chest and/or other areas (for example, the arms, back or jaw) lasting longer than 15 minutes chest pain with nausea and vomiting, marked sweating or breathlessness (or a combination of these), or with haemodynamic instability new onset chest pain, or abrupt deterioration in stable angina, with recurrent pain occurring frequently with little or no exertion and often lasting longer than 15 minutes. Central chest pain may not be the main symptom.

**Do not use** response to GTN to make a diagnosis of acute coronary syndrome.

**Do not assess** symptoms of an acute coronary syndrome differently in men and women or among different ethnic groups. There are no major differences in symptoms of an acute coronary syndrome among different ethnic groups.
Making a diagnosis based on clinical assessment

Anginal pain is:

- constricting discomfort in the front of the chest, or in the neck, shoulders, jaw, or arms
- precipitated by physical exertion
- relieved by rest

Use clinical assessment and the typicality of anginal pain features listed below to estimate the likelihood of CAD (see table 1):

- Three of the features above are defined as typical angina.
- Two of the three features above are defined as atypical angina.
- One or none of the features above are defined as non-anginal chest pain.

---

Percentage of people estimated to have coronary artery disease according to typicality of symptoms, age, sex and risk factors

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Non-anginal chest pain</th>
<th>Atypical angina</th>
<th>Typical angina</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Men</td>
<td>Women</td>
<td>Men</td>
</tr>
<tr>
<td>35</td>
<td>3</td>
<td>35</td>
<td>1</td>
</tr>
<tr>
<td>45</td>
<td>9</td>
<td>47</td>
<td>2</td>
</tr>
<tr>
<td>55</td>
<td>23</td>
<td>59</td>
<td>4</td>
</tr>
<tr>
<td>65</td>
<td>49</td>
<td>69</td>
<td>9</td>
</tr>
</tbody>
</table>

For men older than 70 with atypical or typical symptoms, assume an estimate > 90%.
For women older than 70, assume an estimate of 61–90% EXCEPT women at high risk AND with typical symptoms where a risk of > 90% should be assumed.

Values are per cent of people at each mid-decade age with significant coronary artery disease (CAD).
Hi = High risk = diabetes, smoking and hyperlipidaemia (total cholesterol > 6.47 mmol/litre).
Lo = Low risk = none of these three.
The ‘non-anginal chest pain’ columns represent people with symptoms of non-anginal chest pain, who would not be investigated for stable angina routinely.

Note:
These results are likely to overestimate CAD in primary care populations.
If there are resting ECG ST-T changes or Q waves, the likelihood of CAD is higher in each cell of the table.
<table>
<thead>
<tr>
<th>Classical Anginal Chest Pain</th>
<th>Atypical Chest Pain</th>
<th>Anginal Equivalents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central Anterior Pain</td>
<td>Epigastric discomfort</td>
<td>Dyspnea</td>
</tr>
<tr>
<td>Chest Pressure, tightness</td>
<td>Musculoskeletal</td>
<td>Syncope</td>
</tr>
<tr>
<td>Crushing Pain</td>
<td>Often Unilateral</td>
<td>“Generally Weak”</td>
</tr>
<tr>
<td>Pain radiating to arms, neck and back</td>
<td>Nausea/Vomiting</td>
<td>Palpitations</td>
</tr>
</tbody>
</table>

Unless clinical suspicion is raised based on other aspects of the history and risk factors, exclude a diagnosis of stable angina if the pain is non-anginal. Other features which make a diagnosis of stable angina unlikely are when the chest pain is:

- continuous or very prolonged and/or
- unrelated to activity and/or
- brought on by breathing in and/or
- associated with symptoms such as dizziness, palpitations, tingling or difficulty swallowing.
- Consider causes of chest pain other than angina (such as gastrointestinal or musculoskeletal pain).

Offer pain relief as soon as possible. This may be achieved with GTN (sublingual or buccal), but offer intravenous opioids such as morphine, particularly if an acute myocardial infarction is suspected.

Offer people a single loading dose of 324 mg aspirin as soon as possible unless there is clear evidence that they are allergic to it.

If aspirin is given before arrival at hospital, send a written record that it has been given with the person.

Do not routinely administer oxygen, but monitor oxygen saturation using pulse oximetry as soon as possible, ideally before hospital admission. Only offer supplemental oxygen to:

- people with oxygen saturation (SpO2) of less than 94% who are not at risk of hypercapnic respiratory failure, aiming for SpO2 of 94-98% people with chronic obstructive pulmonary disease who are at risk of hypercapnic respiratory failure, to achieve a target SpO2 of 88-92% until blood gas analysis is available.

Take a resting 12-lead ECG as soon as possible. When people are referred, send the results to hospital before they arrive if possible. Recording and sending the ECG should not delay transfer to hospital.

A number of changes on a resting 12-lead ECG are consistent with CAD and may indicate ischaemia or previous infarction. These include:

- pathological Q waves in particular
- LBBB
- ST-segment and T wave abnormalities (for example, flattening or inversion). Note that the results may not be conclusive.
Consider any resting 12-lead ECG changes together with people's clinical history and risk factors.

If a ST segment elevation is detected, the patient should be immediately referred to hospital, as in these patients

Monitor people with acute chest pain, using clinical judgement to decide how often this should be done, until a firm diagnosis is made. This should include:

- exacerbations of pain and/or other symptoms
- pulse and blood pressure
- heart rhythm
- oxygen saturation by pulse oximetry
- repeated resting 12-lead ECGs
- checking pain relief is effective.

EMT/EMT-INTERMEDIATE STANDING ORDERS

- Routine Patient Care
- **Aspirin** 324 mg. Check allergy status. Check contraindications.
- **Nitroglycerin**. Give glyceryl trinitrate 0.4 mg sublingual or equivalent every 5 min up to 3 doses as SBP allows. Begin IV dosing at 10 micro-grams min^{-1} in persistent pain or pulmonary edema; titrate to desired BP effect. IV must be established before administration of nitroglycerin
- Must be patient’s own NTG
- SBP must be >100mmHg

Oxygen treatment should not be routinely administered. DO NOT administer oxygen to patients with an ST elevation acute coronary syndrome unless they:

- Are breathless
- Are hypoxic, i.e. oxygen saturation < 93%
- Have heart failure
- Are in cardiogenic shock

PARAMEDIC STANDING ORDERS

**NOTE**: A second IV line may be indicated for high-risk patient.

- Medication interventions based on risk for ACS, clinical presentation and/or diagnostic EKG changes.

MEDICAL CONTROL MAY ORDER

- Additional doses of above medications
- **Intravenous (IV) morphine** is effective for severe pain in a patient with an acute coronary syndrome. For example, give morphine 5 -10 mg IV at 1–2 mg/minute, repeat if necessary; morphine 2.5 – 5 mg for older or frail patients.
- **An IV antiemetic**, e.g. metoclopramide 10 mg or cyclizine 25 mg, is usually administered at the same time as, or immediately prior to, IV morphine.
- **Dispersible aspirin** 324 mg, should be given to all patients with an acute coronary syndrome, including those already taking aspirin; if enteric coated aspirin is the only formulation available the patient should chew the tablet. Treatment with aspirin in all patients unless there are contraindications.
- **Clopidogrel** 300 mg (75 mg for patients aged over 75 years) given immediately along with aspirin, is recommended for patients with an acute coronary syndrome who also have evidence of ischaemia on ECG

**N.B.** **Clopidogrel may not be routinely available** in general practices as it is not able to be obtained under a Practitioner’s Supply Order
RED FLAG - Caution

Avoid nitroglycerin in ALL patients who have used a phosphodiesterase inhibitor such as: sildenafil (Viagra), vardenafil (Levitra), tadalafil (Cialis) within the last 48 HOURS. These medications are often used for erectile dysfunction and pulmonary hypertension. Also avoid use in patients receiving intravenous epoprostenol (Flolan) which is also used for pulmonary hypertension.

Administer nitrates with extreme caution, if at all, to patients with inferior-wall STEMI or suspected right ventricular (RV) involvement because these patients require adequate RV preload.

THE PATIENT SHOULD BE IMMEDIATELY REFERRED TO HOSPITAL
GENERAL ADULT ASSESSMENT

12-Lead ECG

STEMI

A Vascular Access

E Oxygen Keep SpO2 >94%

E ASPIRIN 324 mg PO

NITROGLYCERIN

E Assist pt with own NTG as prescribed; may repeat x 3

P 0.4 mg SL; may repeat q 5 min x 3

P Pain Management for continued pain

Nitroglycerin is contraindicated in any patient with hypotension, bradycardia, tachycardia (HR >100 bpm) in the absence of heart failure, and evidence of a right ventricular infarction. Caution is advised in patients with Inferior Wall STEMI and a right-sided ECG should be performed to evaluate RV infarction.

Non-Diagnostic 12-Lead

A Vascular Access

E Oxygen Keep SpO2 >94%

E ASPIRIN 324 mg PO

NITROGLYCERIN

E Assist pt with own NTG as prescribed; may repeat x 3

P 0.4 mg SL; may repeat q 5 min x 3

P Pain Management for continued pain

Consider Anti-emetic for nausea/vomiting: ONDANSETRON 4.0 mg ODT/IM/IV

THE PATIENT SHOULD BE IMMEDIATELY REFERRED TO HOSPITAL
CHEST PAIN

GENERAL ADULT ASSESSMENT

12-Lead ECG

Non-Specific Chest Pain

- **E**: Oxygen Keep SpO2 >94%
- **A**: Consider Vascular Access
- **A**: Consider ALBUTEROL for constricted airways 2.5 mg SVN
- **P**: Consider Pain Management

Suspected Aortic Dissection

- **E**: Oxygen Keep SpO2 >94%
- **A**: Vascular Access
  - N/S 500 ml bolus; may repeat up to 2000 ml for hypotension
- **P**: Pain Management

Suspected Cardiac Origin

- **A**: Acute Coronary Syndrome (Suspected)
History

- Age
- Medications
- Past medical history (MI, angina, diabetes, post menopausal)
- Allergies
- Recent physical exertion
- Palliation/Provocation
- Quality (crampy, constant, sharp, dull, etc.)
- Region/Radiation/Referred
- Time (onset/duration/repetition)

Signs and Symptoms

- CP (pressure, pain, ache, vicelike tightness)
- Location (substernal, epigastric, arm, jaw, neck, shoulder)
- Pale
- Diaphoresis
- Shortness of breath
- Nausea, vomiting, dizzy
- Time of onset
Management of atrial fibrillation in general practice

In people diagnosed with AF, there are two separate but equally important issues that must be considered. These are:

- Symptom management
- Assessment and management of thromboembolic risk

A stepwise approach to management is recommended

1. Confirm the diagnosis with ECG
2. Consider if urgent referral to secondary care is required
3. Determine the type of AF (e.g. persistent, paroxysmal or permanent)
4. Symptom management
5. Assess stroke risk to determine if antithrombotic treatment is required

Typical symptoms include

Symptoms
Identify the presence of the following symptoms:
A. Palpitations
B. Dyspnea
C. Dizziness, presyncope, or syncope
D. Chest pain
E. Weakness or fatigue
Check when the symptoms started, how often they occur and how long they last.

**STEP 2: Functionality**

**Functionality**
Determine if the symptoms associated with AF (or the treatment of AF) affect the patient’s functionality (subjective quality of life).

**The CCS-SAF Scale:**

<table>
<thead>
<tr>
<th>Class</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Asymptomatic with respect to AF.</td>
</tr>
</tbody>
</table>
| 1     | Symptoms attributable to AF have **minimal** effect on patient’s general quality of life:  
• minimal and/or infrequent symptoms; or  
• single episode of AF without syncope or heart failure. |
| 2     | Symptoms attributable to AF have **minor** effect on patient’s general quality of life:  
• mild awareness of symptoms in patients with persistent/permanent AF; or  
• rare episodes (e.g., less than a few per year) in patients with paroxysmal or intermittent AF. |
| 3     | Symptoms attributable to AF have **moderate** effect on patient’s general quality of life:  
• moderate awareness of symptoms on most days in patients with persistent/permanent AF; or  
• more frequent episodes (e.g., more than every few months) or more severe symptoms, or both, in patients with paroxysmal or intermittent AF. |
| 4     | Symptoms attributable to AF have **severe** effect on patient’s general quality of life:  
• very unpleasant symptoms in patients with persistent/paroxysmal AF; and/or  
• frequent and highly symptomatic episodes in patients with paroxysmal or intermittent AF; and/or syncope thought to be due to AF; and/or congestive heart failure secondary to AF. |

**STEP 3: Refer for or start acute management if hemodynamically unstable**

Consider referral to the emergency department or start acute management if highly symptomatic.

The majority of people presenting with symptoms consistent with new onset AF will not be haemodynamically compromised, however, urgent referral to secondary care for possible cardioversion is required if the patient has:

- A pulse rate > 150 beats per minute or a systolic blood pressure of < 90 mmHg
- Chest pain, increasing shortness of breath, severe dizziness or loss of consciousness (includes patients with acute ischaemic changes on ECG)
- Any complications of AF such as TIA, stroke, acute ischaemia or acute heart failure
Probable paroxysmal AF (as this requires medicines not usually initiated in primary care such as amiodarone or sotalol)

- ECG abnormalities such as Wolff-Parkinson-White syndrome or prolonged QT interval
- Known or suspected valvular disease
- Ongoing symptoms despite appropriate rate control treatment

The three types of AF are:

- **Paroxysmal AF** – characterised by recurrent episodes of AF that last less than seven days (although often less than 24 hours) and resolve spontaneously within that time. Rhythm control is the preferred treatment.
- **Persistent AF** – characterised by episodes of AF that last more than seven days and that has not spontaneously resolved within this time. Treatment is rate or rhythm control depending on the individual patient situation.
- **Permanent AF** – AF that has been present for more than one year and cardioversion has failed or not been attempted. Rate control is preferred.

*ENOXAPARIN* 1 mg/kg twice daily

**STEP 6: Start rate/rhythm control if not highly symptomatic**

**EMT/EMT-INTERMEDIATE/ADVANCED EMT STANDING ORDERS**

- Routine Patient Care
  (e.g., CCS-SAF ≤ 3, minimal to moderate effects on patient’s quality of life) and hemodynamically stable (systolic blood pressure > 90 mmHg, a heart rate < 120 beats per minute (BPM) and without clinical signs of shock).
**Symptom management**

**Rate or rhythm control?** The choice between rate or rhythm control is guided by the type of AF and other factors such as age, the presence of co-morbidities, the presence or absence of symptoms and patient preference.

**Rate control is recommended** for the majority of patients. It should be considered in particular for patients with:

- Asymptomatic AF
- Permanent AF

**Rhythm control**, which aims to restore and maintain sinus rhythm, should be considered for patients with:

- Paroxysmal AF
- Persistent AF and ongoing symptoms, any haemodynamic compromise, failure of rate control or persistent symptoms despite rate control
- Structural heart disease, e.g. severe left ventricular dysfunction or hypertrophic cardiomyopathy (AF is usually not well tolerated in these patients)

**Rate control medicines used in atrial fibrillation**

<table>
<thead>
<tr>
<th>Co-morbidity</th>
<th>First line</th>
<th>Second line</th>
<th>Third line</th>
<th>Fourth line</th>
</tr>
</thead>
<tbody>
<tr>
<td>No heart disease</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>Beta- blockers (not sotalol)</td>
<td>Calcium channel blockers</td>
<td>Digoxin</td>
<td>Amiodarone</td>
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<tr>
<td>Ischaemic heart disease</td>
<td>Metoprolol or carvedilol</td>
<td></td>
<td>Diltiazem</td>
<td></td>
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<tr>
<td>Congestive heart failure</td>
<td>Metoprolol or carvedilol</td>
<td>Digoxin</td>
<td>Diltiazem</td>
<td></td>
</tr>
<tr>
<td>COPD</td>
<td>Calcium channel blockers</td>
<td>Beta- blockers (provided no significant reversible bronchospasm)</td>
<td>Digoxin</td>
<td></td>
</tr>
</tbody>
</table>

As a guide, target heart rate should be ≤ 80 beats per minute at rest and ≤ 115 beats per minute with moderate walking.
Assess thromboembolic risk and stroke risk to determine appropriate antithrombotic treatment

<table>
<thead>
<tr>
<th>CHA₂DS₂-VASc</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestive heart failure/LV dysfunction</td>
<td>1</td>
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<tr>
<td>Hypertension</td>
<td>1</td>
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<td>Age ≥ 75 years</td>
<td>2</td>
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<tr>
<td>Diabetes mellitus</td>
<td>1</td>
</tr>
<tr>
<td>Stroke/TIA</td>
<td>2</td>
</tr>
<tr>
<td>Vascular disease (prior MI, peripheral vascular disease)</td>
<td>1</td>
</tr>
<tr>
<td>Age 65–75 years</td>
<td>1</td>
</tr>
<tr>
<td>Sex category (i.e. female gender)</td>
<td>1</td>
</tr>
<tr>
<td><strong>Maximum score</strong></td>
<td><strong>9</strong></td>
</tr>
</tbody>
</table>

N.B. Maximum score is 9 as age is either allocated one or two points.

If the CHADS₂ score is ≥ 2, the patient should be anticoagulated. If a patient has a CHADS₂ score of less than 2, CHA₂DS₂-VASc can be used to further evaluate risk and to guide treatment choice.

**PARAMEDIC STANDING ORDERS**

- If the patient’s systolic blood pressure is **unstable** (less than 100 mm Hg, with signs of hypoperfusion):
  - In Atrial Fibrillation, synchronized cardioversion at 200 J, 300 J, and 360 J or the equivalent biphasic values as per manufacturer).
  - In Atrial Flutter, synchronized cardioversion beginning at 50J.
  - Check rhythm and pulse between each attempted cardioversion.
  - If Cardioversion is warranted, consider use of Sedation for Electrical Therapies.
- **Diltiazem HCL**
  - Heart rate greater than 150 and patient stable but symptomatic:
    - Initial bolus: **0.25 mg/kg slow IV over two (2) minutes.**
    - If inadequate response after 15 minutes, re-bolus **0.35 mg/kg SLOW IV over two (2) minutes.**

**CONTRAINDICATIONS:** Wolff-Parkinson-White Syndrome, second or third degree heart block and sick sinus syndrome (except in the presence of a ventricular pace maker), severe hypotension or cardiogenic shock.

- **Heart rate less than 150 and patient stable but symptomatic:**
  - Contact Medical Control.

**MEDICAL CONTROL MAY ORDER**

- Additional doses of above medications
- **Amiodarone** 150 mg Slow IV/IO over 10 minutes.
- **Metoprolol**: maintenance: 25-100 mg PO q12hr
- **Flecainide**: 50 mg PO BID; do not exceed 300 mg/day
- **Propafenone**: 150 mg PO q8hr;
BRADYCARDIA-ADULT

EMT/EMT-INTERMEDIATE/ADVANCED EMT STANDING ORDERS

- Routine Patient Care.

PARAMEDIC STANDING ORDERS

- If patient is symptomatic (such as altered mental status or ischemia)

THE PATIENT SHOULD BE IMMEDIATELY REFERRED TO HOSPITAL

- **Atropine Sulfate** 0.5 mg IV every three (3) to five (5) minutes up to total dose 3 mg may be considered while waiting for pacer set-up.

MEDICAL CONTROL MAY ORDER

- Additional doses of above medications
- **Norepinephrine** infusion: 0.1mcg/kg/min IV via pump, titrate to goal Systolic Blood Pressure of 90mmHg, OR
- **Dopamine** 2-20 mcg/kg/min IV
- **Epinephrine Infusion** 1-10 mcg/min IV

  For example: mix 1mg of Epinephrine 1:1000 in 250mL of Normal Saline, (15 micro drops/minute = 1 mcg / min.)
BRADYCARDIA-PEDIATRIC

EMT/EMT-INTERMEDIATE/ADVANCED EMT STANDING ORDERS

• Routine Patient Care
• If pulse is less than 60 in a child, AND the patient is severely symptomatic, consider starting Cardiopulmonary Resuscitation (CPR).

PARAMEDIC STANDING ORDERS

• If patient is severely symptomatic:
  • Epinephrine 1:10,000, 0.01 mg/kg IV/IO (max. dose 0.5 mg) or,
  • Atropine 0.02 mg/kg IV (min. single dose 0.1 mg, max. single dose 1 mg). If increased vagal tone or AV block suspected.

MEDICAL CONTROL MAY ORDER

• Additional doses of above medications
• Additional fluid boluses (10-20mL/kg)
• Epinephrine 1:10,000 – 0.01-0.03 mg/kg IV (max. single dose of 0.5 mg)
• Epinephrine Infusion 1:1,000, 0.1-1 mcg/kg/min IV
• For example, mix 1mg of Epinephrine 1:1000 in 250mL of Normal Saline, (15 micro drops/minute = 1 mcg / min.)

THE PATIENT SHOULD BE IMMEDIATELY REFERRED TO HOSPITAL
GENERAL ADULT ASSESSMENT

A Vascular Access
P Cardiac monitor/12-Lead ECG

ECG shows STEMI

HR <50 bpm & ANY of the following:
1. Hypoperfusion
2. Altered mental status
3. Signs of shock

NO

Observe Transport

YES

Consider: ATROPINE 0.5 mg IVP; may repeat q 3-5 min; max dose 3.0 mg

Refractory

Consider: DOPAMINE 5-10 mcg/kg/min IV; titrate to SBP 100 mmHg; max dose 20 mcg/kg/min

THE PATIENT SHOULD BE IMMEDIATELY REFERRED TO HOSPITAL

Acute Coronary Syndrome

Signs of hypotension, AMS, shock

Consider: ATROPINE 0.5 mg IVP; may repeat q 3-5 min; max dose 3.0 mg

THE PATIENT SHOULD BE IMMEDIATELY REFERRED TO HOSPITAL
Red Flag:

- Bradycardia causing symptoms is typically <50/minute. Rhythm should be interpreted in the context of symptoms and pharmacological treatment given only when symptomatic, otherwise monitor and reassess.
- Identifying signs and symptoms of poor perfusion caused by bradycardia are paramount.
- Do not delay pacing while waiting for IV access.
- Do not delay immediately transport to the hospital.
- Hypoxemia is a common cause of bradycardia; be sure to oxygenate the patient and provide ventilatory support as needed.

QI Metrics

- High degree blocks correctly identified.
- Pacer pads on patient if Atropine given.
PEDiATRIC bRADYCARDIA

GENERAL PEDIATRIC ASSESSMENT

Ventilation Management

Bradycardia causing hypotension, altered mental status, poor perfusion or shock?

NO

YES

Identify underlying cause

Blood glucose testing

Consider Vascular Access

Cardiac monitor

Consider Overdose/Poisoning

Monitor, Reassess, Transport to Pediatric Facility

Identify underlying cause

Blood glucose testing

Vascular Access

Cardiac monitor

HR <60 bpm?

NO

YES

CPR

EPINEPHRINE 1:10,000
0.01 mg/kg IV/IO 1:1000
0.1 mg via ETT (max 1.0 mg); repeat q 3-5 min

ATROPINE 0.02 mg/kg
IV/IO (min dose 0.1 mg; max 0.5 mg) may repeat once after 5 min

NS bolus 20 ml/kg; A may repeat up to 60 ml/kg

EPINEPHRINE 1:10,000
0.01 mg/kg IV/IO 1:1000
0.1 mg via ETT (max 1.0 mg); repeat q 3-5 min

ATROPINE 0.02 mg/kg
IV/IO (min dose 0.1 mg; max 0.5 mg) may repeat once after 5 min

Notify Receiving Hospital

Cardiac Arrest

Pulseless
Red Flag:

- Recommended Exam: Mental Status, HEENT, Heart, Lung, Neuro.
- Bradycardia causing symptoms is typically <50/minute. Rhythm should be interpreted in the context of symptoms and pharmacological treatment given only when symptomatic; otherwise, monitor and reassess.
- Identifying signs and symptoms of poor perfusion caused by bradycardia are paramount.
- Hypoxemia is a common cause of bradycardia; be sure to oxygenate the patient and provide ventilatory support as needed.
CARDIAC ARREST (ADULT): ASYSTOLE/PULSELESS ELECTRICAL ACTIVITY

EMT STANDING ORDERS
- Routine Patient Care
- EARLY DEFIBRILLATION.
- Perform CPR until AED device is attached and operable.
- Use AED according to Emergency Cardiovascular Care (ECC) Guidelines or as otherwise noted in these protocols and other advisories.
- Resume CPR when appropriate.

EMT-INTERMEDIATE/ADVANCED EMT STANDING ORDERS
- Consider underlying causes for Asystole/PEA (Pulseless Electrical Activity)
- At all times, minimize interruptions of chest compressions, especially during IV placement.

PARAMEDIC STANDING ORDERS
- Verify Asystole in 2 leads, if possible.
- Consider and treat underlying causes for Asystole/PEA:
  - If cause is unknown and Asystole/PEA persists:
    - Epinephrine 1:10,000 1 mg IV every 3-5 minutes; may substitute Vasopressin 40 UNITS IV in place of first or second dose of epinephrine 1:10,000.

MEDICAL CONTROL MAY ORDER
- Additional doses of above medications.
- Sodium Bicarbonate 1 mEq/kg IV
- Atropine 1 mg IV, repeated to max dose 3 mg.

Note:
REVERSIBLE CAUSES OF CARDIAC ARREST INCLUDE:
- Hypothermia: initiate 2 large bore IVs (warm) normal saline
- Hyperkalemia: Contact Medical Control
- Hypoxia: provide high flow oxygen
- Hypovolemia: 250mL fluid bolus.
- Hydrogen Ion/Acidosis: Contact Medical Control
- Toxins/Tablets: see Toxicology protocol
- Thrombus (Coronary/Pulmonary): Contact Medical Control
- Tension Pneumothorax: Contact Medical Control.
- Tamponade (Pericardial): Contact Medical Control
CARDIAC ARREST (PEDIATRIC): ASYSTOLE/PULSELESS ELECTRICAL ACTIVITY

EMT/EMT-INTERMEDIATE/ADVANCED EMT STANDING ORDERS

- Routine Patient Care—with focus on CPR
- Ventilate with 100% oxygen
- If unable to ventilate child after repositioning of airway: assume upper airway obstruction and follow Pediatric Upper Airway Obstruction Protocol.

**EARLY DEFIBRILLATION**

Use AED according to the guidelines of the ECC or as otherwise noted in these protocols and other advisories.

PARAMEDIC STANDING ORDERS

- Consider treating for reversible causes.
- **Epinephrine**:
  - For **Bradycardia**: 0.01 mg/kg (1:10,000) IV every 3-5 minutes.
  - For **Asystole or PEA**: 0.01 mg/kg (1:10,000) IV every 3-5 minutes.
- **Epinephrine** infusion: initial dose, 0.1 mcg/kg/min IV. Titrate to desired effect to maximum dose of 1 mcg/kg/min.
  
  For example, mix 1mg of Epinephrine 1:1000 in 250mL of Normal Saline, (15 micro drops/minute = 1 mcg / min.)

MEDICAL CONTROL MAY ORDER

- Additional doses of above medications.
- **Sodium Bicarbonate** 1 mEq/kg IV
- **Atropine** 0.02mg/kg IV (minimum single dose 0.1mg, maximum combined doses 1 mg.)
- All other treatment modalities based on suspected etiology for cardiopulmonary arrest.

Note: **REVERSIBLE CAUSES OF CARDIAC ARREST INCLUDE:**

- Hypothermia: initiate 2 large bore IVs (warm) normal saline
- Hyperkalemia: Contact Medical Control
- Hypoxia: provide high flow oxygen
- Hypovolemia: 250mL fluid bolus.
- Hydrogen Ion/Acidosis: Contact Medical Control
- Toxins/Tablets: see Toxicology protocol
- Thrombus (Coronary/Pulmonary): Contact Medical Control
- Tension Pneumothorax: Perform needle chest decompression.
- Tamponade (Pericardial): Contact Medical Control
CARDIAC ARREST (ADULT): VENTRICULAR FIBRILLATION/PULSELESS VENTRICULAR TACHYCARDIA

EMT/EMT-INTERMEDIATE STANDING ORDERS
• Routine Patient Care
• Perform CPR until defibrillator is attached and operable.
• Use AED according to the ECC guidelines or as otherwise noted in these protocols and other advisories
• Resume CPR when appropriate.

ADVANCED EMT STANDING ORDERS
• Minimize interruptions of chest compressions for IV placement.

PARAMEDIC STANDING ORDERS
• Document presenting cardiac rhythm in two separate leads, if possible.
• Defibrillation when available, with minimum interruption in chest compressions (use 360 joules for monophasic and 120 – 200 joules for biphasic defibrillators); then CPR for 5 cycles/2 minutes; then rhythm check; Charge defibrillator while performing chest compressions to minimize hands-off-time.
• Consider Epinephrine (1:10,000) 1mg IV; repeat every 3 – 5 minutes. May substitute Vasopressin 40 units IV in place of first or second dose of epinephrine 1:10,000.
• Continue CPR and defibrillate (each shock at 360J monophasic equivalent) per ECC guidelines if ventricular fibrillation/ventricular tachycardia is persistent.
• Consider Amiodarone 300 mg slow IV push.

MEDICAL CONTROL MAY ORDER
• Additional doses of above medications
• Sodium Bicarbonate 1 mEq/kg IV.
• Magnesium Sulfate 1 – 2 grams IV over 5 minutes, in torsades de pointes or suspected hypomagnesemic state or refractory ventricular fibrillation/ventricular tachycardia.
• Amiodarone 150 mg. slow IV if one dose already given or 300 mg slow IV if not already given.
• Lidocaine 1.5 mg/kg IV; subsequent dosage: 0.5 to 0.75 mg/kg IV every 3 – 5 minutes to a total dose of 3 mg/kg IV.

NOTE:
The need for early defibrillation is clear and should have the highest priority. Since these patients will all be in cardiopulmonary arrest, use of adjunctive equipment should not divert attention or effort from Basic Cardiac Life Support (BCLS) resuscitative measures, early defibrillation and Advanced Cardiac Life Support (ACLS). Remember: rapid defibrillation and high quality CPR is the major determinant of survival.

NOTE:
• Early CPR and early defibrillation are the most effective therapies for cardiac arrest care.
• Minimize interruptions in chest compression, as pauses rapidly return the blood pressure to zero and stop perfusion to the heart and brain.
• Switch compressors at least every two minutes to minimize fatigue.
• Perform “hands on defibrillation.”
  o Compress when charging and resume compressions immediately after the shock is delivered.
• Do not hyperventilate as it increases intrathoracic pressure and decreases blood return to the heart. Ventilate at a rate of 8 – 10 breaths per minutes, with enough volume to produce adequate chest rise.
CARDIAC ARREST (PEDIATRIC): VENTRICULAR FIBRILLATION/PULSELESS VENTRICULAR TACHYCARDIA

EMT/EMT-INTERMEDIATE/ADVANCED EMT STANDING ORDERS
- Routine Patient Care—with focus on high quality CPR
- Apply AED and use as soon as possible (with minimum interruption of chest compressions). From birth to age 8 years use pediatric AED pads.
- If pediatric AED pads are unavailable, providers may use adult AED pads, provided the pads do not overlap.
- If unable to ventilate child after repositioning of airway, assume upper airway obstruction and follow Pediatric Upper Airway Obstruction Protocol.
- Consider treatable causes

PARAMEDIC STANDING ORDERS
- Defibrillate once at 2-4J/kg.
- **Epinephrine:** 0.01mg/kg IV (1:10,000, 0.1mL/kg); repeat every 3-5 minutes.
- Defibrillate 4-10J/kg (do not exceed 10J/kg) every 2 minutes.
- **Amiodarone** 5 mg/kg IV
- Defibrillate 4J/kg 30-60 seconds after each medication.

MEDICAL CONTROL MAY ORDER
- Additional doses of above medications
- **Sodium Bicarbonate** 1 mEq/kg IV.
- All other treatment modalities based upon suspected cause of VT/FT.

NOTE:
The need for early defibrillation is clear and should have the highest priority. Since these patients will all be in cardiopulmonary arrest, use of adjunctive equipment should not divert attention or effort from Basic Cardiac Life Support (BCLS) resuscitative measures, early defibrillation and Advanced Cardiac Life Support (ACLS). Remember: rapid defibrillation and high quality CPR is the major determinant of survival.

NOTE:
- Early CPR and early defibrillation are the most effective therapies for cardiac arrest care.
- Minimize interruptions in chest compression, as pauses rapidly return the blood pressure to zero and stop perfusion to the heart and brain.
- Switch compressors at least every two minutes to minimize fatigue.
- Perform “hands on defibrillation.”
  - Compress when charging and resume compressions immediately after the shock is delivered.
- Do not hyperventilate as it increases intrathoracic pressure and decreases blood return to the heart. Ventilate at an appropriate rate, with enough volume to produce adequate chest rise.
CARDIAC ARREST (NON-TRAUMATIC) (ADULT CCC CPR)

GENERAL ADULT ASSESSMENT

Meets criteria for Prehospital Death

If witnessed by EMS or CPR in progress and patient is unresponsive with no pulse, begin Continuous Chest Compressions (CCC) Push hard, Push fast (≥100/min)

If unwitnessed by EMS or no CPR in progress perform 2 min of CCC

Apply AED and defibrillate
Insert NPA or OPA and begin BVM at 8 BPM
Apply cardiac monitor

Rhythm shockable?

YES
Defibrillate
Continue CPR for 2 min
Vascular Access

NO
Asystole/PEA

Rhythm shockable?

YES
Defibrillate if prompted (AED)
Continue CPR for 2 min
EPINEPHRINE 1.0 mg IV/IO q 3-5 min ETT Administration requires 2 to 2.5 times

NO
Use Asystole/PEA side as indicated
Check pulse, if organized rhythm
If patient remains unresponsive to resuscitation efforts, consider Termination of Resuscitation Protocol

Vascular Access
EPINEPHRINE 1.0 mg IV/IO q 3-5 min ETT Administration requires 2 to 2.5 times
Consider Extraglottic Airway Device
Consider Endotracheal Intubation

EPINEPHRINE 1.0 mg IV/IO q 3-5 min ETT Administration requires 2 to 2.5 times
Red Flag:

- Efforts should be directed at high quality and continuous compressions with limited interruptions and early defibrillation when indicated.
- Consider early IO placement if IV is difficult.
- DO NOT HYPERVENTILATE.
- Reassess and document ETT placement using auscultation and ETCO₂ capnography.
- Switch compressors every two minutes.
- Try to maintain patient modesty.
- Mechanical chest compression devices should be used if available in order to provide for consistent uninterrupted chest compressions and crew safety.

H’s & T’s (reversible causes)

- Hypovolemia – Volume infusion
- Hypoxia – Oxygenation & ventilation, CPR
- Hydrogen ion (acidosis) – Ventilation, CPR
- Hypo/Hyperkalemia – Calcium Chloride, Glucose, Sodium Bicarbonate, Albuterol
- Hypothermia - Warming
- Tension pneumothorax – Needle decompression
- Tamponade, cardiac – Volume infusion
- Toxins – Agent specific antidote
- Thrombosis, pulmonary – Volume infusion
- Thrombosis, coronary – Emergent PCI
GENERAL PEDIATRIC ASSESSMENT

Refer to Termination of Resuscitation or DNR Protocol as Appropriate

Meets Criteria for Prehospital Death Determination or DNR/POLST present?

IF HYPOXIA IS THE CAUSE OF THE ARREST, EARLY VENTILATION IS RECOMMENDED

Begin Age Appropriate CPR Push Hard (4 cm Infant 5 cm in Children) Push Fast (≥ 100/min)

Apply AED and Defib if Prompted

Apply Cardiac Monitor

Rhythm Shockable?

VF/VT

Defibrillate at 2 J/kg

Continue CPR for 2 Minutes

IV Access

Rhythm Shockable?

Asystole/PEA

Continue CPR for 2 Minutes

IV Access

Defibrillate at 0.01 mg/kg 1:10,000 IV or 0.1 mg/kg 1:1,000 ETT Every 3-5 Minutes

Rhythm Shockable?

Defibrillate if Prompted (AED)

Defibrillate at 4 J/kg Not To Exceed Adult Dose

Continue CPR for 2 Minutes

EPINEPHRINE 0.01 mg/kg 1:10,000 IV or 0.1 mg/kg 1:1,000 ETT Every 3-5 Minutes

Rhythm Shockable?

Address H’s & T’s

Rhythm Shockable?
**H’s & T’s (reversible causes)**

- Hypovolemia – Volume infusion
- Hypoxia – Oxygenation & ventilation, CPR
- Hydrogen ion (acidosis) – Ventilation, CPR
- Hypo/Hyperkalemia – Calcium Chloride, Glucose, Sodium Bicarbonate, Albuterol
- Hypothermia - Warming
- Tension pneumothorax – Needle decompression
- Tamponade, cardiac – Volume infusion
- Toxins – Agent specific antidote
- Thrombosis, pulmonary – Volume infusion
- Thrombosis, coronary – Emergent PCI

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<table>
<thead>
<tr>
<th>Step</th>
<th>Action</th>
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<tbody>
<tr>
<td>E</td>
<td>Defibrillate 1 Prompted (AED)</td>
</tr>
<tr>
<td>P</td>
<td>Defibrillate at 4 J/kg Not To Exceed Adult Dose</td>
</tr>
<tr>
<td>E</td>
<td>Continue CPR for 2 Minutes</td>
</tr>
<tr>
<td>E</td>
<td>AMIODARONE 5 mg/kg IV May Repeat Once After 5th Shock Address H’s &amp; T’s</td>
</tr>
</tbody>
</table>

**The Patient Should Be Immediately Referred to Hospital**

**Use VF/VT Side as Indicated**

**If Patient Remains Unresponsive to Resuscitation Efforts Consider Termination of Resuscitation Protocol**

**Use Asystole/PEA Side as Indicated**

**Check Pulse if Organized Rhythm**
SUPRAVENTRICULAR TACHYCARDIA — ADULT

EMT/EMT-INTERMEDIATE/ADVANCED EMT STANDING ORDERS

- Routine Patient Care

PARAMEDIC STANDING ORDERS

- Vagal Maneuvers: Valsalva’s and/or cough.
- If Systolic BLOOD PRESSURE is unstable (less than 100mm Hg): Synchronized cardioversion at 50 J, 100 J, 200 J, 300 J and 360 J or the equivalent biphasic values as per manufacturer. Check rhythm and pulse between each attempted cardioversion.
- If cardioversion is warranted, consider Sedation for Electrical Therapy

- **Adenosine** 6 mg rapid IV over 1-3 seconds. If previous dose failed to resolve rhythm disturbance, **Adenosine** 12mg rapid IV over 1-3 seconds. Repeat **Adenosine** 12 mg rapid IV over 1-3 seconds if previous doses failed to resolve rhythm disturbance.

**Note:** Follow all Adenosine with a 20 mL normal saline bolus and elevate extremity.

MEDICAL CONTROL MAY ORDER

- Additional doses of above medications
- Administration of Diltiazem HCL:
  - Initial bolus: 0.25 mg/kg IV over two (2) minutes.
  - If inadequate response after 15 minutes, **re-bolus** 0.35 mg/kg IV over two (2) minutes.

**CONTRAINDICATIONS:** Wolff-Parkinson-White Syndrome, second or third degree heart block and sick sinus syndrome (except in the presence of a ventricular pace maker), severe hypotension or cardiogenic shock.

**OR**

- **Amiodarone** 150 mg IV slowly over 10 minutes.
SUPRAVENTRICULAR TACHYCARDIA — PEDIATRIC

EMT/EMT-INTERMEDIATE/ADVANCED EMT STANDING ORDERS

• Routine Patient Care
• Tachycardia is related to acute injury or volume loss?

PARAMEDIC STANDING ORDERS

• IV Normal Saline (KVO). If hypovolemic component is suspected, administer 20 mL/kg IV Bolus of Normal Saline.

MEDICAL CONTROL MAY ORDER

• Additional doses of above medications
• Synchronized cardioversion 0.5 joules/kg for symptomatic patients. Subsequent cardioversion may be done at up to 1 joule/kg. If cardioversion is warranted, consider administration of Sedation for Electrical Therapy, per protocol.
• Adenosine 0.1 mg/kg rapid IV/. If no effect, repeat Adenosine 0.2 mg/kg Rapid IV push. MAXIMUM single dose of Adenosine must not exceed 12 mg.
• Consider Vagal maneuvers.

Red Flag:
Synchronized cardioversion should be considered for only those children whose heart rate is in excess of 220, and who demonstrate one or more of the following signs of hypoperfusion: Decreased level of consciousness, weak and thready pulses, capillary refill time of more than 4 seconds, or no palpable BLOOD PRESSURE.

Red Flag:
REMINDER: Vagal maneuvers may precipitate asystole and therefore should be employed with caution in the field and only in a cardiac-monitored child with IV access.
VENTRICULAR TACHYCARDIA WITH PULSES—ADULT & PEDIATRIC

EMT/EMT-INTERMEDIATE/ADVANCED EMT STANDING ORDERS

- Routine Patient Care

PARAMEDIC STANDING ORDERS

- If Systolic BLOOD PRESSURE is unstable (less than 100mm Hg): synchronized cardioversion at 100 J, 200 J, 300 J and 360 J or the equivalent biphasic values as per manufacturer. Check rhythm and pulse between each attempted cardioversion.
- In Pediatric patients, synchronized cardioversion per Pediatric Color-Coded Appendix.
- If cardioversion is warranted, see Sedation for Electrical Therapy.
- If systolic BLOOD PRESSURE is stable (greater than or equal to 100mm Hg) administer **Amiodarone** 150 mg in 10 cc Normal Saline, slow IV over 8-10 minutes.
- In Pediatric patients, **Amiodarone** dose per Pediatric Color-Coded Appendix.

MEDICAL CONTROL MAY ORDER

- Additional doses of above medications or attempts at cardioversion
- **Magnesium Sulfate** (for Torsades de Pointes or suspected hypomagnesemic state or severe refractory VENTRICULAR TACHYCARDIA) 1 – 2 grams IV over 5 minutes.
- CONTRAINDICATIONS: Heart Block, renal disease.
- **Amiodarone infusion** 1 mg/min IV.
- For example: 100mg/100ml – 1mg/minute
- **Lidocaine** 1 – 1.5 mg/kg IV; subsequent dosage: 0.5 – 0.75 mg/kg IV every 3 – 5 minutes to a total dose of 3 mg/kg. If dysrhythmia is successfully converted after administration of Lidocaine bolus, consider IV infusion of Lidocaine 2 – 4 mg/min.
- **Adenosine** 6 mg or 12 mg IV push; in selected cases ONLY.
Tachycardia / Stable (Normal Mental Status, Palpable Radial Pulse)

**GENERAL ADULT ASSESSMENT**

- APPLY CARDIAC MONITOR
- 12-Lead ECG
- Vascular Access

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**Narrow Complex ≤0.11 Sec**

- Vagal Maneuvers
  - NO
  - Administer ADENOSINE 6 mg rapid IVP
  - NO
  - Administer ADENOSINE 12 mg rapid IVP

---

**Wide Complex ≥0.12 Sec**

---

**Undifferentiated Monomorphic VT Suspected to be SVT with Aberrancy**

- AMIODARONE 150 mg in 50 cc NS over 10 min
  - NO
  - MAGNESIUM SULFATE 2.0 gm IV in 50 cc NS over 10 min
  - NO
  - YES
  - Synchronized Cardioversion
    - Consider sedation: ETOMIDATE 0.15 mg/kg IV
  - NO

---

**Regular Monomorphic VT**

---

**Torsades de Pointes**

---

**Continue General Patient Care**

THE PATIENT SHOULD BE IMMEDIATELY REFERRED TO HOSPITAL

**Red Flag:**

- Carefully monitor patients as you treat them; stable tachycardia may convert to unstable rhythms/conditions quickly.
- Sedate patients prior to cardioversion, if time allows.
Tachycardia / Unstable (Mental Status Changes, No Palpable Radial Pulse)

**Narrow Complex ≤0.11 Sec**
- If IV established, administer Adenosine 12 mg rapid IVP
- Rhythm change?
  - NO
  - Synchronized Cardioversion Consider sedation: ETOMIDATE 0.15 mg/kg IV
    - Rhythm change?
      - NO
      - Magnesium Sulfate 2.0 gm IV in 50 cc NS over 10 min
        - Rhythm change?
          - NO
          - Repeat Synchronized Cardioversion; assess need for repeat sedation
            - Rhythm change?
              - NO

**Wide Complex ≥0.12 Sec**
- APPLY CARDIAC MONITOR
- Vascular Access
- GENERAL ADULT ASSESSMENT

**Monomorphic VT**
- Defibrillation Consider sedation: ETOMIDATE 0.15 mg/kg IV
  - Rhythm change?
    - NO
    - Repeat Synchronized Cardioversion Consider sedation: ETOMIDATE 0.15 mg/kg IV
      - Rhythm change?
        - NO
        - AMIODARONE 150 mg in 50 cc NS over 10 min
          - Rhythm change?

The patient should be immediately referred to hospital.
Pediatric Tachycardia / Stable (Normal Mental Status, Palpable Radial Pulse)

Red Flag:
- Recommended exam: Mental Status, Skin, Heart, Lungs, Abdomen, Back, Extremities, Neuro.
- Carefully monitor patients as you treat them; stable tachycardias may convert to unstable rhythms/conditions quickly.
- Sedate patients prior to cardioversion, if time allows.
- The most common tachyarrhythmia in children is sinus.
Pediatric Tachycardia / Unstable (Mental Status Changes, No Palpable Radial Pulse)

Narrow Complex ≤0.11 Sec

- Cardiac monitor
- Vascular Access

If IV established, administer ADENOSINE 0.2 mg/kg rapid IV push not to exceed 12 mg

Rhythm change?

- Synchronized Cardioversion start at 0.5 to 1 J/kg; may use 2 J/kg if unsuccessful; Consider sedation ETOMIDATE 0.15 mg/kg IV

Wide Complex ≥0.12 Sec

Torsades de Pointes

- Defibrillate at 2 J/kg increasing to 4 J/kg; Consider sedation ETOMIDATE 0.15 mg/kg IV

Rhythm change?

- MAGNESIUM SULFATE 25 mg/kg IV in 50 cc NS over 10 min

Monomorphic VT

- Synchronized Cardioversion Start at 0.5 to 1 J/kg; may use 2 J/kg if unsuccessful; or defibrillate at 2 J/kg increasing to 4 J/kg; Consider sedation ETOMIDATE 0.15 mg/kg IV

Rhythm change?

- AMIODARONE 5.0 mg/kg in 50 cc NS over 20 min

Rhythm change?

- Repeat Synchronized Cardioversion or defibrillate if VT not resolved; assess need for repeat sedation; repeat AMIODARONE

The Patient Should Be Immediately Referred to Hospital
Hypertension

- Adult having blood pressure measured
  - Measuring blood pressure
    - Hypertension in pregnancy
    - Hypertension not diagnosed
    - Diagnosis
      - Hypertension diagnosed
        - Assessing cardiovascular risk and target organ damage
          - Management of hypertension
MEASURING BLOOD PRESSURE

Because automated devices may not measure blood pressure accurately if there is pulse irregularity (for example, due to atrial fibrillation), palpate the radial or brachial pulse before measuring blood pressure. If pulse irregularity is present, measure blood pressure manually using direct auscultation over the brachial artery. When measuring blood pressure in the clinic, standardise the environment and provide a relaxed, temperate setting, with the person quiet and seated, and their arm outstretched and supported. Appropriate cuff size for the person's arm is used.

Postural hypotension

In people with symptoms of postural hypotension (falls or postural dizziness): measure blood pressure with the person either supine or seated. Measure blood pressure again with the person standing for at least 1 minute prior to measurement.

If the systolic blood pressure falls by 20 mmHg or more when the person is standing:

- review medication
- measure subsequent blood pressures with the person standing
- consider referral to specialist care if symptoms of postural hypotension persist.

Measuring the clinic blood pressure

When considering a diagnosis of hypertension, measure blood pressure in both arms. If the difference in readings between arms is more than 20 mmHg, repeat the measurements.

If the difference in readings between arms remains more than 20 mmHg on the second measurement, measure subsequent blood pressures in the arm with the higher reading.

If blood pressure measured in the clinic is 140/90 mmHg or higher:

Take a second measurement during the consultation.

If the second measurement is substantially different from the first, take a third measurement.

Record the lower of the last two measurements as the clinic blood pressure.
Definitions and classification of office blood pressure levels (mmHg)

<table>
<thead>
<tr>
<th>Category</th>
<th>Systolic</th>
<th>Diastolic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optimal</td>
<td>&lt;120</td>
<td>and  &lt;80</td>
</tr>
<tr>
<td>Normal</td>
<td>120–129</td>
<td>and/or 80–84</td>
</tr>
<tr>
<td>High normal</td>
<td>130–139</td>
<td>and/or 85–89</td>
</tr>
<tr>
<td>Grade 1 hypertension</td>
<td>140–159</td>
<td>and/or 90–99</td>
</tr>
<tr>
<td>Grade 2 hypertension</td>
<td>160–179</td>
<td>and/or 100–109</td>
</tr>
<tr>
<td>Grade 3 hypertension</td>
<td>≥180</td>
<td>and/or ≥110</td>
</tr>
<tr>
<td>Isolated systolic hypertension</td>
<td>≥140</td>
<td>and  &lt;90</td>
</tr>
</tbody>
</table>

The blood pressure (BP) category is defined by the highest level of BP, whether systolic or diastolic. Isolated systolic hypertension should be graded 1, 2, or 3 according to systolic BP values in the ranges indicated.

Stratification of total CV risk

<table>
<thead>
<tr>
<th>Other risk factors, asymptomatic organ damage or disease</th>
<th>Blood Pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>High normal SBP 130–139 or DBP 85–89</td>
</tr>
<tr>
<td>No other RF</td>
<td>• No BP intervention</td>
</tr>
<tr>
<td>1–2 RF</td>
<td>• Lifestyle changes</td>
</tr>
<tr>
<td></td>
<td>• No BP intervention</td>
</tr>
<tr>
<td>2+3 RF</td>
<td>• Lifestyle changes</td>
</tr>
<tr>
<td></td>
<td>• No BP intervention</td>
</tr>
<tr>
<td>OD, CKD stage 3 or diabetes</td>
<td>• Lifestyle changes</td>
</tr>
<tr>
<td></td>
<td>• No BP intervention</td>
</tr>
<tr>
<td>Symptomatic CVD, CKD stage ≥4 or diabetes with OD/RFs</td>
<td>• Lifestyle changes</td>
</tr>
<tr>
<td></td>
<td>• No BP intervention</td>
</tr>
</tbody>
</table>

BP = blood pressure; CKD = chronic kidney disease; CV = cardiovascular; CVD = cardiovascular disease; DBP = diastolic blood pressure; HT = hypertension; OD = organ damage; RF = risk factor; SBP = systolic blood pressure.

The initial evaluation of a patient with hypertension should

(i) confirm the diagnosis of hypertension,
(ii) detect causes of secondary hypertension, and
(iii) assess CV risk, OD and concomitant clinical conditions.

This calls for BP measurement, medical history including family history, physical examination.

66
Office blood pressure measurement

<table>
<thead>
<tr>
<th>When measuring BP in the office, care should be taken:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• To allow the patients to sit for 3–5 minutes before beginning BP measurements.</td>
</tr>
<tr>
<td>• To take at least two BP measurements, in the sitting position, spaced 1–2 min apart, and additional measurements if the first two are quite different. Consider the average BP if deemed appropriate.</td>
</tr>
<tr>
<td>• To take repeated measurements of BP to improve accuracy in patients with arrhythmias, such as atrial fibrillation.</td>
</tr>
<tr>
<td>• To use a standard bladder (12–13 cm wide and 35 cm long), but have a larger and a smaller bladder available for large (arm circumference &gt;32 cm) and thin arms, respectively.</td>
</tr>
<tr>
<td>• To have the cuff at the heart level, whatever the position of the patient.</td>
</tr>
<tr>
<td>• When adopting the auscultatory method, use phase I and V (dissappearance) Korotkoff sounds to identify systolic and diastolic BP, respectively.</td>
</tr>
<tr>
<td>• To measure BP in both arms at first visit to detect possible differences. In this instance, take the arm with the higher value as the reference.</td>
</tr>
<tr>
<td>• To measure at the first visit, BP 1 and 3 min after assumption of the standing position in elderly subjects, diabetic patients, and in other conditions in which orthostatic hypotension may be frequent or suspected.</td>
</tr>
<tr>
<td>• To measure, in case of conventional BP measurement, heart rate by pulse palpation (at least 30 s) after the second measurement in the sitting position.</td>
</tr>
</tbody>
</table>

Orthostatic hypotension—defined as a reduction in SBP of ≥20 mmHg or in DBP of ≥10 mmHg within 3 min of standing
Offer antihypertensive drug treatment to people aged under 80 years with stage 1 hypertension clinic blood pressure 140/90 mmHg or higher and subsequent ambulatory blood pressure monitoring daytime average or home blood pressure monitoring average blood pressure 135/85 mmHg or higher who have one or more of the following:

- target organ damage
- established cardiovascular disease
- renal disease
- diabetes
- 10-year cardiovascular risk equivalent to 20% or greater.

Offer antihypertensive drug treatment to people of any age with stage 2 hypertension clinic blood pressure 160/100 mmHg or higher and subsequent ambulatory blood pressure monitoring daytime average or home blood pressure monitoring average blood pressure 150/95 mmHg or higher.

A 12-lead ECG should be part of the routine assessment of all hypertensive patients. Its sensitivity in detecting LVH is low but, nonetheless, LVH detected by the Sokolow-Lyon index (SV1 + RV5 >3.5 mV), the modified Sokolow-Lyon index (largest S-wave + largest R-wave >3.5 mV), RaVL >1.1 mV.
Where possible, recommend treatment with drugs taken only once a day.

Offer people with isolated systolic hypertension (systolic blood pressure 160 mmHg or more) the same treatment as people with both raised systolic and diastolic blood pressure.

Antihypertensive treatment in the elderly (including one in hypertensive patients aged 80 years or more) is recommended when SBP is ≥ 160 mmHg.

**Do not combine** an ACE inhibitor with an ARB to treat hypertension.

---

**CHOOSING ANTIHYPERTENSIVE DRUG TREATMENT**

### Person aged under 55 with hypertension

**Step 1**

**ACE inhibitor or ARB**

Beta-blockers are not a preferred initial therapy for hypertension. However, beta-blockers may be considered in younger people, particularly:

- those with an intolerance or contraindication to ACE inhibitors and angiotensin II receptor antagonists or
- women of child-bearing potential or
- people with evidence of increased sympathetic drive.

### Person aged over 55 or black person of African or Caribbean family origin of any age with hypertension

**Step 1**

**CCB (calcium channel blocker)**

If a CCB is not suitable, for example because of oedema or intolerance, or if there is evidence of heart failure or a high risk of heart failure, offer a thiazide-like diuretic.

If diuretic treatment is to be initiated or changed, offer a thiazide-like diuretic, such as chlortalidone (12.5–25.0 mg once daily) or indapamide (1.5 mg modified-release once daily or 2.5 mg once daily) in preference to a conventional thiazide diuretic such as bendroflumethiazide.
Step 2

If blood pressure is not controlled by step 1 treatment, offer step 2 treatment with a CCB in combination with either an ACE inhibitor or an ARB.

If a CCB is not suitable for step 2 treatment, for example because of oedema or intolerance, or if there is evidence of heart failure or a high risk of heart failure, offer a thiazide-like diuretic, such as chlortalidone (12.5–25.0 mg once daily) or indapamide (1.5 mg modified-release once daily or 2.5 mg once daily).

For black people of African or Caribbean family origin, consider an ARB in preference to an ACE inhibitor, in combination with a CCB.

If therapy is initiated with a beta-blocker and a second drug is required, add a CCB rather than a thiazide-like diuretic to reduce the person's risk of developing diabetes.

Step 3

If treatment with three drugs is required, the combination of ACE inhibitor or ARB, CCB and thiazide-like diuretic such as chlortalidone (12.5–25.0 mg once daily) or indapamide (1.5 mg modified-release once daily or 2.5 mg once daily) should be used.

Regard clinic blood pressure that remains higher than 140/90 mmHg after treatment with the optimal or best tolerated doses of an ACE inhibitor or an ARB plus a CCB plus a diuretic as resistant hypertension, and consider adding a fourth antihypertensive drug and/or seeking expert advice.
Step 4

Resistant hypertension

Consider further diuretic therapy with low-dose spironolactone (25 mg once daily) if the blood potassium level is normal. Use particular caution in people with a reduced eGFR because they have an increased risk of hyperkalaemia.

If further diuretic therapy for resistant hypertension at step 4 is not tolerated, or is contraindicated or ineffective, consider an alpha- or beta-blocker.

If blood pressure remains uncontrolled with the optimal or maximum tolerated doses of four drugs, seek expert advice if it has not yet been obtained.
While there is consensus that drug treatment of severe hypertension in pregnancy (>160 for SBP or >110 mmHg for DBP) is required and beneficial.

Consider early initiation of antihypertensive treatment at values ≥140/90 mmHg in women with

(i) gestational hypertension (with or without proteinuria),
(ii) pre-existing hypertension with the superimposition of gestational hypertension or
(iii) hypertension with asymptomatic OD or symptoms at any time during pregnancy.

The recommendations to use methyldopa, labetalol and nifedipine as the only calcium antagonist really tested in pregnancy can be confirmed. Beta-blockers (possibly causing fetal growth retardation if given in early pregnancy) and diuretics (in pre-existing reduction of plasma volume) should be used with caution.

Symptoms of pre-eclampsia

Tell women to seek advice from a healthcare professional immediately if they experience any of:
- severe headache
- problems with vision such as blurring or flashing before the eyes
- severe pain just below the ribs
- vomiting
- sudden swelling of the face, hands or feet.

Mild hypertension (blood pressure 140/90–149/99 mmHg)

Do not treat mild hypertension.

Measure blood pressure at least 4 times a day.

Test kidney function, electrolytes, transaminases and bilirubin 2 times a week.

Moderate hypertension (blood pressure 150/100–159/109 mmHg)

Treat moderate hypertension with first-line oral labetalol to keep blood pressure below 150/80–100 mmHg. Offer treatment other than labetalol only after considering side-effect profiles for the woman, fetus and newborn baby. Alternatives include methyldopa and nifedipine.

Severe hypertension (blood pressure 160/110 mmHg or higher)

Admit women with severe hypertension to hospital until blood pressure is 159/109 mmHg or lower.

Treat with first-line oral labetalol to keep blood pressure below 150/80–100 mmHg. Offer treatment other than labetalol only after considering side-effect profiles for the woman, fetus and newborn baby. Alternatives include methyldopa and nifedipine.
Hypertensive emergencies are defined as large elevations in SBP or DBP (>180 mmHg or >120 mmHg, respectively) associated with impending or progressive OD (organ damage), such as major neurological changes, hypertensive encephalopathy, cerebral infarction, intracranial haemorrhage, acute LV failure, acute pulmonary oedema, aortic dissection, renal failure, or eclampsia.

Isolated large BP elevations without acute OD (hypertensive urgencies)—often associated with treatment discontinuation or reduction as well as with anxiety—should not be considered an emergency but treated by reinstitution or intensification of drug therapy and treatment of anxiety.

Current treatment is founded on agents that can be administered by intravenous infusion and titrated, and so can act promptly but gradually in order to avoid excessive hypotension and further ischaemic OD. Labetalol, sodium nitroprusside, nicardipine, nitrates and furosemide are among the intravenous agents most usually employed but in these severely ill patients, treatment should be individualized.
CONGESTIVE HEART FAILURE (PULMONARY EDEMA)

EMT/EMT-INTERMEDIATE/ADVANCED EMT STANDING ORDERS

• Routine Patient Care

PARAMEDIC STANDING ORDERS

• **Nitroglycerin** 0.4-0.8mg (1/150 gr.) tablet/spray, sublingual
  • SBP must be >120 mm Hg
  • May be repeated every 5 minutes, as dictated by BP.

  • Continuous positive airway pressure (CPAP) assistance, if not contraindicated, and if nebulizer therapy can be continued with the CPAP device.

MEDICAL CONTROL MAY ORDER

• Additional doses of above medications
  • **Furosemide** 20-40mg IV, or 40-80mg IV if patient is already on diuretics.
  • **Norepinephrine** infusion: 0.1mcg/kg/min IV via pump, titrate to goal Systolic Blood Pressure of 90mmHg, OR
  • **Dopamine** 2-20 mcg/kg/min IV

**Red Flag – CAUTION:**
Avoid nitroglycerin in ALL patients who have used a phosphodiesterase inhibitor such as: **sildenafil** (Viagra, Revatio), **vardenafil** (Levitra, Staxyn), **tadalafil** (Cialis, Adcirca) which are used for erectile dysfunction and pulmonary hypertension within the last **48 HOURS**. Also avoid use in patients receiving intravenous epoprostenol (Flolan) which is also used for pulmonary hypertension.
POST RESUSCITATIVE CARE/ROSC — ADULT

EMT/EMT-INTERMEDIATE/ADVANCED EMT STANDING ORDERS

- Routine Patient Care

PARAMEDIC STANDING ORDERS

- Consider treatable causes such as overdose, cardiogenic shock and STEMI.
- Manage dysrhythmias according to specific protocols.
- Perform a 12-lead ECG; If STEMI is present and the patient is stable enough follow the Department – approved STEMI POE plan. Consult with medical control if questions arise.
- Begin induced therapeutic hypothermia, but do not delay transport
- **Norepinephrine** infusion: 0.1 mcg/kg/min IV via pump, titrate to goal Systolic Blood Pressure of 90 mmHg, OR
  - **Dopamine** 2-20 mcg/kg/min IV

MEDICAL CONTROL MAY ORDER

- Additional doses of above medications
- **Epinephrine Infusion** - Administer 1 mcg to 10 mcg per minute IV.
  
  For example, mix 1 mg of 1:1000 Epinephrine in 250 ml Normal Saline, then 15 micro drops/minute = 1 mcg / min
- **Amiodarone** bolus as ordered followed by 1 mg/min IV drip.
  
  For example: 100 mg/100 ml - 1 mg/minute.
- **Lidocaine** 1-1.5 mg/kg IV followed by drip at 2-4 mg/min.

Red Flag:

REMINDER: This is an extremely unstable period. The patient should be monitored closely and frequently. Recurrent dysrhythmias, hypotension and re-arrest are not uncommon occurrences. Avoid hyperthermia and hyperventilation.

Red Flag:

Avoid hyperoxygenation; oxygen administration should be titrated to patient condition, and withheld unless evidence of hypoxemia, dyspnea, or an SpO2 <94%, especially in the presence of a suspected CVA/TIA or ACS.
RESPIRATORY DISEASES
RHINITIS (COMMON COLD)

Rhinitis, which occurs most commonly as allergic rhinitis, is an inflammation of the nasal membranes that is characterized by sneezing, nasal congestion, nasal itching, and rhinorrhea, in any combination. Although allergic rhinitis itself is not life-threatening (unless accompanied by severe asthma or anaphylaxis), morbidity from the condition can be significant.

**Signs and Symptoms**

- Sneezing
- Itching: Nose, eyes, ears, palate
- Rhinorrhea
- Postnasal drip
- Congestion
- Anosmia
- Headache
- Earache
- Tearing
- Red eyes
- Eye swelling
- Fatigue
- Drowsiness
- Malaise

Complications of this allergic rhinitis include the following:

- Acute or chronic sinusitis
- Otitis media
- Sleep disturbance or apnea
- Dental problems (overbite): Caused by excessive breathing through the mouth
- Palatal abnormalities
- Eustachian tube dysfunction

Manifestations of allergic rhinitis affecting the ears, eyes, and oropharynx include the following:

- Ears: Retraction and abnormal flexibility of the tympanic membrane
- Eyes: Injection and swelling of the palpebral conjunctivae, with excess tear production; Dennie-Morgan lines (prominent creases below the inferior eyelid); and dark circles around the eyes (“allergic shiners”), which are related to vasodilation or nasal congestion
- Oropharynx: "Cobblestoning," that is, streaks of lymphoid tissue on the posterior pharynx; tonsillar hypertrophy; and malocclusion (overbite) and a high-arched palate
Management

The management of allergic rhinitis consists of the following 3 major treatment strategies:

- Environmental control measures and allergen avoidance: These include keeping exposure to allergens such as pollen, dust mites, and mold to a minimum
- Pharmacologic management: Patients are often successfully treated with oral antihistamines, decongestants, or both; regular use of an intranasal steroid spray may be more appropriate for patients with chronic symptoms

Key recommendations include the following:

- For patients with a stuffy nose, nasal passage discoloration, and/or red and watery eyes, doctors should forgo sinus imaging process in favor of specific immunoglobulin E screening. Sinonasal imaging exposes patients to unnecessary radiation.
- Intranasal steroids and oral antihistamines are recommended as first lines of treatment. Oral leukotriene receptor antagonists are not.
- Sublingual or subcutaneous immunotherapy should be offered to patients who do not respond to pharmacologic therapy.

Treatment

Most cases of allergic rhinitis respond to pharmacotherapy. Patients with intermittent symptoms are often treated adequately with oral antihistamines, decongestants, or both as needed.

Cetirizine (Zirtec)

Competes with histamine for H1 receptors in GI tract, blood vessels, and respiratory tract, reducing hypersensitivity reactions. Once-daily dosing is convenient. Bedtime dosing may be useful if sedation is a problem.

Adult

Allergies/Hay Fever/Urticaria

Perennial and seasonal allergic and vasomotor rhinitis; relief of symptoms from colds, urticaria, angioedema, anaphylactic reactions, pruritus, allergic conjunctivitis 5-10 mg PO qDay, depending on severity of symptoms; not to exceed 10 mg qDay

Renal impairment

- GFR >50 mL/min: Dose adjustment not necessary
- GFR ≤ 50 mL/min: 5 mg PO qDay

Pediatric

Allergies/Hay Fever/Urticaria

Perennial and seasonal allergic and vasomotor rhinitis

<2 years: Safety and efficacy not established
2-6 years: 2.5 mg (0.5 teaspoon) oral solution PO qDay; can increase to 5 mg PO qDay or 2.5 mg PO twice daily; not to exceed 5 mg qDay
>6 years: 5-10 mg PO qDay, depending on severity of symptoms; not to exceed 10 mg qDay

**Levocetirizine (Xozal)**

Histamine1-receptor antagonist. Active enantiomer of cetirizine. Peak plasma levels reached within 1 h and half-life is about 8 h. Available as a 5-mg breakable (scored) tab. Indicated for seasonal and perennial allergic rhinitis.

**Renal Impairment**
CrCl 50-80 mL/min: 2.5 mg PO qDay
CrCl 30-50 mL/min: 2.5 mg PO qOD
CrCl 10-30 mL/min: 2.5 mg PO 2x/wk
CrCl <10 mL/min: contraindicated

**Allergic Rhinitis**
<6 months: Safety and efficacy not established
6 months to 5 years: 1.25 mg PO qDay (evening)
6-12 years: 2.5 mg PO qDay (evening)
>12 years: 5 mg PO qDay (evening)

**Montelukast (Singulair)**

Selective leukotriene receptor antagonist that inhibits the cysteinyI leukotriene (CysLT 1) receptor. Selectively prevents action of leukotrienes released by mast cells and eosinophils. When used as a single agent, has been shown to result in a reduction of seasonal allergic rhinitis symptoms.

**Adult**

**Asthma**
Propylaxis and maintenance treatment
10 mg (single 10-mg tablet) PO once daily in evening

**Exercise-Induced Bronchospasm**
Propylaxis
10 mg PO 2 hours before exercise; do not take additional dose within 24 hours
If taking drug for another indication, do not take additional dose to prevent exercise-induced bronchospasm (EIB)

**Allergic or Perennial Rhinitis**
10 mg (single 10-mg tablet) PO once daily

**Administration**
Patients aged ≥12 years with both asthma and allergic rhinitis: 1 dose PO at bedtime
Patients with allergic rhinitis: Dosing time may be individualized to patient needs
Granules may be taken directly; mixed in applesauce, carrots, rice, or ice cream; or dissolved in 5 mL of breast milk or baby formula (administer within 15 minutes of opening)
**Pediatric**

**Asthma**
Prophylaxis and maintenance treatment in patients aged ≥12 months  
<12 months: Safety and efficacy not established  
12-24 months: 4 mg (granules) PO once daily in evening  
2-6 years: 4 mg (chewable tablet or granules) PO once daily in evening  
6-15 years: 5 mg (chewable tablet) PO once daily in evening  
>15 years: 10 mg (conventional tablet) PO once daily in evening  

**Exercise-Induced Bronchospasm**
Prophylaxis  
6-15 years: 5 mg (chewable tablet) PO 2 hours before exercise; do not take additional dose within 24 hours  
>15 years: 10 mg PO 2 hours before exercise; do not take additional dose within 24 hours  
If taking drug for another indication, do not take additional dose to prevent EIB

**Perennial Allergic Rhinitis**
<6 months: Safety and efficacy not established  
6-24 months: 4 mg (granules) PO once daily  
2-6 years: 4 mg (chewable tablet or granules) PO once daily  
6-15 years: 5 mg (chewable tablet) PO once daily  
>15 years: 10 mg (conventional tablet) PO once daily

**Seasonal Allergic Rhinitis**
<2 years: Safety and efficacy not established  
2-6 years: 5 mg (chewable tablet) or 4 mg (granules) PO once daily  
6-15 years: 5 mg (chewable tablet) PO once daily  
>15 years: 10 mg (conventional tablet) PO once daily
UPPER RESPIRATORY TRACT INFECTION

URI represents the most common acute illness evaluated in the outpatient setting. URIs range from the common cold—typically a mild, self-limited, catarrhal syndrome of the nasopharynx—to life-threatening illnesses such as epiglottitis.

**Signs and symptoms**

Details of the patient's history aid in differentiating a common cold from conditions that require targeted therapy, such as group A streptococcal pharyngitis, bacterial sinusitis, and lower respiratory tract infections. Clinical manifestations of these conditions, as well as allergy, show significant overlap.

Common URI terms are defined as follows:

- **Rhinitis**: Inflammation of the nasal mucosa
- **Rhinosinusitis or sinusitis**: Inflammation of the nares and paranasal sinuses, including frontal, ethmoid, maxillary, and sphenoid
- **Nasopharyngitis (rhinopharyngitis or the common cold)**: Inflammation of the nares, pharynx, hypopharynx, uvula, and tonsils
- **Pharyngitis**: Inflammation of the pharynx, hypopharynx, uvula, and tonsils
- **Epiglottitis (supraglottitis)**: Inflammation of the superior portion of the larynx and supraglottic area
- **Laryngitis**: Inflammation of the larynx
- **Laryngotracheitis**: Inflammation of the larynx, trachea, and subglottic area
- **Tracheitis**: Inflammation of the trachea and subglottic area
## Symptoms of Allergies, URIs, and Influenza

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Allergy</th>
<th>URI</th>
<th>Influenza</th>
</tr>
</thead>
<tbody>
<tr>
<td>Itchy, watery eyes</td>
<td>Common</td>
<td>Rare; conjunctivitis may occur with adenovirus</td>
<td>Soreness behind eyes, sometimes conjunctivitis</td>
</tr>
<tr>
<td>Nasal discharge</td>
<td>Common</td>
<td>Common</td>
<td>Common</td>
</tr>
<tr>
<td>Nasal congestion</td>
<td>Common</td>
<td>Common</td>
<td>Sometimes</td>
</tr>
<tr>
<td>Sneezing</td>
<td>Very common</td>
<td>Very common</td>
<td>Sometimes</td>
</tr>
<tr>
<td>Sore throat</td>
<td>Sometimes (postnasal drip); itchy throat</td>
<td>Very common</td>
<td>Sometimes</td>
</tr>
<tr>
<td>Cough</td>
<td>Sometimes</td>
<td>Common, mild to moderate, hacking cough</td>
<td>Common, dry cough, can be severe</td>
</tr>
<tr>
<td>Headache</td>
<td>Sometimes, facial pain</td>
<td>Rare</td>
<td>Common</td>
</tr>
<tr>
<td>Fever</td>
<td>Never</td>
<td>Rare in adults, possible in children</td>
<td>Very common, 100-102°F or higher (in young children), lasting 3-4 days; may have chills</td>
</tr>
<tr>
<td>Malaise</td>
<td>Sometimes</td>
<td>Sometimes</td>
<td>Very common</td>
</tr>
<tr>
<td>Fatigue, weakness</td>
<td>Sometimes</td>
<td>Sometimes</td>
<td>Very common, can last for weeks, extreme exhaustion early in course</td>
</tr>
<tr>
<td>Myalgias</td>
<td>Never</td>
<td>Slight</td>
<td>Very common, often severe</td>
</tr>
<tr>
<td>Duration</td>
<td>Weeks</td>
<td>3-14 days</td>
<td>7 days, followed by additional days of cough and fatigue</td>
</tr>
</tbody>
</table>

**Viral nasopharyngitis**

Patients with the common cold may have a paucity of clinical findings despite notable subjective discomfort. Findings may include the following:

- Nasal mucosal erythema and edema are common
- Nasal discharge: Profuse discharge is more characteristic of viral infections than bacterial infections; initially clear secretions typically become cloudy white, yellow, or green over several days, even in viral infections
• Foul breath
• Fever: Less common in adults but may be present in children with rhinoviral infections
• Tonsillar hypertrophy
• Cough: This is more suggestive of a viral, rather than a bacterial, etiology
• Diarrhea: If associated with a URI, diarrhea suggests a viral etiology
• Fever: Can be caused by EBV infections and influenza

**Bacterial pharyngitis**

This may be difficult to distinguish from viral pharyngitis. Assessment for group A streptococcal infection warrants special attention. The following physical findings suggest a high risk for group A streptococcal disease

**Group A streptococcal pharyngitis** The following physical findings suggest a high risk for group A streptococcal disease:

- Erythema, swelling, or exudates of the tonsils or pharynx
- Temperature of 38.3°C or higher
- Tender anterior cervical nodes (≥1 cm)
- Absence of conjunctivitis, cough, and rhinorrhea, which are symptoms that may suggest viral illness

**Acute bacterial rhinosinusitis in children**, acute bacterial sinusitis is defined as a URI with any of the following:

- Persistent nasal discharge (any type) or cough lasting 10 days or more without improvement
- Worsening course (new or worse nasal discharge, cough, fever) after initial improvement
- Severe onset (fever of 102° or greater with nasal discharge) for at least 3 consecutive days

In older children and adults, symptoms (eg, pain, pressure) tend to localize to the affected sinus.

**Epiglottitis**

This condition is more often found in children aged 1-5 years, who present with a sudden onset of the following symptoms:

- Sore throat
- Drooling, difficulty or pain during swallowing, globus sensation of a lump in the throat
- Muffled dysphonia or loss of voice
- Dry cough or no cough, dyspnea
- Fever, fatigue or malaise (may be seen with any URI)
- Tripod or sniffing posture
Laryngotracheitis and laryngotracheobronchitis

- Nasopharyngitis often precedes laryngitis and tracheitis by several days
- Swallowing may be difficult or painful
- Patients may experience a globus sensation of a lump in the throat
- Hoarseness or loss of voice is a key manifestation of laryngeal involvement

Features of whooping cough (pertussis) are as follows:

- The classic whoop sound is an inspiratory gasping squeak that rises in pitch, typically interspersed between hacking coughs
- The whoop is more common in children
- Coughing often comes in paroxysms of a dozen coughs or more at a time and is often worst at night

The 3 classic phases of whooping cough are as follows:

- Catarrhal (7-10 days) with predominantly URI symptoms
- Paroxysmal (1-6 weeks) with episodic cough
- Convalescent (7-10 days) of gradual recovery

Management

Symptom-based therapy represents the mainstay of URI treatment in immunocompetent adults. Antimicrobial or antiviral therapy is appropriate in selected patients.

Epiglottitis

- Immediately admit the patient to the nearest hospital
- Avoid instrumentation; insertion of tongue depressors or other instruments may provoke airway spasm and precipitate respiratory compromise
- Monitor for respiratory fatigue, visually and with continuous pulse oximetry
- Administer oxygen according to pulse oximetry results
- Have equipment and personnel available for immediate intubation if necessary
- Start intravenous (IV) antibiotics after collecting culture specimens
- Empiric coverage for *Haemophilus influenzae* is appropriate; common choices include ceftriaxone or other third-generation cephalosporins, cefuroxime, and cefamandole
- Correct volume deficits with IV fluids; avoid sedatives

Laryngotracheitis

- Hospitalization may be necessary, especially in infants and young children who have hypoxemia, volume depletion, a risk of airway compromise, or respiratory fatigue
- Mild cases of croup (ie, laryngotracheobronchitis) may be managed at home with moist air inhalation
Hospitalized patients require monitoring for respiratory fatigue, visually and with continuous pulse oximetry.

Expertise for immediate intubation and access to the necessary equipment are required if respiratory failure is a possibility.

Administer humidified oxygen to all hypoxemic patients. In patients who do not require oxygen therapy, a cool-mist humidifier may be used.

IV or oral glucocorticoids are commonly used to reduce symptoms and shorten hospitalization in patients with moderate to severe croup.

Inhaled racemic epinephrine may temporarily dilate the airways.

**Rhinosinusitis**

- Most cases of acute rhinosinusitis, including mild and moderate bacterial sinusitis, resolve without antibiotics.
- Consider antibiotic treatment if symptoms persist without improving for 10 or more days, or if symptoms are severe or worsening during a period of 3-4 days or longer.
- Give first-line antibiotics for 5-7 days in most adults; for 10-14 days in children.
- Begin treatment with an agent that most narrowly covers likely pathogens, including *Streptococcus pneumoniae*, nontypeable *H influenzae*, and *Moraxella catarrhalis*.
- Initial first-line options include amoxicillin/clavulanate.
- Alternatives in penicillin-allergic patients are doxycycline and respiratory fluoroquinolones (eg, levofloxacin, moxifloxacin).
- In patients who worsen or do not improve after 3-5 days of empirical therapy, consider resistant pathogens, structural abnormality, or noninfectious etiology.
- Adjunctive therapy for adults includes nasal saline irrigation and intranasal steroids.

**Group A streptococcal disease**

- Oral penicillin or amoxicillin for 10 days for patients without an allergy to penicillin.
- If compliance is a concern, consider a single IM injection of benzathine penicillin G.
- A first-generation cephalosporin may be used in patients with non-anaphylactic penicillin allergy.
- Options for penicillin-allergic patients include clindamycin or clarithromycin for 10 days or azithromycin for 5 days.

**Treatment**

Most URIs are self-diagnosed and self-treated at home. Patients who present with URIs often benefit from reassurance, education, and instructions for symptomatic home treatment. Symptom-based therapy represents the mainstay of URI treatment in immunocompetent adults, although antimicrobial or antiviral therapy is appropriate in selected patients.

3 basic principles for the effective use of antibiotics to treat pediatric URIs, including acute otitis media, acute bacterial sinusitis, and streptococcal pharyngitis. The principles are as follows:

- Accurate diagnosis of a bacterial infection;
• Consideration of the risks vs benefits of antibiotic treatment; and
• Implementation of judicious prescribing strategies, including selection of the most effective antibiotic, prescription of an appropriate dose, and treating for the shortest possible duration.

These principles will help healthcare providers distinguish bacterial infections from viral infections.

**Symptomatic, Nonpharmacologic Self-Care**

The following home-care measures may help to provide relief of nasal and sinus symptoms:

- Warm, moist air
- Nasal saline
- Hydration
- Warm facial packs
- Bulb suction (for infants)
- Avoidance of nasal irritants (eg, cigarette smoke, indoor and outdoor air pollutants)

Nasal and paranasal sinus mucosae may become more irritated with dry air. The following strategies may maintain the moisture of membranes and loosen nasal secretions:

- Turn on hot shower water, close the bathroom door, sit down, and inhale the steam
- Take long, hot showers
- Use a vaporizer to increase humidity in rooms

If a vaporizer is used, the water must be changed daily to prevent microbial growth, especially with heated vaporizers. Heated systems may pose a risk for scalding injuries.

Nasal saline may provide temporary relief of congestion by removing nasal crusts and dried secretions. A systematic review of nasal saline irrigation as an adjunct in chronic rhinosinusitis symptom management concluded that the evidence shows symptom relief and that irrigation is well tolerated by most patients. Patients with sinusitis experienced symptomatic benefit from use of a neti pot method of nasal irrigation.

**Symptomatic, Pharmacologic Therapy**

Treatment of an uncomplicated URI is focused on specific measures to reduce symptoms, including use of the following:

- Oral or topical decongestants
- Antihistamines. Histamines are not thought to play a role in generating URI symptoms; therefore, newer, nonsedating antihistamines are not useful in reducing URI symptoms. However, first-generation oral antihistamines (eg, diphenhydramine, chlorpheniramine, clemastine) have some anticholinergic effects, which, in theory, could reduce sneezing and rhinorrhea.
- Saline nasal drops
- Guaifenesin
- Cromolyn
Cough associated with the common cold may be treated with a first-generation antihistamine combined with a decongestant (eg, brompheniramine with pseudoephedrine).

**Diet**

Increased fluids are warranted to replace insensible losses and reduced oral intake. However, alcohol may cause swelling of the nasal and paranasal sinus mucosae.

Antibiotics alter the gastrointestinal flora, and some foods may not be as digestible for days or weeks after antibiotics are used. Consumption of yogurt containing active cultures has been advocated as an aid to restoring normal flora after antibiotic therapy.

**Oral and topical decongestants**

Oral decongestants may provide symptom relief for patients with persistent rhinorrhea or sneezing associated with URI. However, despite common usage, evidence regarding the effectiveness of oral decongestants in acute sinusitis is scarce.

Adverse effects of oral decongestants include the following:

- Anxiousness
- Insomnia
- Tachycardia and dysrhythmias
- Elevated blood pressure
- Palpitations
- Tremor
- Urinary retention

The risk-to-benefit ratio for using cough and cold medicines in children younger than 2 years requires careful consideration because serious adverse events, including fatalities, have been reported with the use of over-the-counter preparations. Numerous over-the-counter cough and cold preparations are labeled "do not use" in children younger than 4 years.

**Antibiotics used in group A streptococcal infection are as follows:**

- Amoxicillin (Amoxil)
- Erythromycin
- Amoxicillin and clavulanate (Augmentin)
- Cefaclor (Ceclor)
- Azithromycin (Zithromax)

**Antibiotics used in epiglottitis are as follows:**

- Cefuroxime
- Ceftriaxone
- Cefotaxime

**Antibiotics used in pertussis are as follows:**

- Clarithromycin
- Erythromycin
- Azithromycin

**Antibiotics used in acute bacterial rhinosinusitis are as follows:**

- Amoxicillin/clavulanate
- Doxycycline
ACUTE SINUSITIS (Rhinosinusitis)

Sinusitis is characterized by inflammation of the lining of the paranasal sinuses.

Diagnosis and management of acute bacterial sinusitis in children and adolescents

- Previous diagnostic criteria for acute bacterial sinusitis in children were acute URI with either nasal discharge and/or daytime cough for longer than 10 days or severe onset of fever, purulent nasal discharge, and other respiratory symptoms for 3 or more consecutive days. A third criterion added to the updated guideline is URI with worsening symptoms such as nasal discharge, cough, and fever after initial improvement.

- Physicians may now observe children with persistent infection lasting longer than 10 days for an additional 3 days before prescribing antibiotics, but antibiotics should still be given to children with severe onset or worsening symptoms.

- First-line therapy is amoxicillin with or without clavulanate.

- Imaging tests are not recommended for children with uncomplicated acute bacterial sinusitis, although children with suspected orbital or CNS complications should undergo CT scanning of the paranasal sinuses.

Signs and symptoms

Clinical findings in acute sinusitis may include the following:

- Pain over cheek and radiating to frontal region or teeth, increasing with straining or bending down
- Redness of nose, cheeks, or eyelids
- Tenderness to pressure over the floor of the frontal sinus immediately above the inner canthus
- Referred pain to the vertex, temple, or occiput
- Postnasal discharge
- A blocked nose
- Persistent coughing or pharyngeal irritation
- Facial pain
- Hyposmia

Symptoms of acute bacterial rhinosinusitis include the following:

- Facial pain or pressure (especially unilateral)
- Hyposmia/anosmia
- Nasal congestion
- Nasal drainage
- Postnasal drip
- Fever
- Cough
- Fatigue
- Maxillary dental pain
- Ear fullness/pressure

The diagnosis of acute bacterial sinusitis should be entertained under either of the following circumstances:

- Presence of symptoms or signs of acute rhinosinusitis 10 days or more beyond the onset of upper respiratory symptoms
• Worsening of symptoms or signs of acute rhinosinusitis within 10 days after an initial improvement

The following signs may be noted on physical examination:
• Purulent nasal secretions
• Purulent posterior pharyngeal secretions
• Mucosal erythema
• Periorbital edema
• Tenderness overlying sinuses
• Air-fluid levels on transillumination of the sinuses (60% reproducibility rate for assessing maxillary sinus disease)
• Facial erythema

Diagnosis

Acute sinusitis is a clinical diagnosis.

Management

Treatment of acute sinusitis consists of providing adequate drainage of the involved sinus and appropriate systemic treatment of the likely bacterial pathogens. Drainage can be achieved surgically with sinus puncture and irrigation techniques. Options for medical drainage are as follows:
• Oral alpha-adrenergic vasoconstrictors (eg, pseudoephedrine, and phenylephrine) for 10-14 days
• Topical vasoconstrictors (eg, oxymetazoline hydrochloride) for a maximum of 3-5 days

Antibiotic treatment is usually given for 14 days. Usual first-line therapy is with one of the following:
• Amoxicillin, at double the usual dose (80-90 mg/kg/d), especially in areas with known Streptococcus pneumoniae resistance
• Clarithromycin
• Azithromycin

Second-line antibiotic should be considered for patients with any of the following:
• Residence in communities with a high incidence of resistant organisms
• Failure to respond within 48-72 hours of commencement of therapy
• Persistence of symptoms beyond 10-14 days

The most commonly used second-line therapies include the following:
• Amoxicillin-clavulanate
• Second- or third-generation cephalosporins (eg, cefuroxime, cefpodoxime, cefdinir)
• Macrolides (ie, clarithromycin)

Antibiotic selection with respect to previous antibiotic use and disease severity is as follows:
• Adults with mild disease who have not received antibiotics: Amoxicillin/clavulanate, amoxicillin (1.5-3.5 g/day), cefpodoxime proxetil, or cefuroxime is recommended as initial therapy.
• Adults with mild disease who have had antibiotics in the previous 4-6 weeks and adults with moderate disease: Amoxicillin/clavulanate, amoxicillin (3-3.5 g), cefpodoxime proxetil, or cefixime is recommended.
• Adults with moderate disease who have received antibiotics in the previous 4-6 weeks: Amoxicillin/clavulanate, levofloxacin, moxifloxacin, or doxycycline is recommended.

Symptomatic or adjunctive therapies may include the following:
• Humidification/vaporizer
- Warm compresses
- Adequate hydration
- Smoking cessation
- Balanced nutrition
- Nonnarcotic analgesia

Most patients with acute sinusitis are treated in the primary care setting. Further evaluation by an otolaryngologist is recommended when any of the following exist:
- When continued deterioration occurs with appropriate antibiotic therapy
- When episodes of sinusitis recur
- When symptoms persist after 2 courses of antibiotic therapy
- When comorbid immunodeficiency, nosocomial infection, or complications of sinusitis are present

While in the emergency department and upon discharge, patients may obtain significant immediate relief with the administration of first-generation antihistamines, decongestants, and nonsteroidal anti-inflammatory drugs

**Antibiotic therapy**

**In adults:**
Antibiotic therapy is indicated if the patient meets the criteria of duration or severity of symptoms. Oral amoxicillin is the first-line treatment. If the diagnosis is uncertain (moderate symptoms < 10 days) and the patient can be re-examined in the next few days, start with a symptomatic treatment, as for rhinopharyngitis or viral sinusitis.

**In children:**
Antibiotic therapy is indicated if the child has severe symptoms or mild symptoms associated with risk factors (e.g. immunosuppression, sickle cell disease, asthma). Oral amoxicillin is the first-line treatment.

Amoxicillin PO for 7 to 10 days:
- Children: 80 to 90 mg/kg/day in 3 divided doses
- Adults: 3 g/day in 3 divided doses

**In the event of failure to respond within 48 hours of therapy:**
amoxicillin/clavulanic acid PO for 7 to 10 days (the dose is expressed in amoxicillin):
Children < 40 kg: 45 to 50 mg/kg/day in 2 divided doses (if using ratio 8:1 or 7:1) or in 3 divided doses (if using ratio 4:1)
The dose of clavulanic acid should not exceed 12.5 mg/kg/day or 375 mg/day.
Children ≥ 40 kg and adults: 1500 to 2000 mg/day depending on the formulation available:
Ratio 8:1: 2000 mg/day = 2 tablets of 500/62.5 mg 2 times per day
Ratio 7:1: 1750 mg/day = 1 tablet of 875/125 mg 2 times per day
Ratio 4:1: 1500 mg/day = 1 tablet of 500/125 mg 3 times per day
The dose of clavulanic acid should not exceed 375 mg/day.

**In penicillin-allergic patients:**
Erythromycin PO for 7 to 10 days:
Children: 30 to 50 mg/kg/day in 2 to 3 divided doses
Adults: 2 to 3 g/day in 2 to 3 divided doses

In infants with ethmoiditis, immediate treatment is necessary: admit the patient to the nearest hospital
ACUTE LARYNGITIS

Laryngitis, an inflammation of the larynx, manifests in both acute and chronic forms. Acute laryngitis has an abrupt onset and is usually self-limited. If a patient has symptoms of laryngitis for more than 3 weeks, the condition is classified as chronic laryngitis. The etiology of acute laryngitis includes vocal misuse, exposure to noxious agents, or infectious agents leading to upper respiratory tract infections. The infectious agents are most often viral but sometimes bacterial. Because acute laryngitis is usually self-limited and treated with conservative measures, significant morbidity and mortality are not encountered.

Symptoms of laryngitis can begin suddenly and usually get worse over a period of two to three days. Common symptoms of laryngitis include:

- hoarseness
- difficulty speaking
- sore throat
- mild fever
- irritating cough
- a constant need to clear your throat

Treatment & Management

Vaughan states that patients know that laryngitis treatment requires only time and the common-sense avoidance of vocal excess and other irritants. The following measures can help lessen the intensity of the laryngitis while waiting for the condition to resolve:

- Inhaling humidified air promotes moisture of the upper airway, helping to clear secretions and exudate.
- Complete voice rest is suggested, although this recommendation is nearly impossible to follow. If the patient must speak, soft sighing phonation is best. Avoidance of whispering is best, as whispering promotes hyperfunctioning of the larynx.
- Prevailing data do not support the use of antihistamines and corticosteroids. If a patient uses these medications, he or she may have the false impression that the laryngitis is resolving and may continue to use his or her voice, leading to further insult. The drying effect of these medicines may also be deleterious.

The treatment for gastroesophageal reflux disease (GERD)–related laryngitic conditions includes dietary and lifestyle modifications as well as antireflux medications. Antacid medications that suppress acid production, such as H2-receptor and proton pump blocking agents, are highly effective against gastroesophageal reflux.

Spasmodic laryngitis in a child with rhinitis or measles: sudden, nocturnal onset with coughing fits followed by periods of suffocation and inspiratory dyspnoea. The child may develop stridor. The voice remains hoarse after the attack. The child remains afebrile.

- Monitor the child, try to keep him calm. Have him breathe in a humid environment (near a bowl of water or wet towel).
- Nasal irrigation with 0.9% sodium chloride or Ringer Lactate, 4 to 6 times/day to clear the airway.
- An antihistamine may be given for 3 days (promethazine PO or chlorphenamine PO).
- In children with severe dyspnea:
  Dexamethasone IM: 0.1 to 0.2 mg/kg as a single dose or hydrocortisone IM: 1 mg/kg as a single dose
PHARYNGITIS

Viral Pharyngitis

Viral pharyngitis can be caused by numerous viruses. Acute pharyngitis is an inflammatory syndrome of the pharynx and/or tonsils caused by several different groups of microorganisms. Pharyngitis can be part of a generalized upper respiratory tract infection or a specific infection localized in the pharynx.

Pharyngitis in the common cold syndrome
Sore throat is usually not the primary symptom. Nasal symptoms, such as sneezing, watery nasal discharge, nasal congestion, or postnasal discharge, tend to precede throat symptoms. Throat symptoms can be in the form of soreness, scratchiness, or irritation. Nasal discharge may be thick and yellow. Nonproductive cough may be present. Fever, if present, is usually low grade and is more prominent in young children than in adults.

Pharyngitis caused by adenovirus
Pharyngitis caused by adenovirus is common among young children and military recruits. Patients with pharyngitis present with sore throat (more intense than that of a common cold), high fever, dysphagia, and red eyes. This syndrome is named pharyngoconjunctival fever.

Pharyngitis associated with EBV infectious mononucleosis
EBV infectious mononucleosis is most commonly observed in adolescents and young adults. Sore throat and fatigue are the most common symptoms. Pharyngeal symptoms are usually associated with other features of the disease (eg, fatigue, skin rash, anorexia).

Pharyngitis with influenza
Sore throat is the chief symptom in some patients with influenza. The onset of illness is usually abrupt, with myalgia, headache, fever, chills, and dry cough. The pharyngitis usually resolves in 3-4 days.

Treatment & Management
Analgesics and antipyretics may be used for relief of pain or pyrexia. Paracetamol is the drug of choice. Traditionally, aspirin has been used, but it may increase viral shedding. Aspirin should not be used in children or adolescents, especially with influenza, because of its association with Reye syndrome. Antibiotics do not hasten recovery or reduce the frequency of bacterial complications. Drinking large amounts of fluid is recommended.
Bacterial Pharyngitis

The most common and important bacterial cause of pharyngitis is *Streptococcus pyogenes*. When suspected, bacterial pharyngitis should be confirmed with routine diagnostic tests and treated with various antibiotics.

**Clinical Presentation**

The signs and symptoms listed below may be seen with many non-GABHS etiologies. Furthermore, individuals with GABHS pharyngitis may have only a few or mild features listed. Conjunctivitis, cough, hoarseness, coryza, diarrhea, anterior stomatitis, discrete ulcerative lesions, and a viral exanthem are all more consistent with an etiology other than GABHS, particularly viral.

- Sore throat, usually with sudden onset
- Odynophagia
- Headache
- Nausea, vomiting, and abdominal pain

Physical examination may reveal the following:

- Fever
- Tonsillopharyngeal erythema
- Exudates (patchy and discrete)
- Beefy red swollen uvula
- Lymphadenopathy (tender anterior cervical nodes)
- Petechiae on the palate
- Scarlatiniform rash

**Treatment & Management**

Treatment of GABHS pharyngitis should be initiated only after confirmation with a RADT or throat culture. Alternatively, treatment in high-risk patients may be started before throat culture results are available, but antibiotics should be stopped if the culture returns negative results. Even though most cases of GABHS pharyngitis resolve after 3-4 days without treatment, antibiotics decrease the likelihood of local suppurative complications and acute rheumatic fever. Oral antibiotics should be administered for 10 days, although many recent studies show similar efficacy with shorter courses. Antibiotic therapy does not decrease the likelihood of poststreptococcal glomerulonephritis.

Oral penicillin V remains the preferred antibiotic to treat GABHS pharyngitis. Amoxicillin is often prescribed and is an acceptable first-line agent because of its narrow spectrum, the ease of once-daily dosing, and improved palatability, especially for children. Both antibiotics are equally efficacious.

A first generation cephalosporin (Cephalexin, Cefadroxil) is a treatment alternative. In patients with history of severe or anaphylactic reactions to penicillin, macrolides such as azithromycin, clarithromycin, and erythromycin may be used, although resistance has been reported.
The Joachim score diminishes empiric antibiotic use in settings where rapid testing for GAS is not available.

<table>
<thead>
<tr>
<th>Age</th>
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</thead>
<tbody>
<tr>
<td>≤ 35 months</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>36 to 59 months</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>≥ 60 months</td>
<td>3</td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Bacterial signs</th>
<th>One point for each</th>
<th>Total number of bacterial signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tender cervical node</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Headache</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Petechiae on the palate</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abdominal pain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sudden onset (&lt; 12 hours)</td>
<td></td>
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</tr>
</tbody>
</table>

Take age value (1, 2 or 3) and add it to the number of bacterial signs above =

<table>
<thead>
<tr>
<th>Viral signs</th>
<th>One point for each</th>
<th>Total number of viral signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conjunctivitis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Coryza (runny nose)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diarrhoea</td>
<td></td>
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</tr>
</tbody>
</table>

Subtract the number of viral signs to obtain the score =

Joachim score is ≤ 2: viral pharyngitis, which typically resolves within a few days (or weeks): no antibiotic therapy.

Joachim score is ≥ 3: administer antibiotic therapy for GAS pharyngitis
OTITIS

Otitis Media

OM is any inflammation of the middle ear.

Signs and symptoms
AOM implies rapid onset of disease associated with one or more of the following symptoms:
- Otalgia
- Otorrhea
- Headache
- Fever
- Irritability
- Loss of appetite
- Vomiting
- Diarrhea

OME often follows an episode of AOM. Symptoms that may be indicative of OME include the following:
- Hearing loss
- Tinnitus
- Vertigo
- Otalgia

Diagnosis

OME does not benefit from antibiotic treatment. Therefore, it is critical for clinicians to be able to distinguish normal middle ear status from OME or AOM. Doing so will avoid unnecessary use of antibiotics, which leads to increased adverse effects of medication and facilitates the development of antimicrobial resistance.

Examination

Pneumatic otoscopy remains the standard examination technique for patients with suspected OM. In addition to a carefully documented examination of the external ear and TM, examining the entire head and neck region of patients with suspected OM is important.

Every examination should include an evaluation and description of the following four TM characteristics:
- Color – A normal TM is a translucent pale gray; an opaque yellow or blue TM is consistent with MEE
- Position – In AOM, the TM is usually bulging; in OME, the TM is typically retracted or in the neutral position
- Mobility – Impaired mobility is the most consistent finding in patients with OME
- Perforation – Single perforations are most common

Management

Most cases of AOM improve spontaneously. Cases that require treatment may be managed with antibiotics and analgesics or with observation alone.

The recommendations offer more rigorous diagnostic criteria to reduce unnecessary antibiotic use. According to the guidelines, management of AOM should include an assessment of pain. Analgesics, particularly paracetamol and ibuprofen, should be used to treat pain whether antibiotic therapy is or is not prescribed.
Recommendations for prescribing antibiotics include the following:

- Antibiotics should be prescribed for bilateral or unilateral AOM in children aged at least 6 months with severe signs or symptoms (moderate or severe otalgia, otalgia for 48 hours or longer, or temperature 39°C or higher) and for nonsevere, bilateral AOM in children aged 6 to 23 months.
- On the basis of joint decision-making with the parents, unilateral, nonsevere AOM in children aged 6-23 months or nonsevere AOM in older children may be managed either with antibiotics or with close follow-up and withholding antibiotics unless the child worsens or does not improve within 48-72 hours of symptom onset.
- Amoxicillin (for empiric treatment from 40-45 mg/kg/day to 80-90 mg/kg/day because of concerns about increasingly resistant strains of *S pneumoniae*, which are theoretically susceptible to this higher dose) is the antibiotic of choice unless the child received it within 30 days, has concurrent purulent conjunctivitis, or is allergic to penicillin; in these cases, clinicians should prescribe an antibiotic with additional beta-lactamase coverage.

Stressing the importance of documenting true clinical failure of therapy after at least 3 days of treatment with high-dose amoxicillin, the working group suggests tympanocentesis for identification and susceptibility testing of the etiologic bacteria to guide alternate antibiotic therapy.

In cases where second-line therapy is empirically chosen (a common occurrence, because few primary care physicians routinely perform tympanocentesis in the office), the recommendations suggest administering the following three preparations:

- High-dose oral amoxicillin-clavulanate (80-90 mg/kg/day of amoxicillin component, 6.4 mg/kg/day of clavulanate component)
- Oral cefuroxime axetil (suspension, 30 mg/mg/day; tablet, 250 mg twice daily)

**Medical therapy for otitis media with effusion**

Most cases of OME occur after an episode of AOM, and 67% of patients develop a middle ear effusion (MEE).

The following are among the many strategies advocated for medical treatment in patients with OME:

- Antimicrobials
- Antihistamine-decongestants
- Intranasal and systemic steroids
- NSAIDs
- Mucolytics
- Aggressive management of allergic symptoms

Of these options, only antimicrobial therapy has provided measurable benefits.

Selection of an antibiotic agent should be individualized to the patient. If penicillin allergy is not a concern and if the patient has no recent exposure to antibiotics, a reasonable choice for initial therapy is amoxicillin, administered at the same high dose recommended by the CDC for AOM (ie, 80-90 mg/kg/day). Duration of therapy; 10 days is reasonable for amoxicillin, amoxicillin-clavulanate, and cephalosporins.

The steroid regimen should be oral prednisone or prednisolone at a dosage of 1 mg/kg/day for 5-7 days, administered in combination with a beta-lactam antibiotic. Steroids are contraindicated in patients with exposure to varicella who have not received the varicella vaccine because of the possibility of life-threatening disseminated disease.
Bronchitis is characterized by inflammation of the bronchial tubes (bronchi), the air passages that extend from the trachea into the small airways and alveoli.

**Signs and symptoms**
A complete history must be obtained, including information on exposure to toxic substances and smoking. Symptoms of bronchitis include the following:
- Cough (the most commonly observed symptom)
- Sputum production (clear, yellow, green, or even blood-tinged)
- Fever (relatively unusual; in conjunction with cough, suggestive of influenza or pneumonia)
- Nausea, vomiting, and diarrhea (rare)
- General malaise and chest pain (in severe cases)
- Dyspnea and cyanosis (only seen with underlying chronic obstructive pulmonary disease [COPD] or another condition that impairs lung function)
- Sore throat
- Runny or stuffy nose
- Headache
- Muscle aches
- Extreme fatigue

Physical examination findings in acute bronchitis are variable and may include the following:
- Diffuse wheezes, high-pitched continuous sounds, and the use of accessory muscles (in severe cases)
- Diffuse diminution of air intake or inspiratory stridor (indicative of bronchial or tracheal obstruction)
- Sustained heave along the left sternal border (indicative of right ventricular hypertrophy secondary to chronic bronchitis)
- Clubbing on the digits and peripheral cyanosis (indicative of cystic fibrosis)
- Bullous myringitis (suggestive of mycoplasmal pneumonia)
- Conjunctivitis, adenopathy, and rhinorrhea (suggestive of adenoviral infection)

**Acute bronchitis**
Patients typically present with a cough that lasts more than 5 days and may be associated with sputum production. Cough usually resolves within 3 weeks but may linger for up to 8 weeks.
- Acute bronchitis is typically caused by viruses.
- There is limited evidence to support the use of antibiotics for treating acute bronchitis in otherwise healthy adults. Antibiotic administration does not significantly alter presence of productive cough or activity limitations at follow-up doctor visits; however, there is a trend toward increased adverse effects with their use.
- There may be a role for antibiotic treatment of acute bronchitis in the elderly with multiple comorbidities.
- Symptomatic treatment includes the use of cough suppressants (dextromethorphan or codeine), mucolytics, and bronchodilators (albuterol).
- Patients without underlying heart or lung disease who present with a persistent cough lasting more than 14 days should be evaluated for pertussis.
Acute bacterial exacerbation of chronic bronchitis (ABECB)

Bacterial pathogens are identified in less than half of all ABECB cases. The Anthonisen Criteria is typically used to qualify severity of acute exacerbations. Three clinical factors are considered: dyspnea, sputum volume, and sputum purulence. Antibiotic treatment is recommended for moderate (2 of 3 symptoms) or severe (all 3 symptoms) exacerbations. Change in sputum color has also been recognized to be a strong predictor of presence of potentially pathologic microorganisms in ABECB. Green and yellow sputum are more likely than white sputum to be culture positive.

Mild ABECB:
- No antibiotics recommended
- Outpatient symptomatic therapy and monitor for worsening symptoms

Moderate ABECB and/or any one of the following: age < 65 years, FEV1 >50% predicted, no cardiac disease, or < 3 exacerbations per year:
- Azithromycin 500 mg PO on first day then 250 mg PO daily for next 4 days or
- Clarithromycin 250-500 mg PO BID for 7-14 days or
- Doxycycline 100 mg PO BID for 7 days or
- Cefuroxime 250-500 mg PO q12h for 10 days or
- If recent antibiotic exposure within 3 months, use alternative class.

Severe ABECB and/or anyone of the following: age ≥65 years, FEV1 ≤ 50% predicted, cardiac disease, or ≥3 exacerbations per year:
- Consider hospitalization.
- Amoxicillin-clavulanate (875 mg/125 mg) 1 tablet PO BID for 7-10 days or
- Levofloxacin 500 mg PO daily for at least 7 days or
- Gemifloxacin 320mg PO daily for 5 days or
- If at risk for Pseudomonas infection consider sputum culture and treatment with ciprofloxacin 500-750 mg PO BID for 7-14 days.
- If recent antibiotic exposure within 3 months, use alternative class.
PEDIATRIC BRONCHITIS

Acute bronchitis is a clinical syndrome produced by inflammation of the trachea, bronchi, and bronchioles. In children, acute bronchitis usually occurs in association with viral respiratory tract infection.

Symptoms of acute bronchitis usually include productive cough and sometimes retrosternal pain during deep breathing or coughing. Generally, the clinical course of acute bronchitis is self-limited, with complete healing and full return to function typically seen within 10-14 days following symptom onset. Chronic bronchitis is recurring inflammation and degeneration of the bronchial tubes that may be associated with active infection. Patients with chronic bronchitis have more mucus than normal. Chronic bronchitis is often associated with asthma, cystic fibrosis, dyskinetic cilia syndrome, foreign body aspiration, or exposure to an airway irritant.

Physical Examination
Lungs may sound normal. Crackles, rhonchi, or large airway wheezing, if any, tend to be scattered and bilateral. The pharynx may be injected.

Treatment & Management
Emergency care for acute bronchitis or exacerbation of chronic bronchitis must focus on ensuring that the child has adequate oxygenation. Outpatient care is appropriate unless bronchitis is complicated by severe underlying disease. General measures include rest, use of antipyretics, adequate hydration, and avoidance of smoke.

Febrile patients should increase oral fluid intake. Instruct the patient to rest until the fever subsides.

Pharmacologic Therapy
- Fever: paracetamol PO
- Keep the patient hydrated, humidify air (with a bowl of water or a wet towel).
- Nasal irrigation with 0.9% sodium chloride or Ringer Lactate, 4 to 6 times/day to clear the airway.
- Amoxicillin PO 100 mg/kg/day in 3 divided doses for 5 days

If the patient has dyspnoea, fever greater than 38.5°C and purulent expectorations: a secondary infection with *Haemophilus influenzae* or with pneumococcus is probable.

Acute bronchitis
In otherwise healthy individuals, the use of antibiotics has not demonstrated any consistent benefit in relieving symptoms or improving the natural history of acute bronchitis.

Medical therapy generally targets symptoms and includes use of analgesics and antipyretics. Antitussives and expectorants are often prescribed but have not been demonstrated to be useful.

Chronic bronchitis
Antibiotics should not be the primary therapy. They usually do not result in a cure and may delay the start of more appropriate asthma therapies. However, antibiotics may be appropriate in children with chronic wet cough and symptoms persisting beyond 2-4 weeks, most of whom have protracted bacterial bronchitis.

Bronchodilator therapy should be considered and instituted; a beta-adrenergic agonist, such as albuterol or terbutaline may be effective.
In the child who continues to cough despite a trial of bronchodilators and in whom the history and physical examination suggest a wheezy form of bronchitis, corticosteroids should be added. Short courses of dexamethasone (1-2 dose schedules) have been shown to be effective.
Bronchiolitis is an acute inflammatory injury of the bronchioles that is usually caused by a viral infection. This condition may occur in persons of any age, but severe symptoms are usually evident only in young infants.

**Signs and symptoms**
Because bronchiolitis primarily affects young infants, clinical manifestations are initially subtle, such as the following:
- May become increasingly fussy and have difficulty feeding during the 2 to 5-day incubation period
- Low-grade fever (usually < 38.6 C); possible hypothermia in infants younger than 1 month
- Increasing coryza and congestion
- Apnea: May be the presenting symptom in early disease

Severe cases of bronchiolitis may progress over 48 hours to the following signs and symptoms:
- Respiratory distress with tachypnea, nasal flaring, retractions
- Irritability
- Possibly cyanosis

**Diagnosis**
The diagnosis of bronchiolitis is based on clinical presentation, the patient’s age, seasonal occurrence, and findings from the physical examination, which may reveal the following:
- Tachypnea
- Tachycardia
- Fever (38-39°C)
- Retractions
- Fine rales (47%); diffuse, fine wheezing
- Hypoxia
- Otitis media

**Signs of severity:**
- Significant deterioration in general condition, toxic appearance (pallor, greyish colouration)
- Apnoea, cyanosis (check lips, buccal mucosa, fingernails)
- Respiratory distress (nasal flaring, sternal and chest wall indrawing)
- Anxiety and agitation (hypoxia), altered level of consciousness
- Respiratory rate > 60/min
- Decreased respiratory distress and slow respirations (< 30/min below the age of 1 year and < 20/min below the age of 3 years, exhaustion). Exercise caution in interpreting these signs as indicators of clinical improvement.
- Sweats, tachycardia at rest and in the absence of fever
- Silence on auscultation (severe bronchospasm)
- Difficulty drinking or sucking (reduced tolerance for exertion)

**Management**

**Hospitalise children with one of the following criteria:**
- Presence of any sign of severity
- Pre-existing pathology (cardiac or pulmonary disease, malnutrition, HIV, etc.)

**Consider hospitalisation on a case-by-case basis in the following situations:**
- Associated acute pathology (viral gastro-enteritis, bacterial infection, etc.)
• Age less than 3 months

Among numerous medications and interventions used to treat bronchiolitis, thus far, only oxygen appreciably improves the condition of young children. Therefore, therapy is directed toward symptomatic relief and maintenance of hydration and oxygenation.

**Outpatient treatment**
- Nasal irrigation with 0.9% NaCl before each feeding (demonstrate the technique to the mother).
- Small, frequent feedings to reduce vomiting triggered by bouts of coughing.
- Increased fluids if fever and/or significant secretions are present.
- Treat fever.
- Handle the patient as little as possible and avoid unnecessary procedures.

**Nonpharmacotherapy**
Supportive care for patients with bronchiolitis may include the following:
- Supplemental humidified oxygen
- Maintenance of hydration
- Mechanical ventilation
- Nasal and oral suctioning
- Apnea and cardiorespiratory monitoring
- Temperature regulation in small infants

**Pharmacotherapy**
Medications have a limited role in the treatment of bronchiolitis. Healthy children with bronchiolitis usually have limited disease and usually do well with supportive care only.

The following medications are used in selected patients with bronchiolitis:
- Alpha/beta agonists (eg, racemic epinephrine, albuterol)
- Antibiotics (eg, ampicillin, cefotaxime, ceftriaxone)
- Intransal decongestants (eg, oxymetazoline)
- Corticosteroids (eg, prednisone, methylprednisolone)

Bronchodilators should not be routinely used.
Supplemental oxygen should be supplied for saturations below 90% on pulse oximetry; saturation measurement is otherwise unnecessary.
Viral Pneumonia

Signs and symptoms
The common constitutional symptoms of viral pneumonia are as follows:
- Fever
- Chills
- Nonproductive cough
- Rhinitis
- Myalgias
- Headaches
- Fatigue

During physical examination, the patient may also display the following:
- Tachypnea and/or dyspnea
- Tachycardia or bradycardia
- Wheezing
- Rhonchi
- Rales
- Sternal or intercostal retractions
- Dullness to percussion
- Decreased breath sounds
- Pleurisy
- Pleural friction rub
- Cyanosis
- Rash
- Acute respiratory distress

The influenza viruses are the most common viral cause of pneumonia. Primary influenza pneumonia manifests with persistent symptoms of cough, sore throat, headache, myalgia, and malaise for more than three to five days. The symptoms may worsen with time, and new respiratory signs and symptoms, such as dyspnea and cyanosis, appear.

Respiratory syncytial virus (RSV) is the most frequent cause of lower respiratory tract infection in infants and children and the second most common viral cause of pneumonia in adults. Patients with RSV pneumonia typically present with fever, nonproductive cough, otalgia, anorexia, and dyspnea. Wheezes, rales, and rhonchi are common physical findings.
Bacterial Pneumonia

Signs and symptoms
On pulmonary auscultation: decreased vesicular breath sounds, dullness, localized foci of crepitation, sometimes bronchial wheeze.

Cough, particularly cough productive of sputum, is the most consistent presenting symptom of bacterial pneumonia and may suggest a particular pathogen, as follows:
- *Streptococcus pneumoniae*: Rust-colored sputum
- *Pseudomonas, Haemophilus*, and pneumococcal species: May produce green sputum
- *Klebsiella* species pneumonia: Red currant-jelly sputum
- Anaerobic infections: Often produce foul-smelling or bad-tasting sputum

Signs of bacterial pneumonia may include the following:
- Hyperthermia (fever, typically >38°C) or hypothermia (<35°C)
- Tachypnea (>18 respirations/min)
- Use of accessory respiratory muscles
- Tachycardia (>100 bpm) or bradycardia (<60 bpm)
- Central cyanosis
- Altered mental status

Physical findings may include the following:
- Adventitious breath sounds, such as rales/crackles, rhonchi, or wheezes
- Decreased intensity of breath sounds
- Egophony
- Whispering pectoriloquy
- Dullness to percussion
- Tracheal deviation
- Lymphadenopathy
- Pleural friction rub

Examination findings that may indicate a specific etiology include the following:
- Bradycardia: May indicate a *Legionella* etiology
- Periodontal disease: May suggest an anaerobic and/or polymicrobial infection
- Bullous myringitis: May indicate *Mycoplasma pneumoniae* infection
- Cutaneous nodules: May suggest *Nocardia* infection
- Decreased gag reflex: Suggests risk for aspiration

Consider hospitalization
Pediatric Pneumonia

Signs and symptoms
Pneumonia can occur at any age, although it is more common in younger children.

Newborns with pneumonia commonly present with: poor feeding and irritability, as well as tachypnea, retractions, grunting, and hypoxemia. Cough is the most common symptom of pneumonia in infants, along with tachypnea, retractions, and hypoxemia. These may be accompanied by congestion, fever, irritability, and decreased feeding. Adolescents experience similar symptoms to younger children. They may have other constitutional symptoms, such as headache, pleuritic chest pain, and vague abdominal pain. Vomiting, diarrhea, pharyngitis, and otalgia/otitis are also common in this age group.

Diagnosis
The signs and symptoms of pneumonia are often nonspecific and widely vary based on the patient’s age and the infectious organisms involved. Observing the child’s respiratory effort during a physical exam is an important first step in diagnosing pneumonia. The World Health Organization (WHO) respiratory rate thresholds for identifying children with pneumonia are as follows:

- Children younger than 2 months: Greater than or equal to 60 breaths/min
- Children aged 2-11 months: Greater than or equal to 50 breaths/min
- Children aged 12-59 months: Greater than or equal to 40 breaths/min

Assessment of oxygen saturation by pulse oximetry should be performed early in the evaluation when respiratory symptoms are present. Cyanosis may be present in severe cases. Capnography may be useful in the evaluation of children with potential respiratory compromise.

Management
Initial priorities in children with pneumonia include the identification and treatment of respiratory distress, hypoxemia, and hypercarbia. Grunting, flaring, severe tachypnea, and retractions should prompt immediate respiratory support. Children who are in severe respiratory distress should undergo tracheal intubation if they are unable to maintain oxygenation or have decreasing levels of consciousness.

Signs of serious illness (severe pneumonia) include:

- Chest in drawing: the inferior thoracic wall depresses on inspiration as the superior abdomen expands
- Cyanosis (lips, oral mucosa, fingernails) or O₂ saturation < 90%
- Nasal flaring
- Altered consciousness (child is abnormally sleepy or difficult to wake)
- Stridor (hoarse noise on inspiration)
- Grunting (a short repetitive noise produced by a partial closure of the vocal cords) on expiration
- Refusal to drink or feed
- Children under 2 months
- Severe malnutrition

Consider hospitalization
RED FLAG - Caution

- In malnourished children, the RR thresholds should be decreased by 5 breaths/minute from those listed above.
• Chest in drawing is significant if it is clearly visible and present at all times. If it is observed when a child is upset or feeding and is not visible when the child is resting, there is no chest in drawing.
• In children under 2 months of age, moderate chest in drawing is normal as the thoracic wall is flexible.
• Tuberculosis:
  ➢ in a child with cough, fever and poor weight gain and a history of close contact with a tuberculous patient.
  ➢ in the event of pneumonia complicated with empyema

The treatment is administered by parenteral route for at least 3 days then; if the clinical condition has improved and oral treatment can be tolerated, switch to the oral route with amoxicillin PO: 100 mg/kg/day in 3 divided doses, to complete 10 days of treatment.

**Pneumonia with no signs of serious illness**

**Infant under 3 months of age**
Admit the child for inpatient care and treat for severe pneumonia

**Children from 2 months to 5 years of age (outpatients, except young infants)**
amoxicillin PO: 100 mg/kg/day in 3 divided doses for 5 days
Follow-up in 48 to 72 hours or sooner if the child’s condition deteriorates:
• if the condition is improving: continue with the same antibiotic to complete treatment.
• if there is no improvement after 3 days of correct administration: add azithromycin
• if the condition is deteriorating: hospitalize and treat as severe pneumonia.

**Pneumonia in children over 5 years.**

**Consider hospitalization**

Signs of serious illness (severe pneumonia) include:
• cyanosis (lips, oral mucosa, fingernails)
• nasal flaring
• intercostal or subclavian in drawing
• RR > 30 breaths/minute
• heat rate > 125 beats/minute
• altered level of consciousness (drowsiness, confusion)
**RED FLAG - Caution**

- Sudden onset with high fever (higher than 39°C), thoracic pain and oral herpes are suggestive of pneumococcal infection. Symptoms may be confusing, particularly in children with abdominal pain, meningeal syndrome.

- Patients at risk include the elderly, patients suffering from heart failure, sickle cell disease or severe chronic bronchitis; immunocompromised patients

**Pneumonia without signs of serious illness**

**Amoxicillin PO**

Children: 100 mg/kg/day in 3 divided doses for 5 days

Adults: 3 g/day in 3 divided doses for 5 days

Follow-up in 48 to 72 hours or sooner if the patient’s condition deteriorates:

- if the patient is improving: continue with the same antibiotic to complete treatment;
- if the condition is deteriorating: hospitalise and treat as severe pneumonia;
- If there is no improvement after 3 days of correct administration: add azithromycin
ASTHMA

For Adults and Children Older than 5 Years

Asthma is a common and potentially serious chronic disease that imposes a substantial burden on patients, their families and the community. It causes respiratory symptoms, limitation of activity, and flare-ups (attacks) that sometimes require urgent health care and may be fatal.

Asthma causes symptoms such as wheezing, shortness of breath, chest tightness and cough that vary over time in their occurrence, frequency and intensity. These symptoms are associated with variable expiratory airflow, i.e. difficulty breathing air out of the lungs due to bronchoconstriction (airway narrowing), airway wall thickening, and increased mucus. Some variation in airflow can also occur in people without asthma, but it is greater in asthma.

Factors that may trigger or worsen asthma symptoms include viral infections, domestic or occupational allergens (e.g. house dust mite, pollens, and cockroach), tobacco smoke, exercise and stress. These responses are more likely when asthma is uncontrolled. Some drugs can induce or trigger asthma, e.g. beta-blockers, and (in some patients), aspirin or other NSAIDs.

DIAGNOSIS OF ASTHMA

Asthma is a disease with many variations (heterogeneous), usually characterized by chronic airway inflammation. Asthma has two key defining features:

- A history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, AND
- Variable expiratory airflow limitation.
The diagnosis of asthma should be confirmed and, for future reference, the evidence documented in the patient’s notes. Depending on clinical urgency and access to resources, this should preferably be done before starting controller treatment. Confirming the diagnosis of asthma is more difficult after treatment has been started.
CRITERIA FOR MAKING THE DIAGNOSIS OF ASTHMA

### 1. A history of variable respiratory symptoms

Typical symptoms are wheeze, shortness of breath, chest tightness, cough

- People with asthma generally have more than one of these symptoms
- The symptoms occur variably over time and vary in intensity
- The symptoms often occur or are worse at night or on waking
- Symptoms are often triggered by exercise, laughter, allergens or cold air
- Symptoms often occur with or worsen with viral infections

### 2. Evidence of variable expiratory airflow limitation

- At least once during the diagnostic process when FEV1 is low, document that the FEV1/FVC ratio is reduced. The FEV1/FVC ratio is normally more than 0.75–0.80 in adults, and more than 0.90 in children.
- Document that variation in lung function is greater than in healthy people. For example:
  - FEV1 increases by more than 12% and 200mL (in children, >12% of the predicted value) after inhaling a bronchodilator. This is called ‘bronchodilator reversibility’.
  - Average daily diurnal PEF variability* is >10% (in children, >13%)
  - FEV1 increases by more than 12% and 200mL from baseline (in children, by >12% of the predicted value) after 4 weeks of anti-inflammatory treatment (outside respiratory infections)
- The greater the variation, or the more times excess variation is seen, the more confident you can be of the diagnosis
- Testing may need to be repeated during symptoms, in the early morning, or after withholding bronchodilator medications.
- Bronchodilator reversibility may be absent during severe exacerbations or viral infections. If bronchodilator reversibility is not present when it is first tested, the next step depends on the clinical urgency and availability of other tests.
- For other tests to assist in diagnosis, including bronchial challenge tests

Physical examination in people with asthma is often normal, but the most frequent finding is wheezing on auscultation, especially on forced expiration.

Patients with cough as the only respiratory symptom

This may be due to chronic upper airway cough syndrome (‘post-nasal drip’), chronic sinusitis, gastroesophageal reflux (GERD), vocal cord dysfunction, or eosinophilic bronchitis, or cough variant asthma. Cough variant asthma is characterized by cough and airway hyperresponsiveness, and documenting variability in lung function is essential to make this diagnosis. However, lack of variability at the time of testing does not exclude asthma.
How to assess a patient with asthma

<table>
<thead>
<tr>
<th>Asthma control – assess both symptom control and risk factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Assess symptom control over the last 4 weeks</td>
</tr>
<tr>
<td>• Identify any other risk factors for poor outcomes</td>
</tr>
<tr>
<td>• Measure lung function before starting treatment, 3–6 months later, and then periodically, e.g. yearly</td>
</tr>
</tbody>
</table>

2. Treatment issues

• Record the patient’s treatment, and ask about side-effects
• Watch the patient using their inhaler, to check their technique
• Have an open empathic discussion about adherence
• Check that the patient has a written asthma action plan
• Ask the patient about their attitudes and goals for their asthma

3. Are there any comorbidities?

• These include rhinitis, rhinosinusitis, gastroesophageal reflux (GERD), obesity, obstructive sleep apnea, depression and anxiety.
• Comorbidities should be identified as they may contribute to respiratory symptoms and poor quality of life. Their treatment may complicate asthma management.

Asthma control means the extent to which the effects of asthma can be seen in the patient, or have been reduced or removed by treatment. Asthma control has two domains: symptom control (previously called ‘current clinical control’) and risk factors for future poor outcomes.

Poor symptom control is a burden to patients and a risk factor for flare-ups. Risk factors are factors that increase the patient’s future risk of having exacerbations (flare-ups), loss of lung function, or medication side-effects.
### Level of asthma symptom control

<table>
<thead>
<tr>
<th>In the past 4 weeks, has the patient had:</th>
<th>Well controlled</th>
<th>Partly controlled</th>
<th>Uncontrolled</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daytime symptoms more than twice/week?</td>
<td>Yes□ No□</td>
<td>None of these</td>
<td>1–2 of these</td>
</tr>
<tr>
<td>Any night waking due to asthma?</td>
<td>Yes□ No□</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reliever needed* more than twice/week?</td>
<td>Yes□ No□</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any activity limitation due to asthma?</td>
<td>Yes□ No□</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Risk factors for poor asthma outcomes

Assess risk factors at diagnosis and periodically, particularly for patients experiencing exacerbations. Measure FEV1 at start of treatment, after 3–6 months of controller treatment to record personal best lung function, then periodically for ongoing risk assessment.

**Potentially modifiable independent risk factors for exacerbations include:**
- Uncontrolled asthma symptoms (as above)
- ICS not prescribed; poor ICS adherence; incorrect inhaler technique
- High SABA use (with increased mortality if >1x200-dose canister/month)
- Low FEV1, especially if <60% predicted
- Major psychological or socioeconomic problems
- Exposures: smoking; allergen exposure if sensitized
- Comorbidities: obesity; rhinosinusitis; confirmed food allergy
- Sputum or blood eosinophilia
- Pregnancy

**Other major independent risk factors for flare-ups (exacerbations) include:**
- Ever being intubated or in intensive care for asthma
- Having 1 or more severe exacerbations in the last 12 months.

**Risk factors for developing fixed airflow limitation include**
- Lack of ICS treatment; exposure to tobacco smoke, noxious chemicals or occupational exposures; low FEV1; chronic mucus hypersecretion; and sputum or blood eosinophilia

**Risk factors for medication side-effects include:**
- Systemic: frequent OCS; long-term, high dose and/or potent ICS; also taking P450 inhibitors
- Local: high-dose or potent ICS; poor inhaler technique
How to investigate uncontrolled asthma in primary care

<table>
<thead>
<tr>
<th>Step</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Watch patient using their inhaler. Discuss adherence and barriers to use</td>
<td>Compare inhaler technique with a device-specific checklist, and correct errors; recheck frequently. Have an empathic discussion about barriers to adherence.</td>
</tr>
<tr>
<td>Confirm the diagnosis of asthma</td>
<td>If lung function normal during symptoms, consider halving ICS dose and repeating lung function after 2–3 weeks.</td>
</tr>
<tr>
<td>Remove potential risk factors. Assess and manage comorbidities</td>
<td>Check for risk factors or inducers such as smoking, beta-blockers, NSAIDs, allergen exposure. Check for comorbidities such as rhinitis, obesity, GERD, depression/anxiety.</td>
</tr>
<tr>
<td>Consider treatment step-up</td>
<td>Consider step up to next treatment level. Use shared decision-making, and balance potential benefits and risks.</td>
</tr>
<tr>
<td>Refer to a specialist or severe asthma clinic</td>
<td>If asthma still uncontrolled after 3–6 months on Step 4 treatment, refer for expert advice. Refer earlier if asthma symptoms severe, or doubts about diagnosis.</td>
</tr>
</tbody>
</table>
MANAGEMENT OF ASTHMA GENERAL PRINCIPLES

The long-term goals of asthma management are symptom control and risk reduction. The aim is to reduce the burden to the patient and their risk of exacerbations, airway damage, and medication side-effects.

The control-based asthma management cycle

Asthma treatment is adjusted in a continuous cycle to **assess, adjust treatment** and **review response**

**INITIAL CONTROLLER TREATMENT**

For the best outcomes, regular daily controller treatment should be initiated as soon as possible after the diagnosis of asthma is made, because:

- Early treatment with low dose ICS leads to better lung function than if symptoms have been present for more than 2–4 years
- Patients not taking ICS who experience a severe exacerbation have lower long-term lung function than those who have started ICS
- In occupational asthma, early removal from exposure and early treatment increase the probability of recovery

**Regular low dose ICS is recommended** for patients with any of the following:

- Asthma symptoms more than twice a month
- Waking due to asthma more than once a month
- Any asthma symptoms plus any risk factor(s) for exacerbations (e.g. needing OCS for asthma within the last 12 months; low FEV1; ever in intensive care unit for asthma)

Consider starting at a higher step (e.g. medium/high dose ICS, or ICS/LABA) if the patient has troublesome asthma symptoms on most days; or is waking from asthma once or more a week, especially if there are any risk factors for exacerbations.

If the initial asthma presentation is with severely uncontrolled asthma, or with an acute exacerbation, give a short course of OCS and start regular controller treatment (e.g. high dose ICS, or medium dose ICS/LABA).
Before starting initial controller treatment
- Record evidence for the diagnosis of asthma, if possible
- Document symptom control and risk factors
- Assess lung function, when possible
- Train the patient to use the inhaler correctly, and check their technique
- Schedule a follow-up visit

After starting initial controller treatment
- Review response after 2–3 months, or according to clinical urgency
- See Box 7 for ongoing treatment and other key management issues
- Consider step down when asthma has been well-controlled for 3 months
For children 6–11 years, theophylline is not recommended, and the preferred Step 3 treatment is medium dose ICS. **Low dose ICS/formoterol is the reliever medication for patients prescribed low dose budesonide/formoterol or low dose beclometasone/formoterol. ***Tiotropium by soft-mist inhaler is an add-on treatment for patients with a history of exacerbations; it is not indicated in children <18 years.
Low, medium and high daily doses of inhaled corticosteroids (mcg)

<table>
<thead>
<tr>
<th>Inhaled corticosteroid</th>
<th>Adults and adolescents</th>
<th>Children 6–11 years</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Low</td>
<td>Medium</td>
</tr>
<tr>
<td>Beclometasone dipropionate (CFC)*</td>
<td>200–500</td>
<td>&gt;500–1000</td>
</tr>
<tr>
<td>Beclometasone dipropionate (HFA)</td>
<td>100–200</td>
<td>&gt;200–400</td>
</tr>
<tr>
<td>Budesonide (DPI)</td>
<td>200–400</td>
<td>&gt;400–800</td>
</tr>
<tr>
<td>Budesonide (nebules)</td>
<td>250–500</td>
<td>&gt;500–1000</td>
</tr>
<tr>
<td>Ciclesonide (HFA)</td>
<td>80–160</td>
<td>&gt;160–320</td>
</tr>
<tr>
<td>Fluticasone propionate (DPI)</td>
<td>100–250</td>
<td>&gt;250–500</td>
</tr>
<tr>
<td>Fluticasone propionate (HFA)</td>
<td>100–250</td>
<td>&gt;250–500</td>
</tr>
<tr>
<td>Mometasone furoate</td>
<td>110–220</td>
<td>&gt;220–440</td>
</tr>
<tr>
<td>Triamcinolone acetonide</td>
<td>400–1000</td>
<td>&gt;1000–2000</td>
</tr>
</tbody>
</table>

CFC: chlorofluorocarbon propellant; DPI: dry powder inhaler; HFA: hydrofluoroalkane propellant.

STEPWISE APPROACH FOR ADJUSTING TREATMENT

Once asthma treatment has been started, ongoing decisions are based on a cycle to assess, adjust treatment and review response.

STEP 1: As-needed SABA with no controller (this is indicated only if symptoms are rare, there is no night waking due to asthma, no exacerbations in the last year, and normal FEV1). Other options: regular low dose ICS for patients with exacerbation risks.

STEP 2: Regular low dose ICS plus as-needed SABA
Other options: LTRA are less effective than ICS; ICS/LABA leads to faster improvement in symptoms and FEV1 than ICS alone but are more expensive and the exacerbation rate is similar. For purely seasonal allergic asthma, start ICS immediately and cease 4 weeks after end of exposure.

STEP 3: Low dose ICS/LABA either as maintenance treatment plus as-needed SABA, or as ICS/formoterol maintenance and reliever therapy
For patients with ≥1 exacerbation in the last year, low dose BDP/formoterol or BUD/formoterol maintenance and reliever strategy is more effective than maintenance ICS/LABA with as-needed SABA. Other options: Medium dose ICS
Children (6–11 years): Medium dose ICS. Other options: low dose ICS/LABA

STEP 4: Low dose ICS/formoterol maintenance and reliever therapy, or medium dose ICS/LABA as maintenance plus as-needed SABA
Other options: Add-on tiotropium by soft-mist inhaler for adults (≥18 years) with a history of exacerbations; high dose ICS/LABA, but more side-effects and little extra benefit; extra controller, e.g. LTRA or slow-release theophylline (adults)

Children (6–11 years): Refer for expert assessment and advice.

**STEP 5: Refer for expert investigation and add-on treatment**
Add-on treatments include anti-IgE (omalizumab) for severe allergic asthma. Sputum-guided treatment, if available, improves outcomes.

Other options: Add-on tiotropium by soft-mist inhaler for adults (≥18 years) with a history of exacerbations. Some patients may benefit from low dose OCS but long-term systemic side-effects occur.

Patients should preferably be seen 1–3 months after starting treatment and every 3–12 months after that, except in pregnancy when they should be reviewed every 4–6 weeks. After an exacerbation, a review visit within 1 week should be scheduled.

**Identifying patients at risk of asthma-related death**
These patients should be identified, and flagged for more frequent review.
- A history of near-fatal asthma requiring intubation and ventilation
- Hospitalization or emergency care for asthma in last 12 months
- Not currently using ICS, or poor adherence with ICS
- Currently using or recently stopped using OCS (this indicates the severity of recent events)
- Over-use of SABAs, especially more than 1 canister/month
- Lack of a written asthma action plan
- History of psychiatric disease or psychosocial problems
- Confirmed food allergy in a patient with asthma
Summary of stepwise management in children less than 5 years

**STEP 1**
Mild intermittent asthma

- Inhaled short-acting β₂ agonist as required

**STEP 2**
Regular preventer therapy

- Add inhaled corticosteroid (200-400 micrograms/day*) or long-acting β₂ agonist
- Start dose of inhaled corticosteroid appropriate to severity of disease

**STEP 3**
Initial add-on therapy

- In these children taking inhaled corticosteroid 200-400 micrograms/day, consider addition of inhaled anticholinergic or long-acting β₂ agonist
- In these children taking a long-acting β₂ agonist and additional inhaled corticosteroid 200-400 micrograms/day, consider addition of an inhaled anticholinergic
- In children under 5 years consider proceeding to step 4

**STEP 4**
Persistent poor control

Refer to respiratory paediatrician.

---

Summary of stepwise management in children aged 5-12 years

**STEP 1**
Mild intermittent asthma

- Inhaled short-acting β₂ agonist as required

**STEP 2**
Regular preventer therapy

- Add inhaled corticosteroid 200-400 micrograms/day* or long-acting β₂ agonist
- Start dose of inhaled corticosteroid appropriate to severity of disease

**STEP 3**
Initial add-on therapy

- In these children taking inhaled corticosteroid 200-400 micrograms/day, consider addition of inhaled anticholinergic or long-acting β₂ agonist
- In these children taking a long-acting β₂ agonist and additional inhaled corticosteroid 200-400 micrograms/day, consider addition of an inhaled anticholinergic
- In children under 5 years consider proceeding to step 4

**STEP 4**
Continuous or frequent use of oral steroids

* BDP or equivalent
* Higher nominal doses may be required if drug delivery is difficult
Summary of stepwise management in adults

**Step 1:**
Mild intermittent asthma

**Step 2:**
Regular preventer therapy

**Step 3:**
Initial add-on therapy

**Step 4:**
Persistent poor control

**Step 5:**
Continuous or frequent use of oral steroids

- Use daily steroid tablet in lowest dose providing adequate control
- Maintain high dose inhaled corticosteroid at 2,000 micrograms/day
- Consider other treatments to minimise the use of steroid tablets
- Refer patient for specialist care

**Symptoms vs Treatment**

1. Add inhaled long-acting β₂ agonist (LABA)
2. Assess control of asthma:
   - Good response to LABA: continue LABA
   - Benefit from LABA but control still inadequate:
     - Continue LABA and increase inhaled corticosteroid dose to 800 micrograms/day
     - If not already at this dose:
     - More response to LABA:
       - If inadequate, institute trial of other therapies, leukotriene receptor antagonist or SR theophylline

- Increasing inhaled corticosteroid up to 2,000 micrograms/day
- Addition of a fourth drug eg leukotriene receptor antagonist, SR theophylline, β₂ agonist tablet

Inhaled short-acting β₂ agonist as required

Start a dose of inhaled corticosteroid appropriate to severity of disease.

Patients should start treatment at the step most appropriate to the initial severity of their asthma. Check adherence and re-evaluate diagnosis if response to treatment is unexpectedly poor.
Management of asthma exacerbations in primary care

O₂: oxygen; PEF: peak expiratory flow; SABA: short-acting beta2-agonist (doses are for salbutamol)
### MANAGEMENT OF ACUTE ASTHMA IN ADULTS

#### ASSESSMENT OF SEVERE ASTHMA

Healthcare professionals must be aware that patients with severe asthma and one or more adverse psychosocial factors are at risk of death.

<table>
<thead>
<tr>
<th>INITIAL ASSESSMENT</th>
<th>LIFE-THREATENING ASTHMA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MODERATE ASTHMA</strong></td>
<td><strong>In a patient with severe asthma any one of:</strong></td>
</tr>
<tr>
<td>▪ increasing symptoms</td>
<td>▪ PEF &lt; 33% best or predicted</td>
</tr>
<tr>
<td>▪ PEF &gt; 50-75% best or predicted</td>
<td>▪ SpO₂ &lt; 92%</td>
</tr>
<tr>
<td>▪ no features of acute severe asthma</td>
<td>▪ PaO₂ &lt; 8 kPa</td>
</tr>
<tr>
<td><strong>ACUTE SEVERE ASTHMA</strong></td>
<td>▪ normal PaCO₂ (4.6-6.0 kPa)</td>
</tr>
<tr>
<td>Any one of:</td>
<td>▪ silent chest</td>
</tr>
<tr>
<td>▪ PEF 33-50% best or predicted</td>
<td>▪ cyanosis</td>
</tr>
<tr>
<td>▪ respiratory rate ≥ 25/min</td>
<td>▪ poor respiratory effort</td>
</tr>
<tr>
<td>▪ heart rate ≥ 110/min</td>
<td>▪ arrhythmia</td>
</tr>
<tr>
<td>▪ inability to complete sentences in one breath</td>
<td>▪ exhaustion, altered conscious level</td>
</tr>
<tr>
<td>▪ hypotension</td>
<td>▪ NEAR-FATAL ASTHMA</td>
</tr>
<tr>
<td><strong>Clinical features</strong></td>
<td>Raised PaCO₂ and/or requiring mechanical ventilation with raised inflation pressures</td>
</tr>
</tbody>
</table>

- **Clinical features**
  - Severe breathlessness (including too breathless to complete sentences in one breath), tachypnoea, tachycardia, silent chest, cyanosis or collapse
  - *None of these singly or together is specific and their absence does not exclude a severe attack*

- **PEF or FEV₁**
  - PEF or FEV₁ are useful and valid measures of airway calibre. PEF expressed as a % of the patient’s previous best value is most useful clinically. In the absence of this, PEF as a % of predicted is a rough guide

- **Pulse oximetry**
  - Oxygen saturation (SpO₂) measured by pulse oximetry determines the adequacy of oxygen therapy and the need for arterial blood gas measurement (ABG). The aim of oxygen therapy is to maintain SpO₂ 94-98%

- **Blood gases (ABG)**
  - Patients with SpO₂ < 92% or other features of life-threatening asthma require ABG measurement

- **Chest X-ray**
  - Chest X-ray is not routinely recommended in patients in the absence of:
    - suspected pneumomediastinum or pneumothorax
    - suspected consolidation
    - life-threatening asthma
    - failure to respond to treatment satisfactorily
    - requirement for ventilation

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## Management of Acute Asthma in Adults

### Criteria for Admission

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td><strong>B</strong></td>
<td>Admit patients with any feature of a life-threatening or near-fatal asthma attack.</td>
</tr>
<tr>
<td><strong>B</strong></td>
<td>Admit patients with any feature of a severe asthma attack persisting after initial treatment.</td>
</tr>
<tr>
<td><strong>C</strong></td>
<td>Patients whose peak flow is greater than 75% best or predicted one hour after initial treatment may be discharged from ED, unless there are other reasons why admission may be appropriate.</td>
</tr>
</tbody>
</table>

### Treatment of Acute Asthma

#### Oxygen

<p>| | |</p>
<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td><strong>A</strong></td>
<td>Give supplementary oxygen to all hypoxaemic patients with acute severe asthma to maintain an SpO2 level of 94-98%. Lack of pulse oximetry should not prevent the use of oxygen.</td>
</tr>
<tr>
<td><strong>C</strong></td>
<td>In hospital, ambulance and primary care, nebulisers for giving nebulised β₂ agonist bronchodilators should preferably be driven by oxygen.</td>
</tr>
</tbody>
</table>

#### β₂ Agonist Bronchodilators

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td><strong>A</strong></td>
<td>Use high-dose inhaled β₂ agonists as first line agents in patients with acute asthma and administer as early as possible. Reserve intravenous β₂ agonists for those patients in whom inhaled therapy cannot be used reliably.</td>
</tr>
<tr>
<td><strong>✓</strong></td>
<td>In patients with acute asthma with life-threatening features the nebulised route (oxygen-driven) is recommended.</td>
</tr>
</tbody>
</table>

#### Steroid Therapy

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A</strong></td>
<td>Give steroids in adequate doses in all cases of acute asthma attack.</td>
</tr>
<tr>
<td><strong>✓</strong></td>
<td>Continue prednisolone 40-50 mg daily for at least five days or until recovery.</td>
</tr>
</tbody>
</table>

#### Other Therapies

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A</strong></td>
<td>Nebulised magnesium is not recommended for treatment in adults with acute asthma.</td>
</tr>
<tr>
<td><strong>B</strong></td>
<td>Consider giving a single dose of IV magnesium sulphate to patients with:</td>
</tr>
<tr>
<td></td>
<td>acute severe asthma (PEF &lt;50% best or predicted) who have not had a good initial response to inhaled bronchodilator therapy.</td>
</tr>
<tr>
<td><strong>✓</strong></td>
<td>Magnesium sulphate (1.2-2 g IV infusion over 20 minutes) should only be used following consultation with senior medical staff.</td>
</tr>
<tr>
<td><strong>B</strong></td>
<td>Routine prescription of antibiotics is not indicated for patients with acute asthma.</td>
</tr>
</tbody>
</table>

#### Referral to Intensive Care

Refer any patient:
- requiring ventilatory support
- with acute severe or life-threatening asthma, who is failing to respond to therapy, as evidenced by:
  - deteriorating PEF
  - persisting or worsening hypoxia
  - hypercapnia
  - ABG analysis showing ↓ pH or ↑ H⁺
  - exhaustion, feeble respiration
  - drowsiness, confusion, altered conscious state
  - respiratory arrest

#### Follow Up

- It is essential that the patient’s primary care practice is informed within 24 hours of discharge from the emergency department or hospital following an asthma attack.
- Keep patients who have had a near-fatal asthma attack under specialist supervision indefinitely
- A respiratory specialist should follow up patients admitted with a severe asthma attack for at least one year after the admission.
MANAGEMENT OF ACUTE ASTHMA IN CHILDREN AGED 2 YEARS AND OVER

ACUTE SEVERE

- SpO₂ <92% PEF 33-50% best or predicted
- Can’t complete sentences in one breath or too breathless to talk or feed
- Heart rate >125 (>5 years) or >140 (2-5 years)
- Respiratory rate >30 breaths/min (>5 years) or >40 (2-5 years)

LIFE-THREATENING

- SpO₂ <92% PEF <33% best or predicted
- Silent chest
- Cyanosis
- Poor respiratory effort
- Hypotension
- Exhaustion
- Confusion

CRITERIA FOR ADMISSION

- Increase β₂ agonist dose by giving one puff every 30-60 seconds, according to response, up to a maximum of ten puffs
- Parents/carers of children with an acute asthma attack at home and symptoms not controlled by up to 10 puffs of salbutamol via pMDI and spacers, should seek urgent medical attention.
- If symptoms are severe additional doses of bronchodilator should be given as needed whilst awaiting medical attention.
- Paramedics attending to children with an acute asthma attack should administer nebulized salbutamol, using a nebuliser driven by oxygen if symptoms are severe, whilst transferring the child to the emergency department.
- Children with severe or life-threatening asthma should be transferred to hospital urgently
- Consider intensive inpatient treatment of children with SpO₂ <92% in air after initial bronchodilator treatment.

The following clinical signs should be recorded:

- Pulse rate – increasing tachycardia generally denotes worsening asthma; a fall in heart rate in life-threatening asthma is a pre-terminal event
- Respiratory rate and degree of breathlessness – ie too breathless to complete sentences in one breath or to feed
- Use of accessory muscles of respiration – best noted by palpation of neck muscles
- Amount of wheezing – which might become biphasic or less apparent with increasing airways obstruction
- Degree of agitation and conscious level – always give calm reassurance

NB Clinical signs correlate poorly with the severity of airways obstruction. Some children with acute severe asthma do not appear distressed.

INITIAL TREATMENT OF ACUTE ASTHMA

OXYGEN

- Children with life-threatening asthma or SpO₂ <94% should receive high flow oxygen via a tight fitting face mask or nasal cannula at sufficient flow rates to achieve normal saturations of 94–98%.
## MANAGEMENT OF ACUTE ASTHMA IN CHILDREN AGED 2 YEARS AND OVER

### BRONCHODILATORS

- **A** Inhaled $\beta_2$ agonists are the first line treatment for acute asthma.
- **A** A pMDI + spacer is the preferred option in children with mild to moderate asthma.
- **B** Individualise drug dosing according to severity and adjust according to the patient’s response.
- **A** If symptoms are refractory to initial $\beta_2$ agonist treatment, add ipratropium bromide (250 micrograms/dose mixed with the nebulised $\beta_2$ agonist solution).
- **✓** Repeated doses of ipratropium bromide should be given early to treat children who are poorly responsive to $\beta_2$ agonists.
- **C** Consider adding 150 mg magnesium sulphate to each nebulised salbutamol and ipratropium in the first hour in children with a short duration of acute severe asthma symptoms presenting with an oxygen saturation less than 92%.
- **✓** Discontinue long-acting $\beta_2$ agonists when short-acting $\beta_2$ agonists are required more often than four hourly.

### STEROID THERAPY

- **A** Give oral steroids early in the treatment of acute asthma attacks.
  - **•** Use a dose of 20 mg prednisolone for children aged 2–5 years and a dose of 30–40 mg for children >5 years. Those already receiving maintenance steroid tablets should receive 2 mg/kg prednisolone up to a maximum dose of 60 mg.
  - **✓** Repeat the dose of prednisolone in children who vomit and consider intravenous steroids in those who are unable to retain orally ingested medication.
  - **•** Treatment for up to three days is usually sufficient, but the length of course should be tailored to the number of days necessary to bring about recovery. Tapering is unnecessary unless the course of steroids exceeds 14 days.

### SECOND LINE TREATMENT OF ACUTE ASTHMA

- **B** Consider early addition of a single bolus dose of intravenous salbutamol (15 micrograms/kg over 10 minutes) in a severe asthma attack where the patient has not responded to initial inhaled therapy.
- **A** Aminophylline is not recommended in children with mild to moderate acute asthma.
- **B** Consider aminophylline for children with severe or life-threatening asthma unresponsive to maximal doses of bronchodilators and steroids.

**IV magnesium sulphate is a safe treatment for acute asthma although its place in management is not yet established.**
MANAGEMENT OF ACUTE ASTHMA IN CHILDREN AGED UNDER 2 YEARS

- The assessment of acute asthma in early childhood can be difficult
- Intermittent wheezing attacks are usually due to viral infection and the response to asthma medication is inconsistent
- Prematurity and low birth weight are risk factors for recurrent wheezing
- The differential diagnosis of symptoms includes:
  - Aspiration pneumonitis
  - Pneumonia
  - Bronchiolitis
  - Tracheomalacia
  - Complications of underlying conditions such as congenital anomalies and cystic fibrosis

TREATMENT OF ACUTE ASTHMA

BRONCHODILATORS

B Oral β₂ agonists are not recommended for acute asthma in infants.

A For mild to moderate acute asthma attacks, a pMDI + spacer and mask is the optimal drug delivery device.

B Consider inhaled ipratropium bromide in combination with an inhaled β₂ agonist for more severe symptoms.

STEROID THERAPY

B In infants, consider steroid tablets early in the management of severe asthma attacks in the hospital setting.

✓Steroid tablet therapy (10 mg of soluble prednisolone for up to three days) is the preferred steroid preparation for use in this age group.

For children with frequent episodes of wheeze associated with viruses caution should be taken in prescribing multiple courses of oral steroids.
ASTHMA IN ADOLESCENTS

Adolescents are defined by the World Health Organisation (WHO) as young people between the ages 10 and 19 years of age.

Key elements of working effectively with adolescents in the transition to adulthood include:
- seeing them on their own, separate from their parents/careers, for part of the consultation, and
- discussing confidentiality and its limitations.

PREVALENCE OF ASTHMA IN ADOLESCENCE

Asthma is common in adolescents but is frequently undiagnosed because of under-reporting of symptoms.

- Clinicians seeing adolescents with any cardiorespiratory symptoms should consider asking about symptoms of asthma.

DIAGNOSIS AND ASSESSMENT

Symptoms and signs of asthma in adolescents are no different from those of other age groups.

Exercise-related wheezing and breathlessness are common asthma symptoms in adolescents but only a minority show objective evidence of exercise-induced bronchospasm. Other causes such as hyperventilation or poor fitness can usually be diagnosed and managed by careful clinical assessment.

- Questionnaires
  - The asthma control questionnaire (ACQ) and the asthma control test (ACT) have been validated in adolescents with asthma.

- Quality of life measures
  - QoL scales (such as AQLQ12+) can be used.

- Lung Function
  - Tests of airflow obstruction and airway responsiveness may provide support for a diagnosis of asthma but most adolescents with asthma will have normal lung function.

- Bronchial hyper-reactivity
  - A negative response to an exercise test is helpful in excluding asthma in children with exercise-related breathlessness.

- Anxiety and depressive disorders
  - Major depression, panic attacks and anxiety disorder are commoner in adolescents with asthma and make asthma symptoms more prominent.
  - Brief screening questionnaires for anxiety and depression may help identify those with significant anxiety and depression.

NON-PHARMACOLOGICAL MANAGEMENT

- Adolescents with asthma (and their parents and carers) should be encouraged to avoid exposure to ETS and be informed about the risks and urged not to start smoking.

- Adolescents with asthma should be asked if they smoke personally. If they do and wish to stop, they should be offered advice on how to stop and encouraged to use local NHS smoking cessation services.

- Healthcare professionals should be aware that CAM use is common in adolescents and should ask about its use.
**INHALER DEVICES**

- Adolescent preference for inhaler device should be taken into consideration as a factor in improving adherence to treatment.
- As well as checking inhaler technique it is important to enquire about factors that may affect inhaler device use in real life settings, such as school.
- Consider prescribing a more portable device (as an alternative to a pMDI with spacer) for delivering bronchodilators when away from home.

**LONG TERM OUTLOOK AND ENTRY INTO THE WORK PLACE**

Young adults with asthma have a low awareness of occupations that might worsen asthma (e.g., exposure to dusts, fumes, spray, exertion and temperature changes, see page 22).

- Clinicians should discuss future career choices with adolescents with asthma and highlight occupations that might increase susceptibility to work related asthma symptoms.

**ORGANISATION AND DELIVERY OF CARE**

- **B** School based clinics may be considered for adolescents with asthma to improve attendance.
- **B** Peer-led interventions for adolescents in the school setting should be considered.
- **✓** Integration of school based clinics with primary care services is essential.

**Transition to adult based health care**

Transition to adult services is important for all adolescents with asthma, irrespective of the asthma severity. Transition should be seen as a process and not just the event of transfer to adult services. It should begin early, be planned, involve the young person, and be both age and developmentally appropriate. In the UK, general guidance on transition is available from the RCPCH and DOH websites.

**PATIENT EDUCATION AND SELF MANAGEMENT**

Effective transition care involves preparing adolescents with asthma to take independent responsibility for their own asthma management. Clinicians need to educate and empower adolescents to manage as much of their asthma care as they are capable of doing while supporting parents gradually to hand over responsibility for management to their child.

**Adherence**

- When asked, adolescents with asthma admit their adherence with asthma treatment and with asthma trigger avoidance is often poor.
- Strategies to improve adherence emphasise the importance of focusing on the individual and their lifestyle and using individualised asthma planning and personal goal setting.
### Medications

<table>
<thead>
<tr>
<th>Controller Medications</th>
<th>Action and use</th>
<th>Adverse effects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Inhaled corticosteroids (ICS)</strong> (pMDIs or DPIs) e.g. beclometasone, budesonide, ciclesonide, fluticasone propionate, fluticasone furoate, mometasone, triamcinolone</td>
<td>The most effective anti-inflammatory medications for persistent asthma. ICS reduce symptoms, increase lung function, improve quality of life, and reduce the risk of exacerbations and asthma-related hospitalizations or death. ICS differ in their potency and bioavailability, but most of the benefit is seen at low doses (see Box 8 (p14) for low, medium and high doses of different ICS).</td>
<td>Most patients using ICS do not experience side-effects. Local side-effects include oropharyngeal candidiasis and dysphonia. Use of spacer with pMDI, and rinsing with water and spitting out after inhalation, reduce local side effects. High doses increase the risk of systemic side-effects.</td>
</tr>
<tr>
<td><strong>ICS and long-acting beta2 agonist bronchodilator combinations (ICS/LABA)</strong> (pMDIs or DPIs) e.g. beclometasone/formoterol, budesonide/formoterol, fluticasone furoate/vilanterol, fluticasone propionate/formoterol, fluticasone propionate/salmeterol, and mometasone/formoterol.</td>
<td>When a medium dose of ICS alone fails to achieve good control of asthma, the addition of LABA to ICS improves symptoms, lung function and reduces exacerbations in more patients, more rapidly, than doubling the dose of ICS. Two regimens are available: maintenance ICS/LABA with SABA as reliever, and low-dose combination beclometasone or budesonide with formoterol for maintenance and reliever treatment.</td>
<td>The LABA component may be associated with tachycardia, headache or cramps. Current recommendations are that LABA and ICS are safe for asthma when used in combination. Use of LABA without ICS in asthma is associated with increased risk of adverse outcomes.</td>
</tr>
<tr>
<td><strong>Leukotriene modifiers</strong> (tablets) e.g. montelukast, pranlukast, zafirlukast, zileuton</td>
<td>Target one part of the inflammatory pathway in asthma. Used as an option for controller therapy, particularly in children. Used alone: less effective than low dose ICS; added to ICS: less effective than ICS/LABA.</td>
<td>Few side-effects except elevated liver function tests with zileuton and zafirlukast.</td>
</tr>
<tr>
<td><strong>Chromones</strong> (pMDIs or DPIs) e.g. sodium cromoglycate and nedocromil sodium</td>
<td>Very limited role in long-term treatment of asthma. Weak anti-inflammatory effect, less effective than low-dose ICS. Require meticulous inhaler maintenance.</td>
<td>Side effects are uncommon but include cough upon inhalation and pharyngeal discomfort.</td>
</tr>
<tr>
<td><strong>Anti-IgE</strong> (omalizumab)</td>
<td>A treatment option for patients with severe persistent allergic asthma uncontrolled on Step 4 treatment (high dose ICS/LABA).</td>
<td>Reactions at the site of injection are common but minor. Anaphylaxis is rare.</td>
</tr>
<tr>
<td><strong>Long-acting anticholinergic, tiotropium</strong></td>
<td>An add-on option at Step 4 or 5 bny soft mist inhaler for adults (≥18 years) whose asthma is uncontrolled by ICS ± LABA.</td>
<td>Side-effects are uncommon but include dry mouth</td>
</tr>
<tr>
<td><strong>Systemic corticosteroids</strong> (tablets,suspension or intramuscular (IM) or intravenous (IV) injection) e.g. prednisone, prednisolone, methylprednisolone, hydrocortisone</td>
<td>Short-term treatment (usually 5–7 days in adults) is important early in the treatment of severe acute exacerbations, with main effects seen after 4–6 hours. Oral corticosteroid (OCS) therapy is preferred and is as effective as IM or IV therapy in preventing relapse. Tapering is required if treatment given for more than 2 weeks. Long-term treatment with OCS may be required for some patients with severe asthma.</td>
<td>Short-term use: some adverse effects e.g. hyperglycaemia, gastrointestinal side-effects, mood changes. Long-term use: limited by the risk of significant systemic adverse effects e.g. cataract, glaucoma, osteoporosis, adrenal suppression. Patients should be assessed for osteoporosis risk and treated appropriately.</td>
</tr>
</tbody>
</table>
**RELIEVER MEDICATIONS**

<table>
<thead>
<tr>
<th><strong>Short-acting inhaled beta2-agonist bronchodilators (SABA)</strong> (pMDIs, DPIs and, rarely, solution for nebulization or injection) e.g. salbutamol (albuterol), terbutaline.</th>
<th>Inhaled SABAs are medications of choice for quick relief of asthma symptoms and bronchoconstriction including in acute exacerbations, and for pre-treatment of exercise-induced bronchoconstriction. SABAs should be used only as-needed at the lowest dose and frequency required.</th>
<th>Tremor and tachycardia are commonly reported with initial use of SABA, but tolerance to these effects usually develops rapidly. Excess use, or poor response indicate poor asthma control.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Short-acting anticholinergics</strong> (pMDIs or DPIs) e.g. ipratropium bromide, oxitropium bromide</td>
<td>Long-term use: ipratropium is a less effective reliever medication than SABAs. Short-term use in acute asthma: inhaled ipratropium added to SABA reduces the risk of hospital admission</td>
<td>Dryness of the mouth</td>
</tr>
</tbody>
</table>
TUBERCULOSIS

Pulmonary tuberculosis is a bacterial infection due to *Mycobacterium tuberculosis*, spread by airborne route. After contamination, *M. tuberculosis* multiplies slowly in the lungs: this represents the primary infection.

**Signs and symptoms**

Classic clinical features associated with active pulmonary TB are as follows (elderly individuals with TB may not display typical signs and symptoms):

- Cough
- Weight loss/anorexia
- Fever
- Night sweats
- Hemoptysis
- Chest pain (can also result from tuberculous acute pericarditis)
- Fatigue

Symptoms of tuberculous meningitis may include the following:

- Headache that has been either intermittent or persistent for 2-3 weeks
- Subtle mental status changes that may progress to coma over a period of days to weeks
- Low-grade or absent fever

Symptoms of skeletal TB may include the following:

- Back pain or stiffness
- Lower-extremity paralysis, in as many as half of patients with undiagnosed Pott disease
- Tuberculous arthritis, usually involving only 1 joint (most often the hip or knee, followed by the ankle, elbow, wrist, and shoulder)

Symptoms of genitourinary TB may include the following:

- Flank pain
- Dysuria
- Frequent urination
- In men, a painful scrotal mass, prostatitis, orchitis, or epididymitis
- In women, symptoms mimicking pelvic inflammatory disease

Symptoms of gastrointestinal TB are referable to the infected site and may include the following:

- Nonhealing ulcers of the mouth or anus
- Difficulty swallowing (with esophageal disease)
- Abdominal pain mimicking peptic ulcer disease (with gastric or duodenal infection)
- Malabsorption (with infection of the small intestine)
- Pain, diarrhea, or hematochezia (with infection of the colon)

Physical examination findings associated with TB depend on the organs involved. Patients with pulmonary TB may have the following:

- Abnormal breath sounds, especially over the upper lobes or involved areas
- Rales or bronchial breath signs, indicating lung consolidation

Signs of extrapulmonary TB differ according to the tissues involved and may include the following:

- Confusion
- Coma
- Neurologic deficit
- Chorioretinitis
- Lymphadenopathy
- Cutaneous lesions

The absence of any significant physical findings does not exclude active TB. Classic symptoms are often absent in high-risk patients, particularly those who are immunocompromised or elderly.

The most commonly reported symptom of pulmonary tuberculosis is persistent cough that generally, but not always, is productive of mucus and sometimes blood (hemoptysis). In persons with tuberculosis
the cough is often accompanied by systemic symptoms such as fever, night sweats, and weight loss. In addition, findings such as lymphadenopathy consistent with concurrent extrapulmonary tuberculosis, may be noted, especially in patients with HIV infection. However, chronic cough with sputum production is not always present, even among persons having sputum smears showing acid-fast bacilli. Although many patients with pulmonary tuberculosis have cough, the symptom is not specific to tuberculosis; it can occur in a wide range of respiratory conditions, including acute respiratory tract infections, asthma, and chronic obstructive pulmonary disease. **Having cough of 2 weeks or more in duration serves as the criterion for defining suspected tuberculosis and is used in most national and international guidelines**, particularly in areas of moderate to high prevalence of tuberculosis, as an indication to initiate an evaluation for the disease.

**Diagnosis**

**Guidance on approach to diagnose TB in children**

1. Careful history (including history of TB contact and symptoms consistent with TB)
2. Clinical examination (including growth assessment)
3. Tuberculin skin testing
4. Chest X-ray if available
5. Bacteriological confirmation whenever possible
6. Investigations relevant for suspected pulmonary TB and suspected extrapulmonary TB
7. HIV testing

WHO’s Integrated Management of Childhood Illness (IMCI) program, which is widely used in first-level facilities in low- and middle-income countries states that tuberculosis should be considered in any child with:

- Unexplained weight loss or failure to grow normally;
- Unexplained fever, especially when it continues for more than 2 weeks;
- Chronic cough;
- Exposure to an adult with probable or definite pulmonary infectious tuberculosis.

**Findings on examination that suggest tuberculosis include:**

- Fluid on one side of the chest (reduced air entry, dullness to percussion);
- Enlarged non-tender lymph nodes or a lymph node abscess, especially in the neck;
- Signs of meningitis, especially when these develop over several days and the spinal fluid contains mostly lymphocytes and elevated protein;
- Abdominal swelling, with or without palpable lumps;
- Progressive swelling or deformity in the bone or a joint, including the spine.
Approach to evaluation and management of children in contact with an infectious case of tuberculosis when a tuberculin skin test and chest radiograph are not available

1. If tuberculosis is suspected evaluate as described in Standard 6
2. Treat with isoniazid 10 mg/kg/day for six months
3. No treatment should be given unless the child is HIV-infected in which case give isoniazid 10 mg/kg/day
**Doses of first-line antituberculosis drugs in adults and children**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Recommended Dose in mg/kg Body Weight (Range)</th>
<th>Three Times Weekly</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Isoniazid</strong></td>
<td>[Children: 10 (7–15), maximum 300 mg/day, 5 (4–6), maximum 300 mg/day]</td>
<td>10 (8–12), maximum 900 mg/dose</td>
</tr>
<tr>
<td></td>
<td>[Adults: 10 (8–12), maximum 900 mg/dose]</td>
<td></td>
</tr>
<tr>
<td><strong>Rifampicin</strong></td>
<td>[Children: 15 (10–20), maximum 600 mg/day]</td>
<td></td>
</tr>
<tr>
<td></td>
<td>[Adults: 10 (8–12), maximum 600 mg/day]</td>
<td></td>
</tr>
<tr>
<td><strong>Pyrazinamide</strong></td>
<td>[Children: 35 (30–40), maximum 2,000 mg/day]</td>
<td></td>
</tr>
<tr>
<td></td>
<td>[Adults: 25 (20–30), maximum 2,000 mg/day]</td>
<td></td>
</tr>
<tr>
<td><strong>Ethambutol</strong></td>
<td>[Children: 20 (15–25), maximum 1,000 mg/day]</td>
<td></td>
</tr>
<tr>
<td></td>
<td>[Adults: 15 (15–20), maximum 1,600 mg/day]</td>
<td></td>
</tr>
</tbody>
</table>

* The recommended daily doses of all 4 antituberculosis medicines are higher in children who weigh less than 25 kg than in adults, because the pharmacokinetics are different (and to achieve the same plasma concentration as in adults, the doses need to be increased)

** Same dosing for treatment of active disease and treatment of latent tuberculosis infection
BCG vaccination in neonates (0–4 weeks)

Identify and vaccinate pregnant women eligible for vaccination before the birth of their baby, ideally through antenatal services.

Discuss neonatal BCG vaccination for any baby at increased risk of TB with the parents or legal guardian.

Preferably vaccinate babies at increased risk of TB before discharge from hospital or before handover from midwifery to primary care. Otherwise, vaccinate as soon as possible afterwards, for example, at the 6-week postnatal check.

Provide education and training for postnatal ward staff, midwives, health visitors and other clinicians on identifying babies eligible for vaccination, local service information and providing BCG vaccination, including:

- case definition for at-risk groups to be offered vaccination
- information about the local BCG vaccination policy that can be given verbally, in writing or in any other appropriate format to parents and carers at the routine examination of the baby before discharge
- local service information about BCG vaccination, such as pre-discharge availability of neonatal vaccination, local BCG clinics and referral for BCG vaccination if this is not available in maternity services
- administration of BCG vaccination and contraindications.

Primary care organisations with a high incidence of TB should consider vaccinating all neonates soon after birth.

In areas with a low incidence of TB (see Public Health England’s TB rate bands, published in their Annual Report, primary care organisations should offer BCG vaccination to selected neonates who:

- were born in an area with a high incidence of TB or
- have 1 or more parents or grandparents who were born in a high-incidence country or
- have a family history of TB in the past 5 years.

BCG vaccination for infants (0–5 years) and older children (6–15 years)

Routine BCG vaccination is not recommended for children aged 10–14 years.

- Healthcare professionals should opportunistically identify unvaccinated children older than 4 weeks and younger than 16 years at increased risk of TB who would have qualified for neonatal BCG (see recommendation 1.1.3.4) and provide Mantoux testing and BCG vaccination (if Mantoux-negative).

Mantoux testing should not be done routinely before BCG vaccination in children younger than 6 years unless they have a history of residence or prolonged stay (more than 1 month) in a country with a high incidence of TB.

BCG vaccination for new entrants from high-incidence areas
Offer BCG vaccination to new entrants who are Mantoux- or interferon-gamma release assay-negative who:

- are from high-incidence countries and
- are previously unvaccinated (that is, without adequate documentation or a BCG scar) and
- are aged:
  - younger than 16 years or
  - 16–35 years from sub-Saharan Africa or a country with a TB incidence of 500 per 100,000 or more.

Encouraging uptake among infants, older children and new entrants

Deliver the following interventions in primary care settings to improve uptake of BCG vaccination in people from eligible groups:

- education and support for practice staff, including:
  - raising awareness of relevant guidelines and case definition for at-risk groups
  - promoting BCG and TB testing in eligible groups
- incorporating reminders for staff (prompts about eligibility for BCG) on practice computers (for example, embedded in medical records)
- consider financial incentives for practices for identifying eligible groups for BCG and TB testing
- reminders (‘immunisations due’) and recall (‘immunisations overdue’) for people who are eligible for vaccination or for parents of infants and children who are eligible, as outlined in the Green Book. (This could include written reminders, telephone calls from a member of staff or a computerised auto dialler, text messages or a combination of these approaches.)

Use home visits to give information and advice to people who are disadvantaged on the importance of immunisation. This should be delivered by trained lay health workers, community-based healthcare staff or nurses.

BCG vaccination for healthcare workers

Offer BCG vaccination to healthcare workers and other NHS employees who have contact with patients or clinical specimens, irrespective of age, who:

- are previously unvaccinated (that is, without adequate documentation or a BCG scar) and
- are Mantoux- (or interferon-gamma release assay-) negative

BCG vaccination for contacts of people with active TB

Offer BCG vaccination to Mantoux- (or interferon-gamma release assay-) negative contacts of people with pulmonary and laryngeal TB if they:
• have not been vaccinated previously (that is, there is no adequate documentation or a BCG scar) and

• are aged 35 years or younger or

• are aged 36 years and older and a healthcare or laboratory worker who has contact with patients or clinical materials.

If a new employee from low-incidence setting, who has not had a BCG vaccination, has a positive Mantoux test and a positive interferon-gamma release assay, they should have a medical assessment and a chest X-ray. They should be referred to a TB clinic to determine whether they need TB treatment if the chest X-ray is abnormal, or to determine whether they need treatment of latent TB infection if the chest X-ray is normal.
GASTROINTESTINAL DISORDERS
Diarrhea is the reversal of the normal net absorptive status of water and electrolyte absorption to secretion. The augmented water content in the stools (above the normal value of approximately 10 mL/kg/d in the infant and young child, or 200 g/d in the teenager and adult) is due to an imbalance in the physiology of the small and large intestinal processes involved in the absorption of ions, organic substrates, and thus water.

**Causes of diarrhea with acute onset include the following:**

- **Infections**
  - Enteric infections (including food poisoning
  - Extraintestinal infections
- **Drug-induced**
  - Antibiotic-associated
  - Laxatives
  - Antacids that contain magnesium
  - Opiate withdrawal
  - Other drugs
- **Food allergies or intolerances**
  - Cow's milk protein allergy
  - Soy protein allergy
  - Multiple food allergies
  - Olestra
  - Methylxanthines (caffeine, theobromine, theophylline)
- **Disorders of digestive/absorptive processes**
  - Glucose-galactose malabsorption
  - Sucrase-isomaltase deficiency
  - Late-onset (adult-type) hypolactasia, resulting in lactose intolerance
- **Chemotherapy or radiation-induced enteritis**
- **Surgical conditions**
  - Acute appendicitis
  - Intussusception
- **Vitamin deficiencies**
  - Niacin deficiency
  - Folate deficiency
- **Vitamin toxicity**
  - Vitamin C
  - Niacin, vitamin B3
- **Ingestion of heavy metals or toxins (eg, copper, tin, zinc)**
- **Ingestion of plants (eg, hyacinths, daffodils, azalea, mistletoe, *Amanita* species) mushrooms**
Signs and symptoms

Acute diarrhea is defined as the abrupt onset of 3 or more loose stools per day and lasts no longer than 14 days; chronic or persistent diarrhea is defined as an episode that lasts longer than 14 days.

The clinical presentation and course of diarrhea therefore depend on its cause and on the host. Consider the following to determine the source/cause of the patient’s diarrhea:

- Stool characteristics (e.g., consistency, color, volume, frequency)
- Presence of associated enteric symptoms (e.g., nausea/vomiting, fever, abdominal pain)
- Use of child daycare (common pathogens: rotavirus, astrovirus, calicivirus; Campylobacter,
  Shigella, Giardia, and Cryptosporidium species [spp])
- Food ingestion history (e.g., raw/contaminated foods, food poisoning)
- Water exposure (e.g., swimming pools, marine environment)
- Camping history (possible exposure to contaminated water sources)
- Travel history (common pathogens affect specific regions; also consider rotavirus and Shigella,
  Salmonella, and Campylobacter spp regardless of specific travel history, as these organisms are
  prevalent worldwide)
- Animal exposure (e.g., young dogs/cats: Campylobacter spp; turtles: Salmonella spp)
- Predisposing conditions (e.g., hospitalization, antibiotic use, immunocompromised state)

Signs and symptoms of diarrhea may include the following:

- Dehydration: Lethargy, depressed consciousness, sunken anterior fontanel, dry mucous membranes, sunken eyes, lack of tears, poor skin turgor, delayed capillary refill
- Failure to thrive and malnutrition: Reduced muscle/fat mass or peripheral edema
- Abdominal pain/cramping
- Borborygmi
- Perianal erythema
<table>
<thead>
<tr>
<th>Organism</th>
<th>Incubation</th>
<th>Duration</th>
<th>Vomiting</th>
<th>Fever</th>
<th>Abdominal Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rotavirus</td>
<td>1-7 d</td>
<td>4-8 d</td>
<td>Yes</td>
<td>Low</td>
<td>No</td>
</tr>
<tr>
<td>Adenovirus</td>
<td>8-10 d</td>
<td>5-12 d</td>
<td>Delayed</td>
<td>Low</td>
<td>No</td>
</tr>
<tr>
<td>Norovirus</td>
<td>1-2 d</td>
<td>2 d</td>
<td>Yes</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Astrovirus</td>
<td>1-2 d</td>
<td>4-8 d</td>
<td>+/-</td>
<td>+/-</td>
<td>No</td>
</tr>
<tr>
<td>Calicivirus</td>
<td>1-4 d</td>
<td>4-8 d</td>
<td>Yes</td>
<td>+/-</td>
<td>No</td>
</tr>
<tr>
<td>Aeromonas species</td>
<td>None</td>
<td>0-2 wk</td>
<td>+/-</td>
<td>+/-</td>
<td>No</td>
</tr>
<tr>
<td>Campylobacter species</td>
<td>2-4 d</td>
<td>5-7 d</td>
<td>No</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>C difficile</td>
<td>Variable</td>
<td>Variable</td>
<td>No</td>
<td>Few</td>
<td>Few</td>
</tr>
<tr>
<td>C perfringens</td>
<td>Minimal</td>
<td>1 d</td>
<td>Mild</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Enterohemorrhagic E coli</td>
<td>1-8 d</td>
<td>3-6 d</td>
<td>No</td>
<td>+/-</td>
<td>Yes</td>
</tr>
<tr>
<td>Enterotoxigenic E coli</td>
<td>1-3 d</td>
<td>3-5 d</td>
<td>Yes</td>
<td>Low</td>
<td>Yes</td>
</tr>
<tr>
<td>Plesiomonas species</td>
<td>None</td>
<td>0-2 wk</td>
<td>+/-</td>
<td>+/-</td>
<td>+/-</td>
</tr>
<tr>
<td>Salmonella species</td>
<td>0-3 d</td>
<td>2-7 d</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Shigella species</td>
<td>0-2 d</td>
<td>2-5 d</td>
<td>No</td>
<td>High</td>
<td>Yes</td>
</tr>
<tr>
<td>Vibrio species</td>
<td>0-1 d</td>
<td>5-7 d</td>
<td>Yes</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Y enterocolitica</td>
<td>None</td>
<td>1-46 d</td>
<td>Yes</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Giardia species</td>
<td>2 wk</td>
<td>1+ wk</td>
<td>No</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Cryptosporidium species</td>
<td>5-21 d</td>
<td>Months</td>
<td>No</td>
<td>Low</td>
<td>Yes</td>
</tr>
<tr>
<td>Entamoeba species</td>
<td>5-7 d</td>
<td>1-2+ wk</td>
<td>No</td>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>
Food history can be helpful.
- Ingestion of raw or contaminated food is a common cause of infectious diarrhea.
- Organisms that cause food poisoning include the following:
  - Dairy food - *Campylobacter* and *Salmonella* species
  - Eggs - *Salmonella* species
  - Meats - *C. perfringens* and *Aeromonas, Campylobacter, and Salmonella* species
  - Ground beef - Enterohemorrhagic *E coli*
  - Poultry - *Campylobacter* species
  - Pork - *C. perfringens, Y enterocolitica*
  - Seafood - Astrovirus and *Aeromonas, Plesiomonas, and Vibrio* species
  - Oysters - Calicivirus and *Plesiomonas and Vibrio* species
  - Vegetables - *Aeromonas species and C perfringens*

Water exposure can contribute to diarrhea.
- Water is a major reservoir for many organisms that cause diarrhea.
- Swimming pools have been associated with outbreaks of infection with *Shigella* species; *Aeromonas* organisms are associated with exposure to the marine environment.
- *Giardia, Cryptosporidium, and Entamoeba* organisms are resistant to water chlorination; therefore, exposure to contaminated water should raise index of suspicion for these parasites.

A history of camping suggests exposure to water sources contaminated with *Giardia* organisms.

Animal exposure can contribute to diarrhea.
- Exposure to young dogs or cats is associated with *Campylobacter* organisms.

Dehydration Severity, Signs, and Symptoms

<table>
<thead>
<tr>
<th>Hydration</th>
<th>0-5% Dehydration (Mild)</th>
<th>5-10% Dehydration (Moderate)</th>
<th>10% or More (Severe)</th>
</tr>
</thead>
<tbody>
<tr>
<td>General</td>
<td>Well</td>
<td>Restless</td>
<td>Lethargic</td>
</tr>
<tr>
<td>Eyes</td>
<td>Normal</td>
<td>Sunken</td>
<td>Very sunken</td>
</tr>
<tr>
<td>Tears</td>
<td>Present</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>Mouth</td>
<td>Moist</td>
<td>Dry</td>
<td>Very dry</td>
</tr>
<tr>
<td>Thirst</td>
<td>Drinks normally</td>
<td>Thirsty</td>
<td>Drinks poorly</td>
</tr>
<tr>
<td>Skin</td>
<td>Pinch retracts immediately</td>
<td>Pinch retracts slowly</td>
<td>Pinch stays folded</td>
</tr>
</tbody>
</table>
Diagnosis

Fecal laboratory studies include the following:

- Examination for ova and parasites
- Leukocyte count
- pH level: A pH level of 5.5 or less or the presence of reducing substances indicates carbohydrate intolerance, which is usually secondary to viral illness
- Examination of exudates for presence/absence of leukocytes
- Cultures: Always culture for *Salmonella*, *Shigella*, and *Campylobacter* spp and *Y. enterocolitica* in the presence of clinical signs of colitis or if fecal leukocytes are present; look for *Clostridium difficile* in those with diarrhea characterized by colitis and/or bloody stools; assess for *Escherichia coli*, particularly O157:H7, with bloody diarrhea and a history of eating ground beef; screen for *Vibrio* and *Plesiomonas* spp with a history of eating raw seafood or foreign travel
- Enzyme immunoassay for rotavirus or adenovirus antigens
- Latex agglutination assay for rotavirus

Management

Acute-onset diarrhea is usually self-limited; however, an acute infection can have a protracted course. Management is generally supportive: In most cases, the best option for treatment of acute-onset diarrhea is the early use of ORT.

Children with diarrhea may benefit from green tea and pomegranate extract.

Strains of probiotics (defined as live microorganisms that when ingested in adequate doses, provide a benefit to the host) have been found to be effective as an adjunct when treating children with acute diarrhea.

Indications for medical evaluation of children with acute diarrhea include the following:

- Younger than 3 months
- Weight of less than 8 kg
- History of premature birth, chronic medical conditions, or concurrent illness
- Fever of 38°C or higher in infants younger than 3 months or 39°C or higher in children aged 3-36 months
- Visible blood in the stool
- High-output diarrhea
- Persistent emesis
- Signs of dehydration as reported by caregiver, including sunken eyes, decreased tears, dry mucous membranes, and decreased urine output
- Mental status changes
- Inadequate responses to ORT or caregiver unable to administer ORT
General principles:

1. Prevent or treat dehydration: rehydration consists of prompt replacement of fluid and electrolyte losses as required, until the diarrhea stops.
2. Administer zinc sulfate to children under 5 years.
   - Children under 6 months: 10 mg once daily (1/2 tablet once daily) for 10 days
   - Children from 6 months to 5 years: 20 mg once daily (1 tablet once daily) for 10 days
   Place the half-tablet or full tablet in a teaspoon, add a bit of water to dissolve it, and give the entire spoonful to the child. Do not administer this treatment if the child receives ready-to-use therapeutic food (RUTF) which already contains zinc.
3. Prevent malnutrition.
4. Do not systematically administer antimicrobials: only certain diarrheas require antibiotics (see antimicrobial treatment, following page).
5. Do not administer anti-diarrheal drugs or antiemetics.
6. Treat the underlying condition if any (malaria, otitis, respiratory infection, etc.).

Treatment of dehydration due to diarrhea includes the following:

Minimal or no dehydration

- Rehydration therapy - Not applicable
- Replacement of losses
  - Less than 10 kg body weight - 60-120 mL oral rehydration solution for each diarrhea stool or vomiting episode
  - More than 10 kg body weight - 120-140 mL oral rehydration solution for each diarrhea stool or vomiting episode

Mild-to-moderate dehydration

- Rehydration therapy - Oral rehydration solution (50-100 mL/kg over 3-4 h)
- Replacement of losses
  - Less than 10 kg body weight - 60-120 mL oral rehydration solution for each diarrhea stool or vomiting episode
  - More than 10 kg body weight - 120-140 mL oral rehydration solution for each diarrhea stool or vomiting episode

Severe dehydration (at hospital level)

- Rehydration therapy - Intravenous lactated Ringer solution or normal saline (20 mL/kg until perfusion and mental status improve), followed by 100 mL/kg oral rehydration solution over 4 hours or 5% dextrose (half normal saline) intravenously at twice maintenance fluid rates
- Replacement of losses
  - Less than 10 kg body weight - 60-120 mL oral rehydration solution for each diarrhea stool or vomiting episode
  - More than 10 kg body weight - 120-140 mL oral rehydration solution for each diarrhea stool or vomiting episode
  - If unable to drink, administer through nasogastric tube or intravenously administer 5% dextrose (one fourth normal saline) with 20 mEq/L potassium chloride

Not all commercial ORT formulas promote optimal absorption of electrolytes, water, and nutrients. The ideal solution has a low osmolarity (210-250) and a sodium content of 50-60 mmol/L.
Pharmacotherapy

Vaccines (eg, rotavirus) can help increase resistance to infection. Antimicrobial and antiparasitic agents may be used to treat diarrhea caused by specific organisms and/or clinical circumstances. Such medications include the following:

- Cefixime
- Ceftriaxone
- Cefotaxime
- Erythromycin
- Furazolidone
- Iodoquinol
- Metronidazole
- Vancomycin
- Rifaximin

Therapies recommended for some nonviral diarrheas include the following:

- *Aeromonas* species: Use cefixime and most third-generation and fourth-generation cephalosporins.
- *Campylobacter* species: Erythromycin shortens illness duration and shedding.
- *C difficile*: Discontinue potential causative antibiotics. If antibiotics cannot be stopped or this does not result in resolution, use oral metronidazole or vancomycin. Vancomycin is reserved for the child who is seriously ill.
- *C perfringens*: Do not treat with antibiotics.
- *Cryptosporidium parvum*: Administer paromomycin; however, effectiveness is not proven. Nitazoxanide, a newer anthelmintic, is effective against *C parvum*.
- *Entamoeba histolytica*: Metronidazole followed by iodoquinol or paromomycin is administered in symptomatic patients. Asymptomatic carriers in nonendemic areas should receive iodoquinol or paromomycin.
- *E coli*: Trimethoprim-sulfamethoxazole (TMP-SMX) should be administered if moderate or severe diarrhea is noted; antibiotic treatment may increase likelihood of hemolytic-uremic syndrome (HUS). Parenteral second-generation or third-generation cephalosporin is indicated for systemic complications.
- *G lamblia*: Metronidazole or nitazoxanide can be used.
- *Plesiomonas* species: Use TMP-SMX or any cephalosporin.
- *Salmonella* species: Treatment prolongs carrier state, is associated with relapse, and is not indicated for nontyphoid-uncomplicated diarrhea. Treat infants younger than 3 months and high-risk patients (eg, immunocompromised, sickle cell disease). TMP-SMX is first-line medication; however, resistance occurs. Use ceftriaxone and cefotaxime for invasive disease.
- *Shigella* species: Treatment shortens illness duration and shedding but does not prevent complications. TMP-SMX is first-line medication; however, resistance occurs. Cefixime, ceftriaxone, and cefotaxime are recommended for invasive disease.
- *V cholerae*: Treat infected individuals and contacts. Doxycycline is the first-line antibiotic, and erythromycin is second-line antibiotic.
- *Yersinia* species: TMP-SMX, cefixime, ceftriaxone, and cefotaxime are used. Treatment does not shorten disease duration; reserve for complicated cases.
GASTROESOPHAGEAL REFLUX DISEASE

Gastroesophageal reflux disease occurs when the amount of gastric juice that refluxes into the esophagus exceeds the normal limit, causing symptoms with or without associated esophageal mucosal injury.

**Signs and symptoms**

Typical esophageal symptoms include the following:
- Heartburn
- Regurgitation
- Dysphagia

Abnormal reflux can cause atypical (extraesophageal) symptoms, such as the following:
- Coughing and/or wheezing
- Hoarseness, sore throat
- Otitis media
- Non cardiac chest pain
- Enamel erosion or other dental manifestations

A history of nausea, vomiting, or regurgitation should alert the physician to evaluate for delayed gastric emptying.

**Management**

Treatment of gastroesophageal reflux disease involves a stepwise approach. The goals are to control symptoms, to heal esophagitis, and to prevent recurrent esophagitis or other complications. The treatment is based on lifestyle modification and control of gastric acid secretion through medical therapy with antacids or proton pump inhibitors or surgical treatment with corrective antireflux surgery.

**Nonpharmacotherapy**

Lifestyle modifications used in the management of gastroesophageal reflux disease include the following:
- Losing weight (if overweight)
- Avoiding alcohol, chocolate, citrus juice, and tomato-based products
- Avoiding peppermint, coffee, and possibly the onion family
- Eating small, frequent meals rather than large meals
- Waiting 3 hours after a meal to lie down
- Refraining from ingesting food (except liquids) within 3 hours of bedtime
- Avoiding bending or stooping positions

**Pharmacotherapy**

The following medications are used in the management of gastroesophageal reflux disease:
- H2 receptor antagonists (eg, ranitidine, cimetidine, famotidine, nizatidine)
- Proton pump inhibitors (eg, omeprazole, pantoprazole)
- Prokinetic agents (eg, aluminum hydroxide)
- Antacids (eg, aluminum hydroxide, magnesium hydroxide)

**Antacids**

Antacids were the standard treatment and are still effective in controlling mild symptoms of GERD. Antacids should be taken after each meal and at bedtime.

**Aluminum hydroxide** PO: 1.5 to 3 g/day in 3 divided doses one hour after meals or instruct the patient to take 500 mg at the time of a painful attack.

**H2 receptor antagonists and H2 blocker therapy**
H2 receptor antagonists are the first-line agents for patients with mild to moderate symptoms and grades I-II esophagitis. Options include ranitidine (Zantac) 150 mg PO q12hr

**Proton pump inhibitors**

PPIs are the most powerful medications available for treating GERD. These agents should be used only when this condition has been objectively documented. Omeprazole 20 mg PO qDay for 4 weeks
**PEPTIC ULCER DISEASE**

Gastric and duodenal ulcers usually cannot be differentiated based on history alone, although some findings may be suggestive. Epigastric pain is the most common symptom of both gastric and duodenal ulcers. It is characterized by a gnawing or burning sensation and occurs after meals—classically, shortly after meals with gastric ulcer and 2-3 hours afterward with duodenal ulcer.

**Possible manifestations include the following:**
- Dyspepsia, including belching, bloating, distention, and fatty food intolerance
- Heartburn
- Chest discomfort
- Hematemesis or melena resulting from gastrointestinal bleeding. Melena may be intermittent over several days or multiple episodes in a single day.
- Rarely, a briskly bleeding ulcer can present as hematochezia.
- Symptoms consistent with anemia (e.g., fatigue, dyspnea) may be present
- Sudden onset of symptoms may indicate perforation.
- NSAID-induced gastritis or ulcers may be silent, especially in elderly patients.
- Only 20-25% of patients with symptoms suggestive of peptic ulceration are found on investigation to have a peptic ulcer.

Alarm features that warrant prompt gastroenterology referral include the following:
- Bleeding or anemia
- Early satiety
- Unexplained weight loss
- Progressive dysphagia or odynophagia
- Recurrent vomiting
- Family history of GI cancer

**Clinical findings are few and nonspecific and include the following:**
- Epigastric tenderness (usually mild)
- Right upper quadrant tenderness may suggest a biliary etiology or, less frequently, PUD.
- Guaiac-positive stool resulting from occult blood loss
- Melena resulting from acute or subacute gastrointestinal bleeding
- Succussion splash resulting from partial or complete gastric outlet obstruction

**Treatment of non-complicated ulcers**

For an isolated episode:
- Identify patients taking NSAID or acetylsalicylic acid; stop treatment;
- Encourage patients to avoid alcohol and tobacco use;

**Omeprazole:** 40 mg PO qDay for 4-8 weeks

**Duodenal Ulcer**

**Omeprazole:** 20 mg PO qDay for 4-8 weeks

A special diet is not indicated for patients with duodenal ulcers. It is a common-sense approach to avoid any food or beverages that may aggravate symptoms.

Surgical consultation is recommended for all patients with bleeding ulcers, especially those patients who are at high risk of significant bleeding. Such ulcers include those that have caused hemodynamic instability, those that are actively bleeding.
Categorized as an idiopathic disease, aphthous ulcers are frequently misdiagnosed, treated incorrectly, or simply ignored.

The 3 categories of recurrent aphthous ulcers (canker sores) are as follows:

- **Minor aphthous ulcers** (80-85% of recurrent aphthous ulcers [canker sores]) are 1-10 mm in diameter and heal spontaneously in 7-10 days.
- **Major aphthous ulcers** (also called Sutton disease) constitute 10-15% of recurrent aphthous ulcers (canker sores). These lesions are greater than 10 mm in diameter, take 10-30 days or more to heal, and may leave scars.
- **Herpetiform ulcers** (5-10% of recurrent aphthous ulcers [canker sores]) are multiple, clustered, 1-mm to 3-mm lesions that may coalesce into plaques. These usually heal in 7-10 days.

Patients typically describe a prodromal stage of a burning or pricking sensation of the oral mucosa 1-2 days before the ulcer appears.

**Treatment & Management**

The primary goals of medical therapy in patients with aphthous ulcers (canker sores) are pain relief, maintenance of fluid and nutrition intake, early resolution, and prevention of recurrence. Most patients with minor or herpetiform aphthae should be treated empirically before extensive and costly studies are initiated. Treatment of recurrent aphthous ulcers (canker sores) typically includes anti-inflammatory and/or symptomatic therapy, whereas immunomodulators are rarely used, except in severe, refractory cases.

**Lidocaine (Xylocaine)**

Available as gel or viscous PO solution. Does not shorten healing time but may help patient to tolerate eating and drinking. Pain relief may be short, and frequent applications may be necessary.

**Diphenhydramine elixir (Benadryl)**

First-line antihistamine for topical treatment of localized skin and mucus-membrane irritation. May be applied directly to ulcerated submucosal tissue. Relieves PO pain in some patients.
CONSTIPATION

Constipation is the most common digestive complaint. It is a symptom rather than a disease. Despite its frequency, it often remains unrecognized until the patient develops sequelae, such as anorectal disorders or diverticular disease.

Signs and symptoms
According to the Rome III criteria for constipation, a patient must have experienced at least 2 of the following symptoms over the preceding 3 months:
- Fewer than 3 bowel movements per week
- Straining
- Lumpy or hard stools
- Sensation of anorectal obstruction
- Sensation of incomplete defecation
- Manual maneuvering required to defecate

A constipated patient may be otherwise totally asymptomatic or may complain of 1 or more of the following:
- Abdominal bloating
- Pain on defecation
- Rectal bleeding
- Spurious diarrhea
- Low back pain

Management
Initial treatment measures for constipation include manual disimpaction and transrectal enemas. Medical care should focus on dietary change and exercise rather than laxatives, enemas, and suppositories, none of which really address the underlying problem.

The key to treating most patients with constipation is correction of dietary deficiencies, which generally involves increasing intake of fiber and fluid and decreasing the use of constipating agents (eg, milk products, coffee, tea, alcohol).

- Bulk-forming agents (fibers; eg, psyllium): arguably the best and least expensive medication for long-term treatment
- Rapidly acting lubricants (eg, mineral oil): Used for acute or subacute management of constipation
- Prokinetics (eg, tegaserod): Proposed for use with severe constipation-predominant symptoms
- Stimulant laxatives (eg, senna): Over-the-counter agents commonly but inappropriately used for long-term treatment of constipation
PEDIATRIC CONSTIPATION

For practical clinical purposes, constipation is generally defined as infrequent defecation, painful defecation, or both. In most cases, parents are worried that their child's stools are too large, too hard, not frequent enough, and/or painful to pass.

The Paris Consensus on Childhood Constipation Terminology (PACCT) defines constipation as "a period of 8 weeks with at least 2 of the following symptoms: defecation frequency less than 3 times per week, fecal incontinence frequency greater than once per week, passage of large stools that clog the toilet, palpable abdominal or rectal fecal mass, stool withholding behavior, or painful defecation.

Clinical Presentation

In young infants, functional constipation often develops at the time of a dietary transition (eg, from breast milk to formula, the addition of solid foods into the diet, from formula to whole milk).

In toddlers, functional constipation often develops near the time of toilet training. In toddlers and young children, constipation may develop following an illness associated with either a severe diaper dermatitis or dehydration.

Treatment & Management

Although constipation is an extremely common problem among children, few studies have systematically evaluated different management strategies.

Dietary changes, such as increasing the child's intake of fluids and carbohydrates, are commonly recommended as part of the treatment of constipation. Complex carbohydrates and unabsorbable sugars (eg, sorbitol) are found in many fruit juices (eg, prune, pear, apple). These carbohydrates increase stool frequency by increasing fecal water content.

In infants and young children, it is appropriate to consider removing cow-milk protein from the diet for a period is appropriate, because chronic constipation may be precipitated by ingestion of cow-milk proteins.

In several randomized trials, laxatives have been shown to be beneficial in the treatment of chronic childhood constipation. Studies have also shown that polyethylene glycol, mineral oil, magnesium hydroxide, and lactulose are effective and can be used for prolonged time periods without risk.
Polyethylene glycol (Dulcolax Balance)
<6 months: Safety and efficacy not established
≥6 months: 0.5-1.5 g/kg PO once daily for no longer than 2 weeks; adjusted to effect; not to exceed 17 g/day

Magnesium hydroxide (OTC) Milk of Magnesia
<2 years: Safety and efficacy not established
Suspension
- 2-6 years: 5-15 mL/day of regular-strength liquid PO at bedtime or in divided doses
- 6-12 years: 15-30 mL/day (400 mg/5 mL) or 7.5-15 mL/day (800 mg/5 mL) PO at bedtime or in divided doses
- ≥12 years: 30-60 mL/day (400 mg/5 mL) or 15-30 mL/day (800 mg/5 mL) PO at bedtime or in divided doses

Bisacodyl (OTC) Dulcolax
<6 years: PO administration not recommended, because of requirement to swallow tablets
6-12 years: 5 mg or 0.3 mg/kg PO at bedtime or before breakfast
>12 years: 5-15 mg PO at bedtime
Dermatological examination

Observe the type of lesion:

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Macule</td>
<td>A flat, colored lesion, &lt;2 cm in diameter, not raised above the surface of the surrounding skin. A &quot;freckle,&quot; or ephelid, is a prototype pigmented macule.</td>
</tr>
<tr>
<td>Patch</td>
<td>A large (&gt;2 cm) flat lesion with a color different from the surrounding skin. This differs from a macule only in size.</td>
</tr>
<tr>
<td>Papule</td>
<td>A small, solid lesion, &lt;0.5 cm in diameter, raised above the surface of the surrounding skin and, hence, palpable (e.g., a closed comedone, or whitehead, in acne).</td>
</tr>
<tr>
<td>Nodule</td>
<td>A larger (0.5–5.0 cm), firm lesion raised above the surface of the surrounding skin. This differs from a papule only in size (e.g., a dermal nevomelanocytic nevus).</td>
</tr>
<tr>
<td>Tumor</td>
<td>A solid, raised growth &gt;5 cm in diameter.</td>
</tr>
<tr>
<td>Plaque</td>
<td>A large (&gt;1 cm), flat-topped, raised lesion; edges may either be distinct (e.g., in psoriasis) or gradually blend with surrounding skin (e.g., in eczematous dermatitis).</td>
</tr>
<tr>
<td>Vesicle</td>
<td>A small, fluid-filled lesion, &lt;0.5 cm in diameter, raised above the plane of surrounding skin. Fluid is often visible, and the lesions are translucent [e.g., vesicles in allergic contact dermatitis caused by <em>Toxicodendron</em> (poison ivy)].</td>
</tr>
<tr>
<td>Pustule</td>
<td>A vesicle filled with leukocytes. Note: The presence of pustules does not necessarily signify the existence of an infection.</td>
</tr>
<tr>
<td>Bulla</td>
<td>A fluid-filled, raised, often translucent lesion &gt;0.5 cm in diameter.</td>
</tr>
<tr>
<td>Wheal</td>
<td>A raised, erythematous, edematous papule or plaque, usually representing short-lived vasodilatation and vasopermeability.</td>
</tr>
<tr>
<td>Telangiectasia</td>
<td>A dilated, superficial blood vessel.</td>
</tr>
</tbody>
</table>

Look at the distribution of the lesions over the body; observe their arrangement: isolated, clustered, linear, annular (in a ring). Ask if the lesions are itchy.

Look for a possible cause: insect bites; scabies, lice, other parasitic skin infections; contact with plants, animals, jewellery, detergents, etc.

Ask about any ongoing treatment: topical, oral or parenteral.

Look for local or regional signs (secondary infection, lymphangitis, adenopathy, erysipelas) and/or systemic signs (fever, septicemia, distant infectious focus).

Consider the sanitary condition of the family, particularly for contagious skin diseases (scabies, scalp ringworm, lice).
Check tetanus vaccination status.

Patients with skin disease often present late. At this stage, primary lesions and specific signs may be masked by secondary infection. In these cases, it is necessary to reexamine the patient, after treating the secondary infection, in order to identify and treat the underlying skin disease.
SCABIES

Human scabies is an intensely pruritic skin infestation caused by the host-specific mite *Sarcoptes scabiei hominis*. This mite can travel from the infected person to another person. Most people get scabies from direct, skin-to-skin contact. Less often, people pick up mites from infested items such as bedding, clothes, and furniture. The mite can survive for about 48 to 72 hours without human contact.

**Signs and symptoms**

Burrows are a pathognomonic sign and represent the intraepidermal tunnel created by the moving female mite. They appear as serpiginous, grayish, threadlike elevations in the superficial epidermis, ranging from 2-10 mm long.

Scabies can develop anywhere on the skin. The mites, however, prefer to burrow in certain parts of the body.

High-yield locations for burrows include the following:

- Webbed spaces of the fingers
- Flexor surfaces of the wrists
- Elbows
- Axillae
- Belt line
- Feet
- Scrotum (men)
- Areolae (women)

Scabies in children
Some children develop widespread scabies. The scabies rash can cover most of the body. Even a child’s palms, soles, and scalp can be infested with mites. In babies, the rash often appears on the palms and soles. Babies who have scabies are very irritable and often do not want to eat or sleep. Children, too, are often very irritable. The itch can keep them awake at night.

In geriatric patients, scabies demonstrates a propensity for the back, often appearing as excoriations. In infants and small children, burrows are commonly located on the palms and soles. One- to 3-mm erythematous papules and vesicles are seen in typical distributions in adults. The vesicles are discrete lesions filled with clear fluid, although the fluid may appear cloudy if the vesicle is more than a few days old.

**Nodular scabies**
Nodules occur in 7-10% of patients with scabies, particularly young children. In neonates unable to scratch, pinkish brown nodules ranging in size from 2-20 mm in diameter may develop.

**Crusted scabies**
In crusted scabies, lesions are often hyperkeratotic and crusted and cover large areas. Marked scaling is common, and pruritus may be minimal or absent. Nail dystrophy and scalp lesions may be prominent. The hands and arms are the usual locations for lesions, but all sites are vulnerable.

**Secondary lesions**
These lesions result from scratching, secondary infection, and/or the host’s immune response against the scabies mites and their products. Characteristic findings include the following:
- Excoriations
- Widespread eczematous dermatitis
- Honey-colored crusting
- Postinflammatory hyperpigmentation
- Erythroderma
- Prurigo nodules
- Frank pyoderma

**Diagnosis**
The diagnosis of scabies can often be made clinically in patients with a pruritic rash and characteristic linear burrows.

**Management**
**Who needs treatment?**
The person diagnosed with scabies and everyone who has had close contact with that person need treatment. Even people who do not have any signs or symptoms must be treated. This is the only way to prevent new outbreaks of scabies weeks later. People who should be treated include:
- Everyone who lives with the person.
- Recent sexual partners.

Most people can be cured with a medicine that they apply to their skin. These medicines are often applied to all skin from the neck down. Infants and young children often need treatment for their scalp and face, too.

Most medicine is applied at bedtime. The medicine is then washed off when the patient wakes up. You may need to repeat this process one week later.

Scabies treatment includes administration of a scabicidal agent (eg, permethrin, lindane, or ivermectin), as well as an appropriate antimicrobial agent if a secondary infection has developed. 5% permethrin cream: This is the most common treatment for scabies. It is safe for children as young as 1 month old and women who are pregnant.
Pruritus may be partially alleviated with an oral antihistamine, such as hydroxyzine hydrochloride (Atarax), diphenhydramine hydrochloride (Benadryl), or cyproheptadine hydrochloride (Periactin). In rare cases, prednisone may be used to treat severe pruritus. Because of their heavy mite burden, patients with crusted scabies may require repeated applications of topical scabicides or treatment that simultaneously uses oral ivermectin and a topical agent, such as permethrin.

The preferred treatment is 5% permethrin (lotion or cream):
Child > 2 months and adult: one application, with a contact time of 8 hours, then rinse off. Permethrin is easier to use (no dilution required), and preferred over benzyl benzoate in children, and pregnant/lactating women. One application may be sufficient, but a second application 7 days later reduces the risk of treatment failure.

or, if not available, benzyl benzoate 25% lotion:
A second application of benzyl benzoate (e.g. after 24 hours, with a rinse between the 2 applications; or two successive applications, 10 minutes apart, when the first application has dried, with a rinse after 24 hours) reduces the risk of treatment failure.
Second applications are not recommended in pregnant women and children < 2 years.
For infants: Use mixed with three parts of water, just one time
For older children: Use mixed with an equal quantity of water, just one time.

**RED FLAG - Caution**
If you get treatment and people with whom you live or have close contact do not get treatment, you can get the mites again. People do not have to have signs and symptoms of scabies to have mites on their skin. Someone who has never had scabies may not have any symptoms for 2 to 6 weeks.
You should then massage the medicine onto clean, dry skin. The medicine must remain on the skin for 8 to 14 hours. You will then wash off the medicine. For this reason, most people apply the medicine at bedtime and wash it off in the morning.

**Apply the medicine from your neck to your toes.** This includes all skin between your neck and toes — the skin around your nails, the crease between your buttocks, and the skin between your toes. Infants, children, and the elderly often need to treat their scalp, temples, and forehead. You should never apply medicine to the nose, lips, eyelids, nor around the eyes or mouth.

**If you wash your hands after applying the medicine, be sure to reapply the medicine to your hands.** Mites like to burrow in the hands, so it is important to treat the hands. Be sure to apply the medicine to the skin between your fingers.

**The day you start treatment, wash your clothes, bedding, towels, and washcloths.** Mites can survive for a few days without human skin. If a mite survives, you can get scabies again. To prevent this, you must wash clothes, sheets, comforters, blankets, towels, and other items. Be sure to follow these instructions when washing:

- Wash all items in a washing machine, using the hottest water possible.
- After washing, dry everything in a dryer, using the hot setting.
- If you cannot wash something in a washing machine and then dry it in a dryer, take it to a dry cleaner or seal it in plastic bag for at least 1 week.
- Items that have not touched your skin for more than 1 week generally do not need washing. If you are not sure whether you wore clothing or used an item within the past week, be sure to wash and dry it.
URTICARIA

Urticaria, commonly referred to as hives, appears as raised, well-circumscribed areas of erythema and edema involving the dermis and epidermis that are very pruritic (see the image below). It may be acute (<6 wk) or chronic (>6 wk).

For chronic or recurrent urticaria, important considerations include previous causative factors and the effectiveness of various treatments, as follows:

- Precipitants, such as heat, cold, pressure, exercise, sunlight, emotional stress, or chronic medical conditions
- Other medical conditions that can cause pruritus (usually without rash), such as diabetes mellitus, chronic renal insufficiency, primary biliary cirrhosis, or other nonurticarial dermatologic disorders
- Family and personal medical history of angioedema - Characteristics of angioedema include vasodilation and exudation of plasma into the deeper tissues more so than with simple urticaria; angioedema can occur with and without the wheals of simple urticaria and presents clinically as subcutaneous swelling that is generally nonpitting and nonpruritic.

For acute urticaria, the main consideration involves possible precipitants, such as the following:

- Recent illness
- Medication use
- IV radiographic contrast media
- Travel
- Foods
- New perfumes, hair dyes, detergents, lotions, creams, or clothes
- Exposure to new pets (dander), dust, mold, chemicals, or plants
- Pregnancy (usually occurs in last trimester and typically resolves spontaneously soon after delivery)
- Contact with nickel, rubber, latex, industrial chemicals, and nail polish
- Sun or cold exposure
- Exercise
- Alcohol ingestion

The physical examination should focus on conditions that might precipitate urticaria or could be potentially life-threatening and include the following:
- Angioedema of the lips, tongue, or larynx
- Individual urticarial lesions that are painful, long-lasting (>24 h), or ecchymotic or that leave residual hyperpigmentation or ecchymosis upon resolution are suggestive of urticarial vasculitis
- Systemic signs or symptoms
  - Scleral icterus, hepatic enlargement, or tenderness
  - Thyromegaly
  - Pneumonia or bronchospasm (asthma)
  - Cutaneous evidence of bacterial or fungal infection

Acute urticaria may rarely progress to life-threatening angioedema or anaphylactic shock in a very short period, although anaphylactic shock is usually of rapid onset with no urticaria or angioedema.

**Prehospital measures may include the following when there is concern for anaphylactic shock:**
- If associated angioedema is present, IM epinephrine
- If associated bronchospasm is present, nebulized albuterol
- Other measures may be appropriate, such as continuous ECG, blood pressure and pulse oximetry monitoring; administering intravenous crystalloids if the patient is hypotensive; and administering oxygen.
- Diphenhydramine or hydroxyzine, if available

Management of urticaria is focused on treating the symptoms and typically is not altered by underlying etiology. The mainstay is avoidance of further exposure to the antigen causing urticaria.

**Pharmacologic treatment options include the following:**
- Antihistamines, primarily those that block H1 receptors with low sedating activity, such as fexofenadine, loratadine, desloratadine, cetirizine, and levocetirizine are first-line therapy;
- Glucocorticoids
- Methotrexate, colchicine, dapsone, indomethacin, and hydroxychloroquine (for urticarial vasculitis)
- Patients with chronic or recurrent urticaria should be referred to a dermatologist for further evaluation and management.
LICE

Louse infestation remains a major problem throughout the world, making the diagnosis and treatment of louse infestation a common task in general medical practice. Pediculosis capitis results in significant psychological stress in children and adults and missed schooldays in children, particularly in areas with a no-nit policy. Lice are ectoparasites that live on the body. Lice feed on human blood after piercing the skin and injecting saliva, which may cause pruritus due to an allergic reaction. Lice crawl but cannot fly or hop.

Causative organisms include *P. humanus capitis* (head louse), *P. humanus corporis* (body louse), and *P. pubis* (pubic louse).

Pediculosis capitis is spread by direct contact with an infected person. Head-to-head contact with an infested individual at school, at home, and while playing may result in head lice infestation; personal hygiene and environmental cleanliness are not risk factors. Fomites, such as clothing, headgear, hats, combs, hairbrushes, hair barrettes, may occasionally play a role in the spread of head lice.

Risk factors for body lice infestation include close, crowded living situations (eg, crowded buses and trains, prison camps) and infrequent washing and/or changing of clothing. *P. corporis* can be acquired via bedding, towels, or clothing recently used by an individual infested with lice; thus, individuals who are homeless, who are impoverished, or who are living in refugee camps are at high risk for infestation.

Intimate or sexual contact with an individual who is infested with pubic lice is a common risk factor for pubic lice infestation. Risk factors for infestation of the pubic louse include sexual promiscuity and crowded living conditions. Contact with clothing, bedding, and towels used by an infested individual may occasionally be the cause of infestation. It is a myth that pubic lice are spread by sitting on a toilet seat; pubic lice’s feet are not designed to walk on smooth surfaces such a toilet seats, and the lice cannot live for long away from a warm human body.

Pruritus is the most common symptom of infestation. Children often have trouble sleeping because of intense pruritus at night. Areas affected in head louse infestation include the scalp, the back of the neck, and postauricular areas. Scratching can cause secondary infection with bacterial sores. However, lice infestation may be asymptomatic, particularly if it is the first infestation and if the infestation is light. Patients infested with *P. corporis* experience nocturnal pruritus, particularly in the axillary, truncal, and groin regions, when the lice move from the clothing to the body to feed. The investigating physician should inquire about the patient’s socioeconomic status and living conditions, as body louse infestation generally affects people of low socioeconomic status. Adults infested with *P. pubis* are usually sexually active and have groin and body hair involvement. Involvement with pruritus of the groin, axillae, eyelashes, or eyebrows can help differentiate *P. pubis* infestation from head or body louse infestation. Parents of children infested with *P. pubis* on the eyelids and/or eyebrows should be questioned about also being infested because the parents are usually the source of infestation.

Treatment & Management

Treatment of pediculosis has 2 aspects: medication and environmental control measures. Increasing emphasis is being placed on understanding the life cycle of lice in order to provide effective treatment. Not all treatment preparations are ovicidal. For weakly ovicidal or non-ovicidal pediculicides, routine retreatment is recommended typically 7-9 days after the first treatment. For strongly ovicidal pediculicides, retreatment is recommended only if live (ie, crawling) lice are still present after treatment. Retreatment should ideally occur after all eggs have hatched but before new eggs are produced. It is extremely important to use medications as directed to ensure total eradication of the lice.
through their life cycle. In addition, all infested persons in a household and their infested close contacts and bedmates should be treated at the same time.

Head lice have been found on hats, scarves, brushes, combs, hair accessories, linens, towels, and stuffed animals. Since exposure to these fomites could result in infestation, it is recommended that such items used by the infested person within 2 days prior to pediculicide treatment be machine washed with hot water and dried with hot air since the lice and eggs are killed after 5 minutes of exposure to temperatures greater than 53.5°C. Items that cannot be laundered can be dry-cleaned or sealed in a plastic bag for 2 weeks. The floors and furniture should be vacuumed in order to remove hairs from an infested individual, which might have been shed with viable nits attached.

In the treatment of body lice, medications are less essential than environmental measures. Patients with body lice should have infested clothing, bedding, and towels laundered with hot water (at least 55°C) and then dried in a dryer using a hot setting. For items that cannot be washed in a washing machine, the CDC recommends dry-cleaning or sealing and storing for 2 weeks in a plastic bag. If the patient maintains hygiene with regular appropriate laundering of clothing, changes into clean clothing at least weekly, and avoids the sharing of clothing, beds, bedding, and towels used by other infested individuals, pediculicides are generally not required. If hygiene cannot be maintained, treatment with a pediculicide used to treat head lice may be necessary. Fumigation or dusting with chemical insecticides is occasionally needed to control and prevent spread of louse-bourne infections.

Lotions are likely to be more effective than shampoos, and should be applied to all body hair including the beard and moustache if necessary. A second application after 3-7 days is advised.

**Recommended regimens**
- **Permethrin 1% cream rinse.** Apply to damp hair and wash out after 10 minutes. Permethrin is the drug of choice recommended by most authorities as the first line of treatment in head, pubic, and severe body louse infestation, especially for infants older than 2 months and small children. (Permethrin is safe during pregnancy and breastfeeding)
HERPES ZOSTER

Reactivation of varicella-zoster virus (VZV) that has remained dormant within dorsal root ganglia, often for decades after the patient’s initial exposure to the virus in the form of varicella (chickenpox), results in herpes zoster. Although it is usually a self-limited dermatomal rash with pain, herpes zoster can be far more serious; in addition, acute cases often lead to postherpetic neuralgia (PHN).

Signs and symptoms
The clinical manifestations of herpes zoster can be divided into the following 3 phases:
- Preeruptive phase (preherpetic neuralgia)
- Acute eruptive phase
- Chronic phase (PHN)

The preeruptive phase is characterized by the following:
- Sensory phenomena along 1 or more skin dermatomes, lasting 1-10 days (average, 48 hours)
- Phenomena usually are noted as pain or, less commonly, itching or paresthesias
- Pain may simulate headache, iritis, pleurisy, brachial neuritis, cardiac pain, appendicitis or other intra-abdominal disease, or sciatica
- Other symptoms, such as malaise, myalgia, headache, photophobia, and, uncommonly, fever

The acute eruptive phase is marked by the following:
- Patchy erythema, occasionally accompanied by induration, in the dermatomal area of involvement
- Regional lymphadenopathy, either at this stage or subsequently
- Grouped herpetiform vesicles developing on the erythematous base (the classic finding)
- Cutaneous findings that typically appear unilaterally, stopping abruptly at the midline of the limit of sensory coverage of the involved dermatome
- Vesicular involution: Vesicles initially are clear but eventually cloud, rupture, crust, and involute
- After vesicular involution, slow resolution of the remaining erythematous plaques, typically without visible sequelae
- Scarring can occur if deeper epidermal and dermal layers have been compromised by excoriation, secondary infection, or other complications
- Almost all adults experience pain, typically severe
- A few experience severe pain without a vesicular eruption (ie, zoster sine herpete)
- Symptoms tend to resolve over 10-15 days
- Complete healing of lesions may require up to a month
**Diagnosis**

Diagnosis of herpes zoster is based primarily on the history and physical findings. In most cases, confirming the diagnosis via laboratory testing has no utility. In select patient populations, however—particularly immunocompromised patients—the presentation of herpes zoster can be atypical and may require additional testing.

**Management**

Episodes of herpes zoster are generally self-limited and resolve without intervention; they tend to be more benign and mild in children than in adults.

Conservative therapy includes the following:

- NSAIDs
- Wet dressings with 5% aluminum acetate (Burrow solution), applied for 30-60 minutes 4-6 times daily
- Lotions (eg, calamine)

Primary medications for acute zoster-associated pain include the following:

- Narcotic and nonnarcotic analgesics (both systemic and topical)
- Neuroactive agents (eg, tricyclic antidepressants [TCAs])
- Anticonvulsant agents

Oral treatment with the following has been found beneficial:

- Acyclovir

**Usual Adult Dose for Herpes Zoster**

**Acute herpes zoster:**

800 mg orally every 4 hours (5 times a day) for 7 to 10 days

Hospital admission should be considered for patients with any of the following:

- Severe symptoms
- Immunosuppression
- Atypical presentations (eg, myelitis)
- Involvement of more than 2 dermatomes
- Significant facial bacterial superinfection
- Disseminated herpes zoster
- Ophthalmic involvement
- Meningoencephalopathic involvement
HERPES SIMPLEX VIRUSES

Herpes simplex viruses are ubiquitous, host-adapted pathogens that cause a wide variety of disease states. Two types exist: herpes simplex virus type 1 (HSV-1) and type 2 (HSV-2). Both are closely related but differ in epidemiology. HSV-1 is traditionally associated with orofacial disease, while HSV-2 is traditionally associated with genital disease; however, lesion location is not necessarily indicative of viral type.

Acute herpetic gingivostomatitis
This is a manifestation of primary HSV-1 infection that occurs in children aged 6 months to 5 years. Adults may also develop acute gingivostomatitis, but it is less severe and is associated more often with a posterior pharyngitis. Infected saliva from an adult or another child is the mode of infection. The incubation period is 3-6 days. Clinical features include the following:
- Abrupt onset
- High temperature (40°C)
- Anorexia and listlessness
- Gingivitis (This is the most striking feature, with markedly swollen, erythematous, friable gums.)
- Vesicular lesions (These develop on the oral mucosa, tongue, and lips and later rupture and coalesce, leaving ulcerated plaques.)
- Tender regional lymphadenopathy
- Perioral skin involvement due to contamination with infected saliva

Course: Acute herpetic gingivostomatitis lasts 5-7 days, and the symptoms subside in 2 weeks. Viral shedding from the saliva may continue for 3 weeks or more.

Acute herpetic pharyngotonsillitis
In adults, oropharyngeal HSV-1 infection causes pharyngitis and tonsillitis more often than gingivostomatitis. Fever, malaise, headache, and sore throat are presenting features. The vesicles rupture to form ulcerative lesions with grayish exudates on the tonsils and the posterior pharynx. Associated oral and labial lesions occur in fewer than 10% of patients. HSV-2 infection can cause similar symptoms and can be associated with orogenital contact or can occur concurrently with genital herpes.

Herpes labialis
This is the most common manifestation of recurrent HSV-1 infection. A prodrome of pain, burning, and tingling often occurs at the site, followed by the development of erythematous papules that rapidly develop into tiny, thin-walled, intraepidermal vesicles that become pustular and ulcerate. In most patients, fewer than two recurrences manifest each year, but some individuals experience monthly recurrences. Maximum viral shedding is in the first 24 hours of the acute illness but may last 5 days.

Genital herpes
The severity and frequency of the disease and the recurrence rate depend on numerous factors, including viral type, prior immunity to autologous or heterologous virus, gender, and immune status of the host.

Treatment & Management

Overall, medical treatment of herpes simplex virus (HSV) infection is centered around specific antiviral treatment. While the same medications are active against HSV-1 and HSV-2, the location of the lesions...
and the chronicity (primary or reactivation) of the infection dictate the dosage and frequency of medication. It is important to note that life-threatening HSV infections in immunocompromised patients and HSV encephalitis require high-dose intravenous acyclovir, often started empirically.

**Usual Adult Dose for Herpes Simplex - Mucocutaneous/Immunocompetent Host**
Initial episode or intermittent therapy: 200 mg orally every 4 hours (5 times a day) for 10 days
Recurrent episodes: 200 mg orally every 4 hours (5 times a day) for 5 days

**Usual Pediatric Dose for Herpes Simplex**
**Neonatal HSV infection:**
Less than 3 months: 10 to 20 mg/kg or 500 mg/m² IV every 8 hours for 10 to 21 days

**Usual Pediatric Dose for Herpes Simplex - Mucocutaneous/Immunocompetent Host**
**3 months to 11 years:**
Initial episode: 10 to 20 mg/kg orally 4 times a day or 8 to 16 mg/kg orally 5 times a day for 7 to 10 days
The American Academy of Pediatrics (AAP) recommends 40 to 80 mg/kg orally per day in 3 to 4 divided doses for 5 to 10 days.
Maximum dose: 1 g per day

**12 years or older, over 40 kg:**
Initial episode, severe initial episode, and recurrent episodes: Adult dose
PEDIATRIC CHICKENPOX

Varicella (chickenpox), is caused by the varicella-zoster virus. The disease is generally regarded as a mild, self-limiting viral illness with occasional complications. Varicella is common and highly contagious and affects nearly all susceptible children before adolescence.

A significant number of varicella cases are associated with complications, among the most serious of which are varicella pneumonia and encephalitis.

The following are the most common presenting symptoms of varicella:

- Low-grade fever preceding skin manifestations by 1-2 days
- Complaints of abdominal pain by some children
- Pleomorphic rash, usually starting on the head and trunk and spreading to the rest of the body
- Typically, complaints of intense pruritus
- Headache
- Malaise
- Anorexia
- Cough and coryza
- Sore throat

Children with eczema or dermatitis may have severe skin manifestations during varicella. Immunocompromised children often have severe and complicated varicella, and their mortality rate is higher than that in immunocompetent children. Such children are at high risk for developing progressive varicella with multiple organ involvement. These children may have prolonged high fever, prolonged extensive rashes, and hepatitis.

Examination of rash

The diagnosis of varicella is made upon observation of the characteristic chickenpox rash. This rash appears in crops. Skin lesions initially appear on the face and trunk, beginning as red macules and progressing over 12-14 days to become papular, vesicular, pustular, and finally crusted. New lesions continue to erupt for 3-5 days. Lesions usually crust by 6 days (range 2-12 d), and completely heal by 16 days (range 7-34 d). Prolonged eruption of new lesions or delayed crusting and healing can occur with impaired cellular immunity.

Fever is usually low grade (37,7°C) but may be as high as 41°C. In otherwise healthy children, fever typically subsides within 4 days. Prolonged fever should prompt suspicion of complication or immunodeficiency. Although tachypnea may be seen with fever alone, respiratory distress might represent pneumonitis.

Complications

Pneumonia
Perhaps the most serious complication of varicella is viral pneumonia, which primarily occurs in older children and adults. Respiratory symptoms usually appear 3-4 days after the rash. The pneumonia may be unresponsive to antiviral therapy and may lead to death.

Secondary bacterial infections
Varicella may predispose patients to secondary bacterial infections. Signs and symptoms of such infections can be indistinguishable from uncomplicated varicella during the first 3-4 days. Skin lesion infections are common and occur in 5-10% of children. Suspect secondary infection if systemic manifestations do not improve in 3-4 days, the fever returns or worsens, or the child’s condition deteriorates after initial improvement. Suspicion of secondary bacterial infection should prompt early institution of empirical antibiotic therapy until the results of culture studies become available. The most common infectious organisms are group A streptococci and *Staphylococcus aureus*.

Neurological complications
Acute postinfectious cerebellar ataxia is the most common neurological complication, with an incidence of 1 case per 4000 patients with varicella. Ataxia has sudden onset that usually occurs 2-3 weeks after the onset of varicella. Manifestations may range from mild unsteadiness to complete inability to stand and walk, with accompanying incoordination and dysarthria.

**Hepatitis** is a self-limited accompaniment of varicella. Severe hepatitis with clinical manifestations is infrequent in otherwise healthy children with varicella. Liver involvement is independent of the severity of skin and systemic manifestations.

**Treatment & Management**
Treatment approaches include supportive measures, antiviral therapy, and management of secondary bacterial infection.

**Supportive Therapy**
Manage pruritus in patients with varicella with cool compresses and regular bathing. Discourage scratching to avoid scarring. Trimming the child’s fingernails and having the child wear mittens while sleeping may reduce scratching. Oral antihistamines, such as diphenhydramine and hydroxyzine, are used for severe pruritus.

The routine use of acyclovir in healthy children is recommended by the AAP if it can be given within 24 hours after the rash first appears in children older than 12 years, those with chronic cutaneous or pulmonary disorders, those on long-term salicylate therapy, and children receiving corticosteroids.

Children who develop severe and life-threatening varicella complications may require hospitalization. The following findings are indications for admission to the hospital:

- Altered consciousness
- Seizures
- Difficulty walking
- Respiratory distress
- Cyanosis
- Low oxygen saturation

**Pediatric Hydroxyzine**
**Pruritus**
Management of pruritus due to chronic urticaria, contact dermatoses, and histamine-mediated pruritus

- <6 years old: 50 mg/day PO divided q6hr
- >6 years old: 50-100 mg/day PO divided q6hr
HAND-FOOT-AND-MOUTH DISEASE

Hand-foot-and-mouth disease (HFMD) is a viral illness with oral and distal-extremity lesions. It is most commonly caused by coxsackievirus A16 and typically affects children and infants. HFMD is highly contagious during the first week of infection and may lead to epidemics from direct contact with nasal and oral secretions or fecal material. The incubation period typically averages 3-7 days. Symptoms include fever, rash, headache, sore throat, oropharyngeal ulcers, and loss of appetite. The oral lesions are typically 2-3 mm vesicles on an erythematous base. Care is typically supportive with antipyretics and anesthetics for symptomatic relief on a case-by-case basis.

Physical findings include the following:

- Initially, macular lesions appear on the buccal mucosa, tongue, and/or hard palate
- These mucosal lesions rapidly progress to vesicles that erode and become surrounded by an erythematous halo
- Lesions may also be found on the hands, feet, buttocks, and genitalia
- A fever of 38-39°C may be present for 24-48 hours

Management
There is no antiviral agent specific for the etiologic agents of HFMD. Instead, the treatment is supportive, as follows:

- Ensure adequate fluid intake to prevent dehydration; cold liquids are generally preferable
- Spicy or acidic substances may cause discomfort
- Intravenous hydration may be necessary if the patient has moderate-to-severe dehydration or if discomfort precludes oral intake
- Fever may be treated with antipyretics
- Pain may be treated with standard doses of paracetamol or ibuprofen
- Direct analgesia may also be applied to the oral cavity via mouthwashes or sprays
ERYSIPELAS

Erysipelas is a type of cellulitis (bacterial skin infection) that extends into the superficial cutaneous lymphatics. The most common bacteria responsible are group A streptococci. Infection occurs via inoculation into an area of local skin trauma; the legs are most commonly affected, but the face may also be infected. Patients may complain of headache, arthralgia/myalgia, and/or nausea. In severe infections, vesicles, bullae, petechiae, and frank necrosis may be found. The diagnosis is typically clinical. Treatment for 10-20 days is recommended with penicillin (or a first-generation cephalosporin or macrolide in penicillin-allergic patients). Elevation and rest of the affected area may help reduce swelling. Saline wet dressings should be applied to ulcerated and necrotic lesions. Debridement may be required in severe cases with necrosis or gangrene.

Non-steroidal anti-inflammatory drugs are contra-indicated (risk of necrotizing fasciitis).

Hospitalize for the following: children younger than 3 months, critically ill appearing patient, local complications, debilitated patient (chronic conditions, the elderly) or if there is a risk of non-compliance with or failure of outpatient treatment.
Impetigo is a common superficial bacterial infection of skin caused most often by S. aureus and in some cases by group A -hemolytic streptococci. Lesions caused by staphylococci may be tense, clear bullae, and this less common form of the disease is called bullous impetigo.

Bullous impetigo with circumscribed lesions with a thin collarette of scale.

Impetigo is the most common bacterial infection in children.

**Signs and symptoms**
Children with nonbullous impetigo commonly have multiple coalescing lesions on their face (perioral, perinasal) and extremities or in areas with a break in the natural skin defense barrier. The initial lesions are small vesicles or pustules (< 2 cm) that rupture and become a honey-colored crust with a moist erythematous base. Pharyngitis is absent, but mild regional lymphadenopathy is commonly present. Nonbullous impetigo is usually a self-limited process that resolves within 2 weeks. Impetigo usually occurs on exposed areas of the body, most frequently the face and extremities. The lesions remain well-localized but are frequently multiple and may be either bullous or nonbullous in appearance. Bullous impetigo also differs from nonbullous impetigo in that bullous impetigo may involve the buccal mucous membranes, and regional adenopathy rarely occurs. However, extensive lesions in infants may be associated with systemic symptoms such as fever, malaise, generalized weakness, and diarrhea.

**Diagnosis**
The diagnosis of impetigo is usually made on the basis of the history and physical examination.

**Management**
Treatment of impetigo typically involves local wound care in conjunction with either a topical antibiotic or a combination of systemic and topical agents. In general, the antibiotic selection has coverage against both S. aureus and S. pyogenes. In areas with a high prevalence of community-acquired MRSA with susceptible isolates, children older than 8 years may take clindamycin or doxycycline in cases.

**Topical Antibiotic Treatment**

**Mupirocin** ointment (Bactroban) has been used for both the lesions and to clear chronic nasal carriers. It is applied to the affected area 2-3 times daily. A 7-day course is usually standard.

**Clindamycin** (cream, lotion, and foam) is useful in several MRSA infections.

**Systemic Antibiotic Treatment**
Infections that are widespread, complicated, or are associated with systemic manifestations are usually treated with antibiotics that have gram-positive bacterial coverage.

Beta-lactamase resistant antibiotics (eg, cephalosporins, amoxicillin-clavulanate, cloxacillin,) are recommended. Cephalexin appears to be the drug of choice for oral antimicrobial therapy in children.

Treat traumatized skin with mupirocin because this has been shown to decrease the rates of impetigo spread. Treat preexisting underlying skin diseases, such as atopic dermatitis. Antihistamines and topical steroids help decrease scratching. Teach good personal hygiene. For example, keep nails short and clean and wash hands frequently with antibacterial soap and water or waterless antibacterial cleansers.

**ORAL HAIRY LEUKOPLAKIA**

*Oral hairy leukoplakia* often presents as white plaques on the lateral tongue and is associated with Epstein-Barr virus infection.

Patients with oral hairy leukoplakia may report a nonpainful white plaque along the lateral tongue borders. The appearance may change daily. The natural history of hairy leukoplakia is variable. Lesions may frequently appear and disappear spontaneously. Unilateral or bilateral nonpainful white lesions can be seen on the margins, dorsal or ventral surfaces of the tongue, or on buccal mucosa.

**Treatment & Management**

As a benign lesion with low morbidity, oral hairy leukoplakia does not require specific treatment in every case. Indications for treatment include symptoms attributable to the lesion, or a patient’s desire to eliminate the lesion for cosmetic reasons. Systemic antiviral therapy usually achieves resolution of the lesion within 1-2 weeks of therapy. Oral therapy with acyclovir requires high doses (800 mg 5 times per day) to achieve therapeutic levels.
**PRIMARY SYPHILIS**

Syphilis is an infectious venereal disease caused by the spirochete *Treponema pallidum*. Syphilis is transmissible by sexual contact with infectious lesions, from mother to fetus in utero, via blood product transfusion, and occasionally through breaks in the skin that come into contact with infectious lesions. If untreated, it progresses through 4 stages: primary, secondary, latent, and tertiary. Because the manifestations of syphilis (particularly advanced syphilis) are nonspecific and may masquerade as many other diseases, the physician must keep a high index of suspicion regarding the possible diagnosis of syphilis.

**Primary syphilis**

Primary syphilis occurs within 3 weeks of contact with an infected individual. It manifests mainly on the glans penis in males and on the vulva or cervix in females. Ten percent of syphilitic lesions are found on the anus, fingers, oropharynx, tongue, nipples, fingers, or other extragenital sites. Regional nontender lymphadenopathy follows invasion. Lesions (chancres) are usually solitary, raised, firm, red papules that can be several centimeters in diameter. The chancre erodes to create an ulcerative crater within the papule, with slightly elevated edges around the central ulcer. It usually heals within 4-8 weeks, with or without therapy.

**Secondary syphilis**

Secondary syphilis manifests in various ways. It usually presents with a cutaneous eruption within 2-10 weeks after the primary chancre and is most florid 3-4 months after infection. Commonly affects the palms and soles with scaling, firm, red-brown papules. Mild constitutional symptoms of malaise, headache, anorexia, nausea, aching pains in the bones, and fatigue often are present, as well as fever and neck stiffness.
**Tertiary syphilis**
Tertiary (late) syphilis is slowly progressive and may affect any organ. The disease is generally not thought to be infectious at this stage. Manifestations may include the following:
- Altered mental status
- Focal neurologic findings, including sensorineural hearing and vision loss
- Dementia
- Symptoms related to the cardiovascular system or the CNS

The lesions of benign tertiary syphilis usually develop within 3-10 years of infection. The typical lesion is a gumma, and patient complaints usually are secondary to bone pain, which is described as a deep boring pain characteristically worse at night.

**Congenital syphilis**
Early congenital syphilis occurs within the first 2 years of life. Late congenital syphilis emerges in children older than 2 years.

**Treatment & Management**

Penicillin is the treatment of choice for treating syphilis. Patients with known penicillin allergies should undergo penicillin allergy skin testing and penicillin desensitization, if necessary.

The following regimens are recommended for penicillin treatment:
- Primary or secondary syphilis - Benzathine penicillin G 2.4 million units IM in a single dose
- Early latent syphilis - Benzathine penicillin G 2.4 million units IM in a single dose
- Late latent syphilis or latent syphilis of unknown duration - Benzathine penicillin G 7.2 million units total, administered as 3 doses of 2.4 million units IM each at 1-week intervals
- Pregnancy - Treatment appropriate to the stage of syphilis is recommended.

Tetracycline, erythromycin, and ceftriaxone have shown antitreponemal activity in clinical trials; however, they currently are recommended only as alternative treatment regimens in patients allergic to penicillin.
ERYTHEMA MULTIFORME

Erythema multiforme (EM) is an acute, self-limited, and sometimes recurring skin condition that is considered to be a type IV hypersensitivity reaction associated with certain infections, medications, and other various triggers.

Erythema multiforme major and Stevens-Johnson syndrome (SJS), however, are more severe, potentially life-threatening disorders. Lesions of Steven-Johnson syndrome typically begin on the face and trunk.

The clinical descriptions are as follows:

- **Erythema multiforme minor** - Typical targets or raised, edematous papules distributed acrally
- **Erythema multiforme major** - Typical targets or raised, edematous papules distributed acrally with involvement of one or more mucous membranes; epidermal detachment involves less than 10% of TBSA.
- **SJS/TEN** - Widespread blisters predominant on the trunk and face, presenting with erythematous or pruritic macules and one or more mucous membrane erosions; epidermal detachment is less than 10% TBSA for Steven-Johnson syndrome / toxic epidermal necrolysis and 30% or more for toxic epidermal necrolysis.

Most cases of erythema multiforme (EM) are self-limited. In erythema multiforme minor, the lesions evolve over 1-2 weeks and ultimately subside within 2-3 weeks without scarring. However, the recurrence of erythema multiforme minor is common (up to one third of cases) and mostly preceded by apparent or subclinical HSV infection.

**Symptoms**

Prodromal symptoms are usually absent or mild in persons with erythema multiforme minor, consisting of a mild, nonspecific upper respiratory tract infection. The abrupt onset of a rash usually occurs within 3 days, starting on the extremities symmetrically, with centripetal spreading. Pruritus is generally absent. The initial lesion is a dull-red, purpuric macule or urticarial plaque that expands slightly to a maximum of 2 cm over 24-48 hours. In the center, a small papule, vesicle, or bulla develops, flattens, and then may clear. An intermediate ring develops and becomes raised, pale, and edematous. The periphery gradually changes to become cyanotic or violaceous and forms a typical concentric, “target” lesion.

Mild temperature elevation is usually noted. Hyperventilation and mild hypoxia may result from anxiety or tracheobronchial involvement.

**Treatment & Management**
For all forms of erythema multiforme (EM), the most important treatment is usually symptomatic, including oral antihistamines, analgesics, local skin care, and soothing mouthwashes (eg, oral rinsing with warm saline or a solution of diphenhydramine, xylocaine, and). Topical steroids may be considered. In severe cases, prehospital personnel may need to treat respiratory complications and fluid imbalances aggressively, in the same manner as thermal burns. Avoid systemic corticosteroids in minor cases. In severe cases, their use is controversial, because these agents do not improve prognosis and may increase risk of complications.

**Hospitalization**

Erythema multiforme (EM) major may require hospitalization for the treatment of complications and sequelae (eg, severe mucous membrane involvement is present or with impaired oral intake, dehydration, or secondary infection) and to manage the patient’s fluid and electrolytes. The most severe cases should be managed in intensive care or burn units.
EYE DISEASES
ACUTE CONJUNCTIVITIS

This term describes any inflammatory process that involves the conjunctiva; however, to most patients, conjunctivitis (often called pink eye) is a diagnosis in its own right. As with any mucous membrane, infectious agents may adhere to the conjunctiva, thus overwhelming normal defense mechanisms and producing clinical symptoms of redness, discharge, irritation, and possibly photophobia.

Clinical Evaluation
In classic presentations, patients complain of eyelids sticking together on waking. They may describe itching and burning or a gritty, foreign-body sensation. Pus sliding across the eye may distort vision, although visual acuity is normal. Photophobia is minimal. Bilateral disease is typically infectious or allergic. Unilateral disease suggests toxic, chemical, mechanical, or lacrimal origin.

Bacterial Conjunctivitis
Bacterial conjunctivitis is characterized by acute onset, minimal pain, occasional pruritus, and, sometimes, exposure history. Ocular surface disease (e.g., keratitis sicca, trichiasis, chronic blepharitis) predisposes the patient to bacterial conjunctivitis. Staphylococcal and streptococcal species are the most common pathogens.

Allergic Conjunctivitis
Allergic conjunctivitis is characterized by acute or subacute onset, no pain, and no exposure history. Pruritus is extremely common and the hallmark symptom of this condition. Clear, watery discharge is typical, with or without a moderate amount of mucus production.

Prehospital and ED Management
Prehospital transport is rarely indicated for patients with conjunctivitis. More serious concerns may warrant EMS transport. Prehospital personnel, emergency physicians, and other medical personnel must be careful not to transmit this infection and should not overlook more serious comorbidity. Thorough hand washing, glove use, and using eye drops in individual or unit dose containers are necessary. Treatment is often supportive. Artificial tears help the discomfort of keratitis and photophobia. Cold, moist compresses improve the swelling and discomfort of the lids. Antibiotic drops help prevent a secondary bacterial infection. Reserve topical corticosteroids for use by an ophthalmologist when substantial inflammation is present and herpes simplex is excluded.

Clean eyes 4 to 6 times/day with boiled water or 0.9% sodium chloride

Broad-spectrum antibiotics, such as nafloxin (ciprofloxacin) or oxolin (ofloxacin), are good choices (2 times/day into both eyes for 7 days).

Consult with an ophthalmologist for all serious eye complaints. Simple conjunctivitis usually can be followed up by the patient's primary care provider.
XEROPHTHALMIA

Is a medical condition in which the eye fails to produce tears. It may be caused by vitamin A deficiency, which is sometimes used to describe that condition, although there may be other causes.

Xerophthalmia caused by a severe vitamin A deficiency is described by pathologic dryness of the conjunctiva and cornea. The conjunctiva becomes dry, thick and wrinkled. If untreated, it can lead to corneal ulceration and ultimately to blindness as a result of corneal damage.

Xerophthalmia usually affects children under nine years old.

Prophylaxis consists of periodic administration of Vitamin A supplements. WHO recommended schedule, which is universally recommended is as follows:

- Infants 6–12 months old and any older children weighing less than 8 kg - 100,000 IU orally every 3–6 months
- Children over 1 year and under 6 years of age - 200,000 IU orally every 6 months
- Infants less than 6 months old, who are not being breastfed - 50,000 IU orally should be given before they attain the age of 6 months

Treatment can occur in two ways: treating symptoms and treating the deficiency. Treatment of symptoms usually includes use of artificial tears in the form of eye drops, increasing the humidity of the environment with humidifiers, and wearing wrap around glasses when outdoors. Treatment of the deficiency can be accomplished with a Vitamin A or multivitamin supplement or by eating foods rich in Vitamin A.
VIRAL DISEASES
INFLUENZA

Influenza, one of the most common infectious diseases, is a highly contagious airborne disease that occurs in seasonal epidemics and manifests as an acute febrile illness with variable degrees of systemic symptoms, ranging from mild fatigue to respiratory failure and death.

Signs and symptoms
The presentation of influenza virus infection varies, but it usually includes many of the following signs and symptoms:
- Fever
- Sore throat
- Myalgias
- Frontal or retro-orbital headache
- Nasal discharge
- Weakness and severe fatigue
- Cough and other respiratory symptoms
- Tachycardia
- Red, watery eyes

Fever may vary widely among patients, with some having low fevers (in the 37.7°C range) and others developing fevers as high as 40°C. Some patients report feeling feverish and feeling chills. In children, diarrhea may be a feature.

The incubation period of influenza is 2 days long on average but may range from 1 to 4 days in length. Aerosol transmission may occur 1 day before the onset of symptoms; thus, it may be possible for transmission to occur via asymptomatic persons or persons with subclinical disease, who may be unaware that they have been exposed to the disease.

Diagnosis
Influenza has traditionally been diagnosed on the basis of clinical criteria.
The criterion standard for diagnosing influenza A and B is a viral culture of nasopharyngeal samples or throat samples. In elderly or high-risk patients with pulmonary symptoms, chest radiography should be performed to exclude pneumonia.

Management
 Prevention
Prevention of influenza is the most effective management strategy. Influenza A and B vaccine is administered each year before flu season.

Treatment
Patients with influenza generally benefit from bed rest. Most patients with influenza recover in 3 days; however, malaise may persist for weeks.
Patients most often require hospitalization when influenza exacerbates underlying chronic diseases.

1. Rest until the flu is fully resolved, especially if the illness has been severe.
2. Fluids – Drink enough fluids so that you do not become dehydrated.
3. Paracetamol can relieve fever, headache, and muscle aches.
4. Cough medicines are not usually helpful; cough usually resolves without treatment.

Following is a list of all the health and age factors that are known to increase a person’s risk of getting serious complications from the flu:
People younger than 19 years of age on long-term aspirin therapy
People with COPD
Weakened immune system due to disease or medication (such as people with HIV or AIDS, or cancer, or those on chronic steroids)

Other people at high risk from the flu:
- Adults 65 years and older
- Children younger than 5 years old, but especially children younger than 2 years old
g- Pregnant women and women up to 2 weeks after the end of pregnancy
- Asthma
- Blood disorders (such as sickle cell disease)
- Chronic lung disease (such as COPD and cystic fibrosis)
- Endocrine disorders (such as diabetes mellitus)
- Heart disease (such as congenital heart disease, congestive heart failure and coronary artery disease)
- Kidney disorders
- Liver disorders
- Metabolic disorders (such as inherited metabolic disorders and mitochondrial disorders)
- Morbid obesity
- Neurological and neurodevelopmental conditions

Supportive care for pediatric influenza can include the following:
- Paracetamol for fever
- Cough suppressants and expectorants
- Steam inhalation
- Oral or intravenous fluids if dehydration occurs
- Antiviral therapy for selected patients

Who should take antiviral drugs?
- People who are very sick with the flu (for example, people who are in the hospital).
- People who are sick with the flu and have a high-risk health condition like asthma, diabetes or chronic heart disease.

Oseltamivir (Tamiflu)
It must be administered within 48 hours of symptom onset. The sooner it is taken after symptom onset, the better the effect.

Adults: 75 mg PO q12hr x5 days
Pediatric:
<2 weeks: Safety and efficacy not established for treatment
Aged 2 weeks to <1 year
- 3 mg/kg PO BID x5 days
≥1 year
- <15 kg: 30 mg PO q12hr x5 days
- 15-23 kg: 45 mg PO q12hr x5 days
- 23-40 kg: 60 mg PO q12hr x5 days
- >40 kg: 75 mg PO q12hr x5 days
**POLIO (POLIOMYELITIS)**

Acute poliomyelitis is caused by small ribonucleic acid (RNA) viruses. Acute poliomyelitis is a disease of the anterior horn motor neurons of the spinal cord and brain stem caused by poliovirus. Flaccid asymmetric weakness and muscle atrophy are the hallmarks of its clinical manifestations, due to loss of motor neurons and denervation of their associated skeletal muscles. As of 2006, 6 countries were endemic to polio: Afghanistan, Egypt, India, Niger, Nigeria, and Pakistan. Most cases of acute poliomyelitis occur in the pediatric population. Infection or immunization against poliovirus provides lifelong protection.

**Clinical Presentation**
Most patients (95%) with poliomyelitis virus infections are asymptomatic or have only mild systemic symptoms, such as pharyngitis or gastroenteritis. These cases are referred to as minor illness or abortive poliomyelitis. The mild symptoms are related to viremia and immune response against dissemination of the virus. Only 5% of patients exhibit different severities of nervous system involvement, from nonparalytic poliomyelitis to the most severe form of paralytic poliomyelitis.

**Nonparalytic poliomyelitis or preparalytic poliomyelitis**
- The prodromal symptoms include generalized, nonthrobbing headache; fever of 38-40 °C; sore throat; anorexia; nausea; vomiting; and muscle aches. These symptoms may or may not subside in 1-2 weeks.
- Headache and fever, as well as signs and symptoms of nervous system involvement (eg, irritability, restlessness, apprehensiveness, emotional instability, stiffness of the neck and back) and Kernig and Brudzinski signs because of meningitis, then may follow.
- Children generally exhibit milder systemic symptoms than do adults.
- Preparalytic symptoms also may develop into paralytic ones.

**Paralytic poliomyelitis**
- The incubation period from virus exposure to the neurologic phase can last 4-10 days but may extend to 4-5 weeks.
- Severe muscle pain and spasms, followed by weakness, develop. Muscle weakness tends to become maximal within 48 hours but may develop for longer than a week. No progression of weakness should be noted after the temperature drops to normal for 48 hours. Weakness is asymmetric, with the lower limbs affected more than upper limbs.
- Muscle tone is flaccid, and the reflexes initially are brisk but then become absent. The transient or occasionally persistent coarse fasciculations also are observed frequently in patients with paralytic poliomyelitis.
- Patients also complain of paresthesias in the affected limbs without real sensation loss.
- Paralysis remains for days or weeks before slow recovery occurs over months or years. Which factors favor development of paralytic disease remains unclear, but some evidence exists that physical activity and intramuscular injections during the prodrome may be important exacerbating factors.
The encephalitic form of poliomyelitis

- This form is very rare and manifests as agitation, confusion, stupor, and coma.
- Autonomic dysfunction is common, and it has a high mortality.

Vital signs are the key to monitoring patients with poliovirus infection.

- Muscle weakness can be assessed by muscle strength testing.
  - Usually asymmetric proximal weakness is present with more involvement of lumbar than cervical segments and more spinal cord than brainstem segments.
  - The trunk muscles are affected least.
  - Sensation should be within normal limits objectively.
  - Deep tendon reflexes are diminished or absent.
  - Atrophy of muscle may be detected 3 weeks after onset of paralysis, which becomes maximal at 12-15 weeks and remains permanent.

Medical Issues/Complications

**Immediate communication to stakeholders and health professionals at national and local level**

No specific treatment exists for acute poliomyelitis except supportive care, which may help to ensure survival, modify the disability, and improve the outcome.

All patients should be placed on bedrest in an isolation unit. Monitor patients' vital signs carefully; focus especially on the swallowing function, vital capacity, pulse, and blood pressure, in anticipation of respiratory or circulatory complications.
CHOLERA

Cholera is an acute diarrheal illness caused by an intestinal infection with the bacteria *Vibrio cholerae*; it affects only humans. The bacteria release a toxin that causes increased release of water from intestinal cells, resulting in severe diarrhea. The main reservoirs for *V cholerae* are people and aquatic/marine sources (eg, brackish water, estuaries) that are often associated with algal blooms; people become infected by eating or drinking fecal- or vomitus-contaminated food or water, particularly during cholera epidemics. Cholera can quickly spread through areas with inadequate water treatment, poor sanitation, and populations with inadequate personal hygiene, problems commonly seen in situations of war, famine, and crowding. Cholera is common in developing regions (eg, Africa, Asia, India, Mexico, South and Central America). Early detection and preventative measures such as the practice of good hygiene and the establishment of clean food and water supplies and sanitation are essential to minimizing the spread of cholera during epidemics.

Clinical Presentation

After a 24- to 48-hour incubation period, symptoms begin with the sudden onset of painless watery diarrhea that may quickly become voluminous and is often followed by vomiting. The patient may experience accompanying abdominal cramps, probably from distention of loops of small bowel as a result of the large volume of intestinal secretions. Fever is typically absent.

Cholera should be suspected when a patient older than 5 years develops severe dehydration from acute, severe, watery diarrhea (usually without vomiting) or in any patient older than 2 years who has acute watery diarrhea and is in an area where an outbreak of cholera has occurred. Stool volume during cholera is more than that of any other infectious diarrhea. Patients with severe disease may have a stool volume of more than 250 mL/kg body weight in a 24-hour period. Because of the large volume of diarrhea, patients with cholera have frequent and often uncontrolled bowel movements. The stool may contain fecal material early in the course of clinical illness. The characteristic cholera stool is an opaque white liquid that is not malodorous and often is described as having a “rice water” appearance (ie, in color and consistency, it resembles water that has been used to wash or cook rice).

Vomiting, although a prominent manifestation, may not always be present. Early in the course of the disease, vomiting is caused by decreased gastric and intestinal motility; later in the course of the disease it is more likely to result from acidemia.

Dehydration

If untreated, the diarrhea and vomiting lead to isotonic dehydration, which can lead to acute tubular necrosis and renal failure. In patients with severe disease, vascular collapse, shock, and death may ensue. Dehydration can develop with remarkable rapidity, within hours after the onset of symptoms.

Clinical signs of cholera parallel the level of volume contraction. The amount of fluid loss and the corresponding clinical signs of cholera are as follows:

- 3-5% loss of normal body weight - Excessive thirst
- 5-8% loss of normal body weight - Postural hypotension, tachycardia, weakness, fatigue, dry mucous membranes or dry mouth
- >10% loss of normal body weight - Oliguria; glassy or sunken eyes; sunken fontanelles in infants; weak, thready, or absent pulse; wrinkled "washerwoman" skin; somnolence; coma
Treatment & Management

Immediate communication to stakeholders and health professionals at national and local level

Rehydration is the first priority in the treatment of cholera. Rehydration is accomplished in 2 phases: rehydration and maintenance.

The goal of the rehydration phase is to restore normal hydration status, which should take no more than 4 hours. Set the rate of intravenous infusion in severely dehydrated patients at 50-100 mL/kg/hr. Lactated Ringer solution is preferred over isotonic sodium chloride solution because saline does not correct metabolic acidosis.

The goal of the maintenance phase is to maintain normal hydration status by replacing ongoing losses. The oral route is preferred, and the use of oral rehydration solution (ORS) at a rate of 500-1000 mL/hr is recommended.

Fluid Replacement for Dehydration

<table>
<thead>
<tr>
<th>Severe dehydration</th>
<th>Intravenous (IV) drips of Ringer Lactate or, if not available, normal saline and oral rehydration salts as outlined below</th>
<th>• 100 mL/kg in 3-h period (in 6 h for children &lt; 1 y) • Start rapidly (30 mL/kg within 30 min, then slow down) • Total amount for first 24 h: 200 L/kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Some dehydration</td>
<td>Oral rehydration salts (amount in first 4 h)</td>
<td>• Infants &lt; 4 mo (&lt; 5 kg): 200–400 mL • Infants 4–11 mo (5–7.9 kg): 400–600 mL • Children 1–2 y (8–10.9 kg): 600–800 mL • Children 2–4 y (11–15.9 kg): 800–1200 mL • Children 5–14 y (16–29.9 kg): 1200–2200 mL • Patients &gt;14 y (≥30 kg): 2200–4000 mL</td>
</tr>
<tr>
<td>No dehydration</td>
<td>Oral rehydration salts</td>
<td>• Children &lt; 2 y: 50–100 mL, up to 500 mL/day • Children 2–9 y: 100–200 mL, up to 1000 mL/day • Patients &gt;9 y: As much as wanted, up to 2000 mL/day</td>
</tr>
</tbody>
</table>

Approximate Amount of Oral Rehydration Solution to Administer in the First 4 Hours

<table>
<thead>
<tr>
<th>Age</th>
<th>&lt; 4 mo</th>
<th>4-11 mo</th>
<th>12-23 mo</th>
<th>2-4 y</th>
<th>5-14 y</th>
<th>≥15 y</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight</td>
<td>&lt; 5 kg</td>
<td>5-7.9 kg</td>
<td>8-10.9 kg</td>
<td>11-15.9 kg</td>
<td>16-29.9 kg</td>
<td>≥30 kg</td>
</tr>
<tr>
<td>ORS solution in mL</td>
<td>200-400</td>
<td>400-600</td>
<td>600-800</td>
<td>800-1200</td>
<td>1200-2200</td>
<td>2200-4000</td>
</tr>
</tbody>
</table>

Antibiotic Treatment

An effective antibiotic can reduce the volume of diarrhea in patients with severe cholera and shorten the period during which V cholerae O1 is excreted. In addition, it usually stops the diarrhea within 48 hours, thus shortening the period of hospitalization. Whenever possible, antibiotic therapy should be guided by susceptibility reports.

Antibiotic treatment is indicated for severely dehydrated patients who are older than 2 years. Begin antibiotic therapy after the patient has been rehydrated (usually in 4-6 h) and vomiting has stopped. No advantage exists to using injectable antibiotics, which are expensive. No other drugs should be used in...
the treatment of cholera. Antimicrobial agents typically are administered for 3-5 days. However, single-dose therapy with tetracycline, doxycycline, furazolidone, or ciprofloxacin has been shown effective in reducing the duration and volume of diarrhea. Because single dose doxycycline has been shown to be as effective as multiple doses of tetracycline, this has become the preferred regimen.

Antimicrobial Therapy Used in the Treatment of Cholera*

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>Single Dose (PO)</th>
<th>Multiple Dose (PO)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Doxycycline†</td>
<td>7 mg/kg; not to exceed 300 mg/dose‡</td>
<td>2 mg/kg bid on day 1; then 2 mg/kg qd on days 2 and 3; not to exceed 100 mg/dose</td>
</tr>
<tr>
<td>Tetracycline†</td>
<td>25 mg/kg; not to exceed 1 g/dose‡</td>
<td>40 mg/kg divided qid for 3 d; not to exceed 2 g/d</td>
</tr>
<tr>
<td>Furazolidone</td>
<td>7 mg/kg; not to exceed 300 mg/dose‡</td>
<td>5 mg/kg/d divided qid for 3 d; not to exceed 400 mg/d</td>
</tr>
<tr>
<td>Trimethoprim and sulfamethoxazole</td>
<td>Not evaluated</td>
<td>&lt; 2 months: Contraindicated</td>
</tr>
<tr>
<td></td>
<td></td>
<td>≥2 months: 5-10 mg/kg/d (based on trimethoprim component) divided bid for 3 d;</td>
</tr>
<tr>
<td></td>
<td></td>
<td>not to exceed 320 mg/d trimethoprim and 1.6 g/d of sulfamethoxazole</td>
</tr>
<tr>
<td>Ciprofloxacin§</td>
<td>30 mg/kg; not to exceed 1 g/dose‡</td>
<td>30 mg/kg/d divided q12h for 3 d; not to exceed 2 g/d</td>
</tr>
<tr>
<td>Ampicillin</td>
<td>Not evaluated</td>
<td>50 mg/kg/d divided qid for 3 d; not to exceed 2 g/d</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>Not evaluated</td>
<td>40 mg/kg/d erythromycin base divided tid for 3 d; not to exceed 1 g/d</td>
</tr>
</tbody>
</table>

* Antimicrobial therapy is an adjunct to fluid therapy of cholera and is not an essential component. However, it reduces diarrhea volume and duration by approximately 50%. The choice of antibiotics is determined by the susceptibility patterns of the local strains of *V cholerae* O1 or O139.

† Tetracycline and doxycycline can discolor permanent teeth of children younger than 8 years. However, the risk is small when these drugs are used for short courses of therapy, especially if used in a single dose.

‡ Single-dose therapy of these drugs has not been evaluated systematically in children, and recommendations are extrapolated from experience in adults.

§ Fluoroquinolones (eg, ciprofloxacin) are not approved in the United States for use in persons younger than 18 years. When given in high doses to juvenile animals, they cause arthropathy. Clinical experience indicates that this risk is very small in children when used for short courses of therapy.
WHO Guidelines for Cholera Management

Steps in the treatment of a patient with suspected cholera are as follows:

1. Assess for dehydration (see Table 1)
2. Rehydrate the patient and monitor frequently, then reassess hydration status
3. Maintain hydration; replace ongoing fluid losses until diarrhea stops
4. Administer an oral antibiotic to the patient with severe dehydration
5. Feed the patient

More detailed guidelines for the treatment of cholera are as follows:

- Evaluate the degree of dehydration upon arrival
- Rehydrate the patient in 2 phases; these include rehydration (for 2-4 h) and maintenance (until diarrhea abates)
- Register output and intake volumes on predesigned charts and periodically review these data
- Use the intravenous route only (1) during the rehydration phase for severely dehydrated patients for whom an infusion rate of 50-100 mL/kg/h is advised, (2) for moderately dehydrated patients who do not tolerate the oral route, and (3) during the maintenance phase in patients considered high stool purgers (ie, >10 mL/kg/h)
- During the maintenance phase, use oral rehydration solution at a rate of 800-1000 mL/h; match ongoing losses with ORS administration
- Discharge patients to the treatment center if oral tolerance is greater than or equal to 1000 mL/h, urine volume is greater than or equal to 40 mL/h, and stool volume is less than or equal to 400 mL/h.
MALARIA

Malaria is a potentially life-threatening disease caused by infection with *Plasmodium* protozoa transmitted by an infective female *Anopheles* mosquito. *Plasmodium falciparum* infection carries a poor prognosis with a high mortality if untreated, but it has an excellent prognosis if diagnosed early and treated appropriately.

**Signs and symptoms**

Patients with malaria typically become symptomatic a few weeks after infection, though the symptomatology and incubation period may vary, depending on host factors and the causative species. Clinical symptoms include the following:

- Headache (noted in virtually all patients with malaria)
- Cough
- Fatigue
- Malaise
- Shaking chills
- Arthralgia
- Myalgia
- Paroxysm of fever, shaking chills, and sweats (every 48 or 72 hours, depending on species)

Less common symptoms include the following:

- Anorexia and lethargy
- Nausea and vomiting
- Diarrhea
- Jaundice

Most patients with malaria have no specific physical findings, but splenomegaly may be present. Severe malaria manifests as the following:

- Cerebral malaria (sometimes with coma)
- Severe anemia
- Respiratory abnormalities: Include metabolic acidosis, associated respiratory distress, and pulmonary edema; signs of malarial hyperpneic syndrome include alar flaring, chest retraction, use of accessory muscles for respiration, and abnormally deep breathing
- Renal failure (typically reversible)

**Diagnosis**

The patient history should include inquiries into the following:

- Recent or remote travel to an endemic area
- Immune status, age, and pregnancy status
- Allergies or other medical conditions
- Medications currently being taken
- Blood studies
- If the patient is to be treated with primaquine, glucose-6-phosphate dehydrogenase (G6PD) level

Most patients with malaria have no specific physical findings, but splenomegaly may be present. Symptoms of malarial infection are nonspecific and may manifest as a flulike illness with fever, headache, malaise, fatigue, and muscle aches. Some patients with malaria present with diarrhea and...
other gastrointestinal (GI) symptoms. Immune individuals may be completely asymptomatic or may present with mild anemia. Nonimmune patients may quickly become very ill. In returning travelers from endemic areas, malaria is suggested by the triad of thrombocytopenia, elevated lactate dehydrogenase (LDH) levels, and atypical lymphocytes. These findings should prompt obtaining malarial smears.

**Rapid diagnostic tests (RDT)**
Rapid tests detect parasite antigens. They give only a qualitative result (positive or negative) and may remain positive several days or weeks following effective treatment.

*Note: even with positive diagnostic results, rule out other causes of fever.*

Patients with suspected malaria should have parasitological confirmation of diagnosis with either microscopy or rapid diagnostic test (RDT) before antimalarial treatment is started. Treatment based on clinical grounds should only be given if diagnostic testing is not immediately accessible within two hours of patients presenting for treatment.

**Definition of uncomplicated malaria**
A patient who presents with symptoms of malaria and a positive parasitological test (microscopy or RDT) but with no features of severe malaria is defined as having uncomplicated malaria.

**Management**

**Immediate communication to stakeholders and health professionals at national and local level**

Treatment is influenced by the species causing the infection, including the following:

- *Plasmodium falciparum*
- *P. vivax*
- *P. ovale*
- *P. malariae*
- *P. knowlesi*

Patients with *P. falciparum* infection are often treated on an inpatient basis to allow observation for complications. Patients with non–*P. falciparum* malaria who are well can usually be treated on an outpatient basis.

**General recommendations for pharmacologic treatment of malaria** are as follows:

- *P. falciparum* malaria: Quinine-based therapy is with quinine (or quinidine) sulfate plus doxycycline or clindamycin or pyrimethamine-sulfadoxine; alternative therapies are artemether-lumefantrine, atovaquone-proguanil, or mefloquine
- *P. falciparum* malaria with known chloroquine susceptibility (only a few areas in Central America and the Middle East): Chloroquine
- *P. vivax, P. ovale* malaria: Chloroquine plus primaquine
- *P. malariae* malaria: Chloroquine
- *P. knowlesi* malaria: Same recommendations as for *P. falciparum* malaria

**Pregnant women** (especially primigravida) are up to 10 times more likely to contract malaria than nongravid women and have a greater tendency to develop severe malaria. Medications that can be used for the treatment of malaria in pregnancy include the following:

- Chloroquine
- Quinine
- Atovaquone-proguanil
- Clindamycin
- Mefloquine (avoid in first trimester)

**Pediatrics**

In children, malaria has a shorter course, often rapidly progressing to severe malaria. Children are more likely to present with hypoglycemia, seizures, severe anemia, and sudden death, but they are much less likely to develop renal failure, pulmonary edema, or jaundice.

Cerebral malaria results in neurologic sequelae in 9-26% of children, but of these sequelae, approximately one half completely resolve with time.

**Treating uncomplicated *P. falciparum* malaria**

*Treatment of uncomplicated *P. falciparum* malaria*

Treat children and adults with uncomplicated *P. falciparum* malaria (except pregnant women in their first trimester) with one of the following recommended ACTs:
- artemether + lumefantrine
- artesunate + amodiaquine
- artesunate + mefloquine
- dihydroartemisinin + piperaquine
- artesunate + sulfadoxine–pyrimethamine (SP).

**Duration of ACT treatment**

ACT regimens should provide 3 days’ treatment with an artemisinin derivative.

**Revised dose recommendation for dihydroartemisinin + piperaquine in young children**

Children weighing <25kg treated with dihydroartemisinin + piperaquine should receive a minimum of 2.5 mg/kg bw per day of dihydroartemisinin and 20 mg/kg bw per day of piperaquine daily for 3 days.

**Reducing the transmissibility of treated *P. falciparum* infections**

In low-transmission areas, give a single dose of 0.25 mg/kg bw primaquine with ACT to patients with *P. falciparum* malaria (except pregnant women, infants aged < 6 months and women breastfeeding infants aged < 6 months) to reduce transmission. G6PD testing is not required.
BACTERIAL DISEASES
Pediatric Bacterial Meningitis

Pediatric bacterial meningitis is a life-threatening illness that results from bacterial infection of the meninges and leaves some survivors with significant sequelae.

**Signs and symptoms**

**The 3 classic symptoms (less likely in younger children):**
- Fever
- Headache
- Meningeal signs

**Symptoms in neonates:**
- Poor feeding
- Lethargy
- Irritability
- Apnea
- Listlessness
- Apathy
- Fever
- Hypothermia
- Seizures
- Jaundice
- Bulging fontanelle
- Pallor
- Shock
- Hypotonia
- Shri111 i cry
- Hypoglycemia
- Intractable metabolic acidosis

**Symptoms in infants and children:**
- Nuchal rigidity
- Opisthotonos
- Bulging fontanelle
- Convulsions
- Photophobia
- Headache
- Alterations of the sensorium
- Irritability
- Lethargy
- Anorexia
- Nausea
- Vomiting
- Coma
- Fever (generally present, although some severely ill children present with hypothermia)

The younger the child, the less likely he or she is to exhibit the classic symptoms of fever, headache, and meningeal signs.
Infants and children

Kernig (A) and Brudzinski (B) signs are helpful indicators when present, but they may be absent (along with nuchal rigidity) in very young, debilitated, or malnourished infants. Skin findings range from a nonspecific blanching, erythematous, maculopapular rash to a petechial or purpuric rash, and most characteristic of meningococcal meningitis.

Diagnosis

Definitive diagnosis is based on the following:

- Bacteria isolated from the CSF obtained via lumbar puncture
- Meningeal inflammation demonstrated by increased pleocytosis, elevated protein level, and low glucose level in the CSF
Management in the pre-hospital setting
Recognize shock and manage urgently in secondary care
Primary care healthcare professionals should transfer children and young people with suspected bacterial meningitis or suspected meningococcal septicaemia to secondary care as an emergency

http://www.guidelines.co.uk/nice/meningitis
General supportive care is required, depending on the child’s condition. Subsequent diagnosis of a potentially transmissible disease must be communicated to prehospital care providers, especially with *N. meningitidis* infections.

If the child is critically ill or experiencing a seizure, immediate stabilization and support are necessary. If the child is hemodynamically stable, IV fluids should be administered at maintenance. Careful record of the patient’s weight, urine specific gravity, and serum osmolarity will help guide further fluid therapy. Patients who present with dehydration should be rehydrated and should not undergo fluid restriction. Seizures should be treated promptly and should be expected at any time during the initial management. By prescribing the correct type and volume of fluid, the risk of brain edema can be minimized. The child should receive sufficient amounts of fluid to maintain systolic blood pressure at around 80 mm Hg, urinary output at 500 mL/m²/day, and adequate tissue perfusion.

Administration of antimicrobial agents to contacts is divided into high- and low-risk categories. Only contacts stratified as high-risk require prophylaxis. Candidates for chemoprophylaxis against meningococcal disease include the following:

- All household contacts
- Childcare or nursery school contacts during the 7 days before illness onset
- Contacts directly exposed to index case secretions through kissing, sharing toothbrushes or eating utensils, or other markers of close social contact during the 7 days before illness onset
- Persons who had mouth-to-mouth resuscitation or unprotected contact during endotracheal intubation in the 7 days before illness onset
- Contacts who frequently slept or ate in the same dwelling as the index patient during the 7 days before illness onset

### Chemoprophylaxis for Meningococcal Disease

| Adults and children >12 years | 1st line: Rifampicin 600mg every 12 hours for 4 doses  
| Female adults on the oral contraceptive pill | Ciprofloxacin 500mg po stat  
| Pregnant women | Ceftriaxone 250mg im stat  
| Children: 1-12 years | Rifampicin syrup 10mg/kg every 12 hours for 4 doses  
| Children 0 – 11 months | Rifampicin syrup 5mg/kg every 12 hours for 4 doses  

#### Chemoprophylaxis for Hib Disease

| Children and adults | Rifampicin 20mg/kg once daily for 4 days up to max of 600mg/day  
| Infants under 1 year of age | Rifampicin 10mg/kg once daily for 4 days  
| Pregnant women | Not indicated  

**Notes on rifampicin:** Rifampicin may colour urine / tears red and stain contact lenses – do not wear contact lenses for a few days after rifampicin treatment.
Pediatric aseptic meningitis is an inflammation of the meninges caused mainly by nonbacterial organisms, specific agents, or other disease processes. Aseptic meningitis (including viral meningitis) is the most common infection of the CNS in the pediatric population, occurring most frequently in children younger than 1 year.

Because the classic signs and symptoms are often absent, especially in younger children, diagnosing pediatric CNS infections is a challenge to the emergency department (ED).

**Clinical Presentation**

Headache, neck stiffness, and photophobia are classic symptoms of aseptic meningitis in older children. These symptoms may be absent in younger children, who more commonly present with rash, diarrhea, and cough. Fever may be present. Seizures are more common in aseptic meningitis caused by specific viruses (eg, arboviruses). Other nonspecific symptoms may include arthralgia, myalgia, sore throat, weakness, and lethargy and hypotonia.

Onset is usually acute but can be insidious over the week before presentation or can follow an acute febrile illness. Rash, when present, is erythematous, maculopapular, and vesicular, appearing on the soles of the feet, palms, or mucous membranes. Fever may last up to 5 days. Anorexia, nausea, and vomiting are common. Sore throat may occur. Rare symptoms include flaccid paralysis.

**Physical Examination**

The younger the child, the less specific the signs: In a young infant, findings that definitely point to meningitis are rare, but as the child grows older, the physical examination becomes more reliable. The infant may be febrile or hypothermic. Lymphadenopathy may be present. Bulging of the fontanel, diastasis of the sutures and nuchal rigidity point to meningitis but are usually late findings. Examination should specifically exclude a nonblanching petechial rash, other signs of bacterial meningitis.

Neurologic examination includes evaluating for signs of meningism (eg, headache, photophobia, neck stiffness, and positive Kernig or Brudzinski sign) and focal or generalized neurologic signs.

A definitive diagnosis of meningitis requires examination of CSF via lumbar puncture.

**Treatment & Management**

Management of pediatric aseptic meningitis is primarily supportive. Consultation with a pediatrician, an infectious disease specialist, a critical care specialist, or combinations thereof may be needed.

Administer adequate analgesia. Treat seizures with appropriate emergency therapies.
TETANUS

Tetanus is a severe infection due to the bacillus *Clostridium tetani*, which is found in soil, and human and animal waste. Tetanus can be acquired outdoors as well as indoors. The source of infection usually is a wound (approximately 65% of cases), which often is minor (eg, from wood or metal splinters or thorns). Tetanus may be categorized into the following 4 clinical types:

- Generalized tetanus
- Localized tetanus
- Cephalic tetanus
- Neonatal tetanus

Approximately 50-75% of patients with generalized tetanus present with trismus (“lockjaw”), which is the inability to open the mouth secondary to masseter muscle spasm. Nuchal rigidity and dysphagia are also early complaints that cause risus sardonicus, the scornful smile of tetanus, resulting from facial muscle involvement.

Localized tetanus present with persistent rigidity in the muscle group close to the injury site.

Cephalic tetanus is uncommon and usually occurs after head trauma or otitis media. Patients with this form present with cranial nerve (CN) palsies. The infection may be localized or may become generalized.

A high risk of mortality is associated with the following:

- Short incubation period
- Early onset of convulsions
- Delay in treatment
- Contaminated lesions of the head and the face
- Neonatal tetanus

Most cases of tetanus occur in patients with a history of underimmunization, either because they were never vaccinated or because they completed a primary series but have not had a booster in the preceding 10 years.

The median incubation period is 7 days, and for most cases (73%), incubation ranges from 4 to 14 days.

**Physical Examination**

Common first signs of tetanus are headache and muscular stiffness in the jaw (ie, lockjaw), followed by neck stiffness, difficulty swallowing, rigidity of abdominal muscles, spasms, and sweating. Patients often are afebrile. Stimulation of the posterior pharyngeal wall may elicit reflex spasms of the masseter muscles that cause patients to bite down as opposed to gag.

Severe tetanus results in opisthotonos, flexion of the arms, extension of the legs, periods of apnea resulting from spasm of the intercostal muscles and diaphragm, and rigidity of the abdominal wall. Late in the disease, autonomic dysfunction develops, with hypertension and tachycardia alternating with hypotension and bradycardia; cardiac arrest may occur.

Tetanic seizures may occur. Their presence portends a poor prognosis, and their frequency and severity are related to the severity of the disease. These seizures resemble epileptic seizures, with the presence of a sudden burst of tonic contractions. However, the patient does not lose consciousness and usually experiences severe pain.

Seizures frequently occur in the muscle groups causing opisthotonos, flexion and abduction of the arms, clenching of the fists against the thorax, and extension of the lower extremities. Patients with tetanus may present with abdominal tenderness and guarding, mimicking an acute abdomen.
Treatment & Management

Immediate communication to stakeholders and health professionals at national and local level. An intensive care medicine specialist should be the primary physician coordinating the patient’s care.

The goals of treatment in patients with tetanus include the following:

- Initiating supportive therapy
- Debriding the wound to eradicate spores and alter conditions for germination
- Stopping the production of toxin within the wound
- Neutralizing unbound toxin
- Controlling disease manifestations
- Managing complications

Patients should be admitted to the ICU. Because of the risk of reflex spasms, a dark and quiet environment should be maintained. Unnecessary procedures and manipulations should be avoided. Prophylactic intubation should be seriously considered in all patients with moderate-to-severe clinical manifestations.

Metronidazole (eg, 0.5 g every 6 hours) has comparable or better antimicrobial activity. Tetanus immune globulin (TIG) is recommended for treatment of tetanus. The World Health Organization recommends TIG 500 units by IM injection or intravenously (IV)—depending on the available preparation—as soon as possible.

Benzodiazepines have emerged as the mainstay of symptomatic therapy for tetanus. Diazepam is the most frequently studied and used drug; it reduces anxiety, produces sedation, and relaxes muscles. Lorazepam is an effective alternative. High dosages of either may be required (up to 600 mg/day). Maintenance of adequate nutrition is extremely important. Because of the risk of aspiration, patients should not be given any food by mouth.

Prevention

Prevention of tetanus is accomplished through vaccination with DTP or DTaP at the ages of 2 months, 4 months, 6 months, 12-18 months, and 4-6 years. Pregnancy is not a contraindication to the use of Tdap in the second and third trimester.

The following wounds should be considered prone to tetanus:

- Wounds that have been present for longer than 6 hours
- Deep (>1 cm) wounds
- Grossly contaminated wounds
- Wounds that are exposed to saliva or feces, stellate, or ischemic or infected (including abscesses
- Avulsions, punctures, or crush injuries

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<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Vaccination history</th>
<th>Clean, minor wounds</th>
<th>All other wounds</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 through 6</td>
<td>Unknown or not up-to-date on DTaP series based on age</td>
<td>DTaP</td>
<td>DTaP TIG</td>
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<tr>
<td></td>
<td>Up-to-date on DTaP series based on age</td>
<td>No indication</td>
<td>No indication</td>
</tr>
<tr>
<td>7 through 10</td>
<td>Unknown or incomplete DTaP series</td>
<td>Tdap and recommend catch-up vaccination</td>
<td>Tdap and recommend catch-up vaccination TIG</td>
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<td></td>
<td>Completed DTaP series AND &lt;5 years since last dose</td>
<td>No indication</td>
<td>No indication</td>
</tr>
<tr>
<td></td>
<td>Completed DTaP series AND ≥5 years since last dose</td>
<td>No indication</td>
<td>Td, but Tdap preferred if child is 10 years of age</td>
</tr>
<tr>
<td>11 years and older (*if pregnant, see footnote)</td>
<td>Unknown or &lt;3 doses of tetanus toxoid containing vaccine</td>
<td>Tdap and recommend catch-up vaccination</td>
<td>Tdap and recommend catch-up vaccination TIG</td>
</tr>
<tr>
<td></td>
<td>3 or more doses of tetanus toxoid containing vaccine AND &lt;5 years since last dose</td>
<td>No indication</td>
<td>No indication</td>
</tr>
<tr>
<td></td>
<td>3 or more doses of tetanus toxoid containing vaccine AND 5-10 years since last dose</td>
<td>No indication</td>
<td>Tdap preferred (if not yet received) or Td</td>
</tr>
<tr>
<td></td>
<td>3 or more doses of tetanus toxoid containing vaccine AND &gt;10 years since last dose</td>
<td>Tdap preferred (if not yet received) or Td</td>
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</tr>
</tbody>
</table>

**Pregnant Women:** As part of standard wound management care to prevent tetanus, a tetanus toxoid – containing vaccine might be recommended for wound management in a pregnant woman if 5 years or more have elapsed since the previous Td booster. If a Td booster is recommended for a pregnant woman, health-care providers should administer Tdap.
LEPTOSPIROSIS

Leptospirosis is an infectious disease of humans and animals that is caused by pathogenic spirochetes of the genus *Leptospira*. It is considered the most common zoonosis in the world.

**Signs and symptoms**
A good clinical history is often the key to accurate diagnosis in leptospirosis. Important features include a plausible exposure history and a clinical picture consistent with the disease. The exposure history may reveal direct contact with body fluids or organs of infected animals, or indirectly (eg, via contaminated soil or water). Leptospirosis occurs as two recognizable clinical syndromes: anicteric and icteric. Anicteric leptospirosis is a self-limited, mild flulike illness characterized by sudden onset of some combination of the following:

- Headache
- Fever (38-40°C)
- Rigors
- Muscle pain (typically localized to the calf and lumbar areas)
- Nausea and vomiting
- Anorexia
- Diarrhea
- Cough
- Pharyngitis
- Conjunctivitis
- Nonpruritic rash

Icteric leptospirosis, also known as Weil disease, is a severe illness whose classic manifestations include the following:

- Fever
- Jaundice
- Renal failure
- Hemorrhage

**Diagnosis**
Laboratory studies used to confirm the diagnosis include the following:
Culture of leptospires from body fluids or tissue (criterion standard)

**Management**
*Transfer to a facility with an appropriate level of care should be considered in patients with severe disease.*
Use of antibiotics in mild leptospirosis is controversial. If used, antibiotic treatment may include the following:

- Doxycycline
- Ampicillin
- Amoxicillin

Antibiotics for severe leptospirosis include the following:

- IV penicillin G (long the drug of choice)
- Third-generation cephalosporins (cefotaxime and ceftriaxone)
- Ampicillin or amoxicillin (second-line agents)
• Erythromycin (in penicillin-allergic pregnant women)
Patients with severe cases also require supportive therapy and careful management of renal, hepatic, hematologic, and central nervous system complications.
URINARY TRACT INFECTIONS
URINARY TRACT INFECTIONS IN ADULTS

Urinary tract infections are caused by the presence and multiplication of microorganisms in the urinary tract. A urinary tract infection can result in several clinical syndromes, including acute and chronic pyelonephritis (infection of the kidney and renal pelvis), cystitis (infection of the bladder), urethritis (infection of the urethra), epididymitis (infection of the epididymis) and prostatitis (infection of the prostate gland).

A urinary tract infection is defined by a combination of clinical features and the presence of bacteria in the urine. Asymptomatic bacteriuria is the occurrence of bacteria in the urine without causing symptoms. When symptoms occur as a result of bacteria this is referred to as symptomatic bacteriuria. The incidence of urinary tract infection is highest in young women. Most infections in adult men are complicated and related to abnormalities of the urinary tract, although some can occur spontaneously in otherwise healthy young men. Urinary tract infection incidence increases with age for both sexes.

URINARY TRACT INFECTION IN MALES

Signs and symptoms
Dysuria is the most frequent chief complaint in men with UTI. The combination of dysuria, urinary frequency, and urinary urgency is about 75% predictive for UTI.

Relevant clinical history includes the following:
- Previous UTI
- Nocturia, gross hematuria, any changes in the color and/or consistency of the urine
- Prostatic enlargement
- Urinary tract abnormalities: Personally and within the family
- Comorbid conditions (eg, diabetes)
- Human immunodeficiency virus (HIV) status
- Immunosuppressive treatments for other conditions (eg, prednisone)
- Any previous surgeries or instrumentation involving the urinary tract

Diagnosis
Perform a thorough physical examination in males presenting with genitourinary complaints. Focus particularly on the patient’s vital signs, kidneys, bladder, prostate, and external genitalia.

Examination findings may include the following:
- Fever
- Tachycardia
- Flank pain/costovertebral angle tenderness
- Abdominal tenderness in the suprapubic area
- Scrotal hematoma, hydrocele, masses, or tenderness
- Penile meatal discharge
- Prostatic tenderness
- Inguinal adenopathy

Consider imaging and urologic intervention in patients with the following:
- History of kidney stones, especially struvite stones: Potential for urosepsis
- Diabetes: Susceptibility to emphysematous pyelonephritis and may require immediate nephrectomy; diabetic patients may also develop obstruction from necrotic renal papillae that are sloughed into the collecting system and obstruct the ureter
- Polycystic kidneys: Prone to abscess formation
- Tuberculosis: Prone to developing ureteral strictures, fungus balls, and stones

If concomitant obstructive uropathy is suspected, this is an emergent condition that requires prompt intervention.

**Treatment & Management**

As a general rule, all urinary tract infections (UTIs) in men are considered complicated. Therefore, the possibility that infection has ascended to the kidneys must be considered, and treatment regimens must assume that infection of the upper urinary tract has occurred.

Initial inpatient treatment includes the following:
- Intravenous (IV) antimicrobial therapy with a third-generation cephalosporin (eg, ceftriaxone, ceftazidime), a fluoroquinolone (eg, ciprofloxacin, levofloxacin)
- Antipyretics
- Analgesics
- IV fluid resuscitation: To restore appropriate circulatory volume and promote adequate urinary flow

Consultation with a urologist is essential for the treatment of UTI in adult males with the following:
- Suspected underlying anatomic abnormality - However, this consultation can be completed on an outpatient basis, unless obstructive uropathy is present
- Acute scrotum - Consultation is needed in all but the most clear-cut cases of acute scrotum
- All forms of prostatitis - In acute bacterial prostatitis, suprapubic drainage may be required if acute urinary retention occurs

The following are suggested consultations:
- Infectious disease specialist - When unusual or resistant microorganisms have been isolated or if the infection is in an unusual host
- Pharmacokinetics specialist - When using aminoglycosides

**CYSTITIS IN FEMALES**

UTI are common in females.

**Signs and symptoms**

Symptoms and signs of UTI in the adult are as follows:
- Dysuria
- Urinary urgency and frequency
- A sensation of bladder fullness or lower abdominal discomfort
- Suprapubic tenderness
- Flank pain and costovertebral angle tenderness (may be present in cystitis but suggest upper UTI)
- Bloody urine
- Fevers, chills, and malaise (may be noted in patients with cystitis, but more frequently associated with upper UTI)

**Diagnosis**

Diagnostic studies for UTI consist of dipstick, urinalysis, and culture. No imaging studies are indicated in the routine evaluation of cystitis.

Current emphasis in the diagnosis of UTI rests with the detection of pyuria, as follows:
- A positive leukocyte esterase dipstick test suffices in most instances
- In females with clinical findings suggestive of UTI, urine microscopy may be indicated even if the leukocyte esterase dipstick test is negative
- Pyuria is most accurately measured by counting leukocytes in unspun fresh urine using a hemocytometer chamber; more than 10 white blood cells (WBCs)/mL is abnormal

Other findings are as follows:
- Microscopic hematuria is found in about half of cystitis cases
- Low-grade proteinuria is common
- A positive nitrate test is highly specific for UTI, but it occurs in only 25% of patients with UTI

Urine culture remains the criterion standard for the diagnosis of UTI.

Definitions of UTI in women, based on culture results in clean-catch urine specimens, are as follows:
- Cystitis: More than 1000 colony-forming units (CFU)/mL
- Pyelonephritis: More than 10,000 CFU/mL
- Asymptomatic bacteriuria: In a female, more than 100,000 CFU/mL in an asymptomatic individual

**Management**

Oral therapy with an empirically chosen antibiotic that is effective against gram-negative aerobic coliform bacteria (eg, *Escherichia coli*) is the principal treatment intervention in patients with cystitis. The first-choice agents for treatment of uncomplicated acute cystitis in women include the following:
- Nitrofurantoin monohydrate (FUROLIN TAB 100MG)
- Trimethoprim-sulfamethoxazole (TMP-SMX) (BACTRIMEL)
- Fosfomycin (FOSFOCIN)

Considerations in antibiotic selection are as follows:
- Empiric antibiotic selection is determined in part by local resistance patterns
- Beta-lactam antibiotics (eg, amoxicillin-clavulanate, cefaclor) may be used when other recommended agents cannot be used
- Fosfomycin and nitrofurantoin monohydrate should be avoided in patients with possible early pyelonephritis
- Clinicians may wish to limit use of TMP-SMX, to reduce the emergence of resistant organisms

Fluoroquinolones (CIPROFLOXACIN) are typically reserved for complicated cystitis.
Duration of antibiotic treatment for acute, uncomplicated cystitis in women who are not pregnant is as follows:

- TMP-SMX is given for 3 days
- Fosfomycin is given in a single dose
- Nitrofurantoin monohydrate is given for 5-7 days
- Beta-lactam agents are given for 3–7-days
- For cystitis in older women or infection caused by *Staphylococcus saprophyticus*, 7 days of therapy is suggested

Hospital admission may be indicated for some patients with complicated UTI. Complicating factors include the following:

- Structural abnormalities (eg, calculi, tract anomalies, indwelling catheter, obstruction)
- Metabolic disease (eg, diabetes, renal insufficiency)
- Impaired host defenses (eg, HIV infection, current chemotherapy, underlying active cancer)

**URINARY TRACT INFECTIONS IN PREGNANCY**

In general, pregnant patients are considered immunocompromised UTI hosts because of the physiologic changes associated with pregnancy.

Oral antibiotics are the treatment of choice for asymptomatic bacteriuria and cystitis. The standard course of treatment for pyelonephritis is hospital admission and intravenous antibiotics.

UTI is defined as the presence of at least 100,000 organisms per milliliter of urine in an asymptomatic patient, or as more than 100 organisms/mL of urine with accompanying pyuria (> 7 white blood cells [WBCs]/mL) in a symptomatic patient. A diagnosis of UTI should be supported by a positive culture for a uropathogen, particularly in patients with vague symptoms. UTIs are associated with risks to both the fetus and the mother, including pyelonephritis, preterm birth, low birth weight, and increased perinatal mortality.

Acute cystitis involves only the lower urinary tract; it is characterized by inflammation of the bladder as a result of bacterial or nonbacterial causes. Signs and symptoms include hematuria, dysuria, suprapubic discomfort, frequency, urgency, and nocturia. These symptoms are often difficult to distinguish from those due to pregnancy itself.

Pyelonephritis is the most common urinary tract complication in pregnant women. Acute pyelonephritis is characterized by fever (>38°C), flank pain, and tenderness in addition to significant bacteriuria. Other symptoms may include nausea, vomiting, frequency, urgency, and dysuria.

**Treatment & Management**

**Treatment of bacteriuria and cystitis**

Because of the dangers of maternal and fetal complications, acute care (eg, in the ED) should focus on identifying and treating asymptomatic and symptomatic bacteriuria, along with ensuring that an alternate process is not the cause of the symptoms.

Treatment of asymptomatic bacteriuria in pregnant patients is important because of the increased risk of UTI and its associated sequelae. ED care may involve the following:
• Administration of appropriate antibiotics
• Administration of fluid if the patient is dehydrated
• Admission if any indication of complicated UTI exists

may be used to ensure good hygiene and reduce bacterial contamination of the urethral meatus, thereby preventing inadequate treatment and recurrent infection. Behavioral methods include the following:
• Avoid baths
• Wipe front-to-back after urinating or defecating
• Wash hands before using the toilet
• Use washcloths to clean the perineum
• Use liquid soap to prevent colonization from bar soap
• Clean the urethral meatus first when bathing

Antibiotic therapy
Oral antibiotics are the treatment of choice for asymptomatic bacteriuria and cystitis. Appropriate oral regimens include the following:

Treatment Regimens for Pregnant Women with UTI

First-line therapy

• Nitrofurantoin monohydrate/ 100 mg orally twice daily for 5-7 days or
• Amoxicillin 500 mg orally twice daily (alternative: 250 mg orally three times daily) for 5-7 days or
• Amoxicillin-clavulanate 500/125 mg orally twice daily for 3-7 days (alternative: 250/125 mg orally three times daily for 5-7 days)

Second-line therapy

• Fosfomycin 3 g orally as single dose with 3-4 oz. of water

Treatment of pyelonephritis
The standard course of treatment for pyelonephritis consists of hospital admission and IV administration of cephalosporins or gentamicin. IV fluids must be administered with caution. Antibiotic selection should be based on urine culture sensitivities, if known. Often, therapy must be initiated on an empirical basis, before culture results are available.
PEDIATRIC URINARY TRACT INFECTION

UTI is one of the most common pediatric infections. Febrile infants younger than 2 months constitute an important subset of children who may present with fever without a localizing source. The workup of fever in these infants should always include evaluation for UTI. The chart below details a treatment approach for febrile infants younger than 3 months who have a temperature higher than 38°C.

**Signs and symptoms**
The history and clinical course of a UTI vary with the patient's age and the specific diagnosis. No one specific sign or symptom can be used to identify UTI in infants and children.

**Children aged 0-2 months**
Neonates and infants up to age 2 months who have pyelonephritis usually do not have symptoms localized to the urinary tract. UTI is discovered as part of an evaluation for neonatal sepsis. Neonates with UTI may display the following symptoms:

- Jaundice
- Fever
- Failure to thrive
- Poor feeding
- Vomiting
Infants and children aged 2 months to 2 years
Infants with UTI may display the following symptoms:
- Poor feeding
- Fever
- Vomiting
- Strong-smelling urine
- Abdominal pain
- Irritability

Children aged 2-6 years
Preschoolers with UTI can display the following symptoms:
- Vomiting
- Abdominal pain
- Fever
- Strong-smelling urine
- Enuresis
- Urinary symptoms (dysuria, urgency, frequency)

Children older than 6 years and adolescents
School-aged children with UTI can display the following symptoms:
- Fever
- Vomiting, abdominal pain
- Flank/back pain
- Strong-smelling urine
- Urinary symptoms (dysuria, urgency, frequency)
- Enuresis
- Incontinence

Physical examination findings in pediatric patients with UTI can be summarized as follows:
- Costovertebral angle tenderness
- Abdominal tenderness to palpation
- Suprapubic tenderness to palpation
- Palpable bladder
- Dribbling, poor stream, or straining to void

Diagnosis
Criteria for the diagnosis of UTI in children 2-24 months are the presence of pyuria and/or bacteriuria on urinalysis and of at least 50,000 colony-forming units (CFU) per mL of a uropathogen from the quantitative culture of a properly collected urine specimen. Urinalysis alone is not sufficient for diagnosing UTI. However, urinalysis can help in identifying febrile children who should receive antibacterial treatment while culture results from a properly collected urine specimen are pending.

Management
Patients with a nontoxic appearance may be treated with oral fluids and antibiotics. Outpatient care is reasonable if the following criteria are met:
- A caregiver with appropriate observational and coping skills
- Telephone and automobile at home
The ability to return to the hospital within 24 hours
The patient has no need for oxygen therapy, intravenous fluids, or other inpatient measures

Hospitalization is necessary for the following patients with UTI:

- Patients who are toxemic or septic
- Patients with signs of urinary obstruction or significant underlying disease
- Patients who are unable to tolerate adequate oral fluids or medications
- Infants younger than 2 months with febrile UTI (presumed pyelonephritis)
- All infants younger than 1 month with suspected UTI, even if not febrile

Treat febrile UTI as pyelonephritis, and consider parenteral antibiotics and hospital admission for these patients.

Antibiotics for parenteral treatment are as follows:

- Ceftriaxone
- Cefotaxime
- Ampicillin
- Gentamicin

_Patients aged 2 months to 2 years with a first febrile UTI_

If clinical findings indicate that immediate antibiotic therapy is indicated, a urine specimen for urinalysis and culture should be obtained before treatment is started. Common choices for empiric oral treatment are as follows:

- A second- or third-generation cephalosporin
- Amoxicillin/clavulanate, or sulfamethoxazole-trimethoprim (SMZ-TMP)

_Children with cystitis_

- Antibiotic therapy is started on the basis of clinical history and urinalysis results before the diagnosis is documented
- A 4-day course of an oral antibiotic agent is recommended for the treatment of cystitis
- Nitrofurantoin can be given for 7 days or for 3 days after obtaining sterile urine
- If the clinical response is not satisfactory after 2-3 days, alter therapy on the basis of antibiotic susceptibility
- Symptomatic relief for dysuria consists of increasing fluid intake (to enhance urine dilution and output), paracetamol, and NSAID

_Empiric Therapy Regimens_

Empiric therapeutic regimes for pediatric urinary tract infections are provided below based on patient age.

**Age < 2mo**

See the list below:

- Cefotaxime 150 mg/kg/day IV/IM divided q6-8h
- Ceftriaxone 75 mg/kg/day IV/IM as a single dose or divided q12h (ceftriaxone should not be used in infants younger than 6wk) or
- Ampicillin 100 mg/kg/day IV/IM divided q8h plus gentamicin 3.5-5 mg/kg/dose IV q24h if patient younger than 7d, otherwise gentamicin 5-7.5 mg/kg/dose IV q24h
• Transition to oral antibiotic active against the offending organism after 24-48h for total of 14d course

**Age 2mo to 18y**

**Outpatient therapy:**

• Nitrofurantoin 5-7 mg/kg PO divided q6h for 3-10d or
  - Contraindicated in Children < 3 months of age or when GFR < 50% or in children with G6PD deficiency
  - Should not be used in children with symptoms consistent with pyelonephritis as it is poorly concentrated in the bloodstream and has poor tissue penetration or

• Trimethoprim (TMP) and sulfamethoxazole 6-12 mg/kg/day PO divided q12h, based on TMP component or
  - Contraindicated in children < 6 weeks of age

• Amoxicillin clavulanic acid - 20-40 mg/kg/day divided q8h or

• Cefuroxime axetil 20-30 mg/kg/day divided q12h

**Red Flag**

Studies have shown oral antibiotics to be as effective as IV antibiotics in most cases of simple pediatric cystitis

Most children may be treated with oral medications; those deemed “toxic” or are unable to retain oral intake may require parental treatment

Short-course (3d or 5d) oral antibiotic therapy has been shown to be as effective as 10-d or 14-d courses for non-febrile UTIs

For febrile UTIs, the minimum treatment duration should be 7d and may extend to 10-14d
PEDIATRIC NEPHROTIC SYNDROME

Nephrotic syndrome, or nephrosis, is defined by the presence of nephrotic-range proteinuria, edema, hyperlipidemia, and hypoalbuminemia. While nephrotic-range proteinuria in adults is characterized by protein excretion of 3.5 g or more per day, in children it is defined as protein excretion of more than 40 mg/m²/h or a first-morning urine protein/creatinine of 2-3 mg/mg creatinine or greater.

Signs and symptoms
Pitting edema is the presenting symptom in about 95% of children with nephrotic syndrome. It is typically found in the lower extremities, face and periorbital regions, scrotum or labia, and abdomen (ascites). Early on, the edema is intermittent and insidious, and its presence may not be appreciated. A common story is for the child to present to a primary care practitioner repeatedly for periorbital edema, which is ascribed to "allergies" until the edema progresses.

Other signs and symptoms of nephrotic syndrome may include the following:
- Respiratory tract infection - A history of a respiratory tract infection immediately preceding the onset of nephrotic syndrome is frequent
- Allergy - Approximately 30% of children with nephrotic syndrome have a history of allergy
- Macrohematuria
- Symptoms of infection - Such as fever, lethargy, irritability, or abdominal pain due to sepsis or peritonitis
- Hypotension and signs of shock - Can be present in children presenting with sepsis
- Respiratory distress - Due to either massive ascites and thoracic compression or frank pulmonary edema, effusions, or both
- Tachypnea - To compensate for mechanical restriction to breathing
- Seizure - Due to cerebral thrombosis
- Anorexia
- Irritability
- Fatigue
- Abdominal discomfort
- Diarrhea
- Hypertension

Diagnosis
In order to establish the presence of nephrotic syndrome, laboratory tests should confirm the existence of (1) nephrotic-range proteinuria, (2) hypoalbuminemia, and (3) hyperlipidemia. Therefore, initial laboratory testing should include the following:
- Urinalysis
- Urine protein quantification
- Serum albumin
- Lipid panel

The following tests should be performed to determine whether the nephrotic syndrome is idiopathic or secondary and, if INS has been determined, whether signs of chronic kidney disease, kidney insufficiency, or other signs exclude the possibility of minimal change nephrotic syndrome:
- CBC
- Metabolic panel - levels of serum electrolytes, calcium, phosphorus, and ionized calcium, as well as of blood urea nitrogen (BUN) and creatinine
- Testing for HIV
- Testing for hepatitis B and C
- Complement studies (C3, C4)
- ANA, anti–double-stranded DNA antibody (in selected patients)

Other tests and procedures in selected patients can include the following:
- Genetic studies
- Kidney ultrasonography
- Chest radiography
- Mantoux test
- Kidney biopsy

**Management**

**Hospitalize the child for initial therapy.**

**Corticosteroids**

If kidney biopsy is not initially indicated, a trial of corticosteroids is the first step in the treatment of INS.

Definition of nephrotic syndrome: Edema, urine protein: creatinine ratio ≥2000 mg/g; urine protein ≥300 mg/dL, dipstick urine protein 3+, hypoalbuminemia ≤2.5 mg/L

Initial treatment: Oral prednisone, starting as a daily dose of 60 mg/m$^2$/day or 2 mg/kg/day (maximum, 60 mg/day) for 4–6 weeks. After 4–6 weeks, switch to 40 mg/m$^2$ or 1.5 mg/kg (maximum, 40 mg) on alternate days for 2–5 months with tapering, with a minimum total duration of treatment of 12 weeks

**Diuretics**

Loop diuretics, such as furosemide (starting at 1-2 mg/kg/d), may improve edema. Metolazone may be beneficial in combination with furosemide for resistant edema.

**Antihypertensive agents**

ACE inhibitors and ARBs can reduce hypertension and may also contribute to reducing proteinuria. Calcium channel blockers and beta blockers may also be used as first-line agents for hypertension.

Home monitoring of urine protein and fluid status is an important aspect of management. All patients and parents should be trained to monitor first morning urine proteins at home with urine dipstick. Urine testing at home is also useful in monitoring response (or nonresponse) to steroid treatment.
NEPHROLITHIASIS

Nephrolithiasis specifically refers to calculi in the kidneys, but renal calculi and ureteral calculi (ureterolithiasis) are often discussed in conjunction. The majority of renal calculi contain calcium. The pain generated by renal colic is primarily caused by dilation, stretching, and spasm because of the acute ureteral obstruction.

Signs and symptoms
The classic presentation for a patient with acute renal colic is the sudden onset of severe pain originating in the flank and radiating inferiorly and anteriorly; at least 50% of patients will also have nausea and vomiting. Patients with urinary calculi may report pain, infection, or hematuria. Patients with small, nonobstructing stones or those with staghorn calculi may be asymptomatic or experience moderate and easily controlled symptoms.

The location and characteristics of pain in nephrolithiasis include the following:
- Stones obstructing ureteropelvic junction: Mild to severe deep flank pain without radiation to the groin; irritative voiding symptoms (eg, frequency, dysuria); suprapubic pain, urinary frequency/urgency, dysuria, stranguria, bowel symptoms
- Stones within ureter: Abrupt, severe, colicky pain in the flank and ipsilateral lower abdomen; radiation to testicles or vulvar area; intense nausea with or without vomiting
- Upper ureteral stones: Radiate to flank or lumbar areas
- Midureteral calculi: Radiate anteriorly and caudally
- Distal ureteral stones: Radiate into groin or testicle (men) or labia majora (women)
- Stones passed into bladder: Mostly asymptomatic; rarely, positional urinary retention

Diagnosis
The diagnosis of nephrolithiasis is often made on the basis of clinical symptoms alone, although confirmatory tests are usually performed.

Examination in patients with nephrolithiasis includes the following findings:
- Dramatic costovertebral angle tenderness; pain can move to upper/lower abdominal quadrant with migration of ureteral stone
- Generally unremarkable abdominal evaluation: Possibly hypoactive bowel sounds; usually absence of peritoneal signs; possibly painful testicles but normal-appearing
- Constant body positional movements (eg, writhing, pacing)
- Tachycardia
- Hypertension
- Microscopic hematuria

Laboratory tests in all patients with an acute stone episode:
- Urinary sediment/dipstick test: To demonstrate blood cells, with a test for bacteriuria (nitrite) and urine culture in case of a positive reaction
- Serum creatinine level: To measure renal function

Other laboratory tests that may be helpful include the following:
- CBC with differential in febrile patients
- Serum electrolyte assessment in vomiting patients (eg, sodium, potassium, calcium, PTH, phosphorus)
- Serum and urinary pH level: May provide insight regarding patient’s renal function and type of calculus (e.g., calcium oxalate, uric acid, cystine), respectively
- Microscopic urinalysis
- 24-Hour urine profile

Non-contrast abdominopelvic CT scan: The imaging modality of choice for assessment of urinary tract disease, especially acute renal colic
Renal ultrasonography: To determine presence of a renal stone and the presence of hydronephrosis or ureteral dilation; used alone or in combination with plain abdominal radiography

Management

Supportive care and pharmacotherapy
Medical treatment of nephrolithiasis involves supportive care and administration of agents, such as the following:
- IV hydration
- Nonnarcotic analgesics
- PO/IV narcotic analgesics
- NSAID
- Uricosuric agents (e.g., allopurinol)
- Antiemetics (e.g., metoclopramide)
- Antibiotics (e.g., ampicillin, gentamicin, ciprofloxacin, levofloxacin, ofloxacin)
- Alkalinizing agents (e.g., potassium citrate, sodium bicarbonate): For uric acid and cysteine calculi

Pain relief
For patients with ureteral stones that are expected to pass spontaneously, NSAID tablets or suppositories (e.g., diclofenac sodium, 100-150 mg/day, 3-10 days) may help reduce inflammation and risk of recurrent pain. Although diclofenac can affect renal function in patients with already reduced function, it has no effect in patients with normal kidney function. NSAIDs are effective in patients with acute stone colic, and have better analgesic efficacy than opioids. Patients receiving NSAIDs are less likely to require further analgesia in the short term.

Recommendations for analgesia during renal colic
1. First choice: start with an NSAID, e.g. diclofenac, indomethacin or ibuprofen.
2. Second choice: hydromorphone, pentazocine or tramadol.
3. a-blockers to reduce recurrent colics.

Morphine is a potent narcotic analgesic that controls severe pain primarily through a CNS mechanism via specific receptor site interactions. The usual dosage is 10 mg/70 kg body weight IM every 4 hours. The actual dosage required varies according to each individual patient’s tolerance and severity of discomfort.
The obstructed kidney with all signs of UTI is a urological emergency. Urgent decompression is often necessary to prevent further complications in infected hydronephrosis secondary to stone-induced, unilateral or bilateral renal obstruction.
Hospital admission is clearly necessary when any of the following is present:

- Oral analgesics are insufficient to manage the pain.
- Ureteral obstruction from a stone occurs in a solitary or transplanted kidney.
- Ureteral obstruction from a stone occurs in the presence of a urinary tract infection (UTI), fever, sepsis, or pyonephrosis.
ACUTE PYELONEPHRITIS

Acute pyelonephritis is a potentially organ- and/or life-threatening infection that often leads to renal scarring. Acute pyelonephritis results from bacterial invasion of the renal parenchyma. Bacteria usually reach the kidney by ascending from the lower urinary tract. Bacteria may also reach the kidney via the bloodstream.

Signs and symptoms
The classic presentation in patients with acute pyelonephritis is as follows:
- Fever - This is not always present, but when it is, it is not unusual for the temperature to exceed 39.4°C
- Costovertebral angle pain - Pain may be mild, moderate, or severe; flank or costovertebral angle tenderness is most commonly unilateral over the involved kidney, although bilateral discomfort may be present
- Nausea and/or vomiting - These vary in frequency and intensity, from absent to severe; anorexia is common in patients with acute pyelonephritis

Gross hematuria (hemorrhagic cystitis), unusual in males with pyelonephritis, occurs in 30-40% of females, most often young women, with the disorder.

Symptoms of acute pyelonephritis usually develop over hours or over the course of a day but may not occur at the same time. If the patient is male, elderly, or a child or has had symptoms for more than 7 days, the infection should be considered complicated until proven otherwise.

The classic manifestations of acute pyelonephritis observed in adults are often absent in children, particularly neonates and infants. In children aged 2 years or younger, the most common signs and symptoms of UTI are as follows:
- Failure to thrive
- Feeding difficulty
- Fever
- Vomiting

Elderly patients may present with typical manifestations of pyelonephritis, or they may experience the following:
- Fever
- Mental status change
- Decompensation in another organ system
- Generalized deterioration

Diagnosis
In the outpatient setting, pyelonephritis is usually suggested by a patient’s history and physical examination and supported by urinalysis results.
- When diagnosis suspected, always obtain urine (via clean-catch, mid-stream sample OR catheterized specimen) for urinalysis and culture (with antibiotic susceptibility testing).
  - Urine specimen: should be received in the laboratory within 1 h of collection (or stored at 4°C and tested within 18h) to reduce risk of overgrowth of bacteria.
  - Bacterial colony counts typically >100,000 CFU/mL.
  - Absence of pyuria and bacteriuria suggest an alternative diagnosis (unless obstruction present).
• Although blood cultures are positive in 20–30% of cases, there is little evidence that results influence management or outcome.
• Physical examination should include costovertebral angle percussion, abdominal examination, and possibly pelvic examination.
• Pregnancy testing should be performed for all women of child-bearing age.

TREATMENT
Empiric Outpatient

• **Empiric, initial, oral, outpatient treatment:** if local rates of *E. coli* fluoroquinolone resistance are low (< 10%):
  - Ciprofloxacin 500 mg PO twice daily x 7 d
  - Ciprofloxacin extended release 1000 mg PO x 7 d
  - Levofoxacin 750 mg orally x 5-7 d

Modify initial treatment based upon results of urine culture and sensitivity.

• While trimethoprim/sulfamethoxazole should not be used for initial empiric therapy because of high rates of resistance, TMP/SMX 160/800 mg (one DS tablet) PO twice daily x 14 days is appropriate treatment of uncomplicated cystitis for pathogens known to be sensitive.
• Oral beta-lactams are second-line agents due to high rates of relapse (even when pathogen is susceptible).

Duration: typically 48h parenteral therapy or until afebrile, then switch to oral therapy based upon susceptibility data to complete 7d (fluoroquinolone) or 14d (TMP-SMX) course.

• If beta-lactam is used to complete therapy, 10-14 days duration needed.

Criteria for hospitalization: can be treated as an outpatient if patient stable. If below factors present, consider inpatient treatment.

• Pregnancy
• Emesis (inability to reliably keep down oral medications)
• Sepsis parameters, systemic inflammatory response syndrome (SIRS)
• Complicated pyelonephritis infection (including men)

Complications: renal or perinephric abscess, emphysematous pyelonephritis, nephronia (focal bacterial nephritis), renal papillary necrosis.
VAGINITIS

Vaginitis (inflammation of the vagina) is the most common gynecologic condition encountered in the office. It is a diagnosis based on the presence of symptoms of abnormal discharge, vulvovaginal discomfort, or both. Cervicitis may also cause a discharge and sometimes occurs with vaginitis.

Vulvovaginal Candidiasis

Patients with vaginitis almost always present with a chief complaint of abnormal vaginal discharge. Ascertain the following attributes of the discharge:

- Quantity
- Duration
- Color
- Consistency
- Odor

A diagnosis of *Candida* vaginitis is suggested clinically by the presence of external dysuria and vulvar pruritus, pain, swelling, and redness. Signs include vulvar edema, fissures, excoriations, and thick curdy vaginal discharge.

Treatment

- Clotrimazole 2% cream 5 g intravaginally daily for 3 days or
- Miconazole 2% cream 5 g intravaginally daily for 7 days or
- Fluconazole 150 mg orally in a single dose

Management of Sex Partners

Uncomplicated VVC is not usually acquired through sexual intercourse; thus, data do not support treatment of sex partners. A minority of male sex partners have balanitis, characterized by erythematous areas on the glans of the penis in conjunction with pruritus or irritation. These men benefit from treatment with topical antifungal agents to relieve symptoms.

Complicated VVC

Diagnostic Considerations

Vaginal cultures should be obtained from women with complicated VVC to confirm clinical diagnosis and identify unusual species, including nonalbicans species, particularly *Candida glabrata*.

CERVICITIS

Two major diagnostic signs characterize cervicitis:

1) A purulent or mucopurulent endocervical exudate visible in the endocervical canal or on an endocervical swab specimen (commonly referred to as mucopurulent cervicitis) and

2) Sustained endocervical bleeding easily induced by gentle passage of a cotton swab through the cervical os. Either or both signs might be present. Cervicitis frequently is asymptomatic, but some women complain of an abnormal vaginal discharge and intermenstrual vaginal bleeding (e.g., after sexual intercourse).
When an etiologic organism is isolated in the presence of cervicitis, it is typically *C. trachomatis* or *N. gonorrhoeae*. Cervicitis also can accompany trichomoniasis and genital herpes (especially primary HSV-2 infection). However, in most cases of cervicitis, no organism is isolated, especially in women at relatively low risk for recent acquisition of these STDs (e.g., women aged >30 years).

**Clinical Presentation**

A focused review of symptoms is recommended that asks about the following:

- Dyspareunia
- Vaginal discharge
- Genital skin lesions
- Abnormal vaginal bleeding
- Dysuria
- Genital burning
- Genital itching
- Genital malodor
- Lower abdominal or pelvic pain

**Recommended Regimens for Presumptive Treatment***

- **Azithromycin** 1 g orally in a single dose
  - OR
- **Doxycycline** 100 mg orally twice a day for 7 days

*Consider concurrent treatment for gonococcal infection if patient is at risk for gonorrhea or lives in a community where the prevalence of gonorrhea is high.
A spontaneous complaint of abnormal vaginal discharge—abnormal in terms of quantity, colour or odour—most commonly indicates a vaginal infection or vaginitis. Vaginal discharge due to bacterial vaginosis (multiple organisms) or yeast infection (Candida albicans) is not sexually transmitted, while trichomoniasis (Trichomonas vaginalis) usually is. Much less often, vaginal discharge may be the result of mucopurulent cervicitis due to gonorrhoea (Neisseria gonorrhoeae) or chlamydia (Chlamydia trachomatis).

All women presenting with abnormal vaginal discharge should receive treatment for bacterial vaginosis and trichomoniasis. Additional treatment for yeast infection is indicated when clinically apparent (white, curd-like discharge, redness of the vulva and vagina, and itching).
Hospitalization of patients with acute pelvic inflammatory disease should be seriously considered when:

- a surgical emergency, such as appendicitis or ectopic pregnancy, cannot be excluded;
- a pelvic abscess is suspected;
- severe illness precludes management on an outpatient basis;
- the patient is pregnant;
- the patient is an adolescent;
- the patient is unable to follow or tolerate an outpatient regimen; or
- the patient has failed to respond to outpatient therapy.
GENITAL ULCER (FOR BOTH MEN AND WOMEN)

Patient complains of a genital sore or ulcer

Take history and examine

Only vesicles present? NO

Sore or ulcer present? NO

TREAT FOR HSV2
TREAT FOR SYPHILIS IF INDICATED*

TREAT FOR SYPHILIS AND CHANCROID
TREAT FOR HSV2**

* Indications for syphilis treatment
  - RPR positive
  - No recent syphilis treatment
** Treat for HSV2 where prevalence is 30% or higher, or adapt to local conditions

Programme manager: Adapt based on local prevalence

Educate and counsel on risk reduction
Promote and provide condoms
Offer HIV counselling and testing if available
Review in 7 days

Educate and counsel on risk reduction
Promote and provide condoms
Offer HIV counselling and testing if available

Ulcer(s) healed? NO

Ulcer(s) improving? NO

Refer

YES

Continue treatment for a further 7 days
Genital bleeding unrelated to the menstrual period. Abnormal uterine bleeding is a common presenting problem in the ED. Dysfunctional uterine bleeding is defined as abnormal uterine bleeding in the absence of organic disease. Dysfunctional uterine bleeding is the most common cause of abnormal vaginal bleeding during a woman’s reproductive years.

**Clinical Presentation**

The amount and frequency of bleeding and the duration of symptoms, as well as the relationship to the menstrual cycle, should be established. Ask patients to compare the number of pads or tampons used per day in a normal menstrual cycle to the number used at the time of presentation. The average tampon or pad absorbs 20-30 mL or vaginal effluent. Personal habits vary greatly among women; therefore, the number of pads or tampons used is unreliable. The patient should be questioned about the possibility of pregnancy.

A reproductive history should always be obtained, including the following:

- Age of menarche and menstrual history and regularity
- Last menstrual period (LMP), including flow, duration, and presence of dysmenorrhea
- Postcoital bleeding
- Gravida and para
- Previous abortion or recent termination of pregnancy
- Contraceptive use, use of barrier protection, and sexual activity (including vigorous sexual activity or trauma)
- History of sexually transmitted diseases (STDs) or ectopic pregnancy

Questions about medical history should include the following:

- Signs and symptoms of anemia or hypovolemia (including fatigue, dizziness, and syncope)
- Diabetes mellitus
- Thyroid disease
- Endocrine problems or pituitary tumors
- Liver disease
- Recent illness, psychological stress, excessive exercise, or weight change
- Medication usage, including exogenous hormones, anticoagulants, aspirin, anticonvulsants, and antibiotics
- Alternative and complementary medicine modalities, such as herbs and supplements

Patients who are hemodynamically stable require a pelvic speculum, bimanual, and rectovaginal examination to define the etiology of vaginal bleeding. A careful physical examination will exclude vaginal or rectal sources of bleeding. The examination should look for the following:

- The vagina should be inspected for signs of trauma, lesions, infection, and foreign bodies.
- The cervix should be visualized and inspected for lesions, polyps, infection, or intrauterine device (IUD).
- Bleeding from the cervical os
- A rectovaginal examination should be performed to evaluate the cul-de-sac, posterior wall of the uterus, and uterosacral ligaments.

Patients with severe, acute abnormal uterine bleeding and hemodynamic instability will require urgent gynecologic consultation and hospitalization.
Hemodynamically unstable patients with uncontrolled bleeding and signs of significant blood loss should have aggressive resuscitation with saline and blood as with other types of hemorrhagic shock.

- Evaluate ABCs and address the priorities.
- Initiate 2 large-bore intravenous lines (IVs), oxygen, and cardiac monitor.
HEADACHES IN OVER 12S: DIAGNOSIS AND MANAGEMENT

All recommendations apply to adults and young people aged 12 years and over.

Assessment

- Evaluate people who present with headache and any of the following features, and consider the need for further investigations and/or referral:
  - worsening headache with fever
  - sudden-onset headache reaching maximum intensity within 5 minutes
  - new-onset neurological deficit
  - new-onset cognitive dysfunction
  - change in personality
  - impaired level of consciousness
  - recent (typically within the past 3 months) head trauma
  - headache triggered by cough, valsalva (trying to breathe out with nose and mouth blocked) or sneeze
  - headache triggered by exercise
  - orthostatic headache (headache that changes with posture)
  - symptoms suggestive of giant cell arteritis
  - symptoms and signs of acute narrow-angle glaucoma
  - a substantial change in the characteristics of their headache

- Consider further investigations and/or referral for people who present with new-onset headache and any of the following:
  - compromised immunity, caused, for example, by HIV or immunosuppressive drugs
  - age under 20 years and a history of malignancy
  - a history of malignancy known to metastasise to the brain
  - vomiting without other obvious cause

- Consider using a headache diary to aid the diagnosis of primary headaches

- If a headache diary is used, ask the person to record the following for a minimum of 8 weeks:
  - frequency, duration and severity of headaches
  - any associated symptoms
  - all prescribed and over the counter medications taken to relieve headaches
  - possible precipitants
  - relationship of headaches to menstruation

**Cluster headache bout:** The duration over which recurrent cluster headaches occur, usually lasting weeks or months. Headaches occur from 1 every other day to 8 times per day
### Diagnosis of tension-type headache, migraine, and cluster headache

<table>
<thead>
<tr>
<th>HEADACHE FEATURE</th>
<th>TENSION-TYPE HEADACHE</th>
<th>MIGRAINE (WITH OR WITHOUT AURA)</th>
<th>CLUSTER HEADACHE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pain location</td>
<td>Bilateral</td>
<td>Unilateral or bilateral</td>
<td>Unilateral (around the eye, above the eye and along the side of the head/face)</td>
</tr>
<tr>
<td>Pain quality</td>
<td>Pressing/tightening (non-pulsating)</td>
<td>Pulsating (throbbing or banging in young people aged 12–17 years)</td>
<td>Variable (can be sharp, boring, burning, throbbing or tightening)</td>
</tr>
<tr>
<td>Pain intensity</td>
<td>Mild or moderate</td>
<td>Moderate or severe</td>
<td>Severe or very severe</td>
</tr>
<tr>
<td>Effect on activities</td>
<td>Not aggravated by routine activities of daily living</td>
<td>Aggravated by, or causes avoidance of, routine activities of daily living</td>
<td>Restlessness or agitation</td>
</tr>
</tbody>
</table>
| Other symptoms   | None                  | Unusual sensitivity to light and/or sound or nausea and/or vomiting Aura (see Migraine with aura in main text) Symptoms can occur with or without headache and:  
  - are fully reversible  
  - develop over at least 5 minutes  
  - last 5–60 minutes  
  Typical aura symptoms include visual symptoms such as flickering lights, spots or lines and/or partial loss of vision; sensory symptoms such as numbness and/or pins and needles; and/or speech disturbance | On the same side as the headache:  
  - red and/or watery eye  
  - nasal congestion and/or runny nose  
  - swollen eyelid  
  - forehead and facial sweating  
  - constricted pupil and/or drooping eyelid |
| Duration of      | 30 minutes–continuous | 4–72 hours in adults 1–72 hours in young people | 15–180 minutes |
### Diagnosis of tension-type headache, migraine, and cluster headache

<table>
<thead>
<tr>
<th>HEADACHE FEATURE*†</th>
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<th>MIGRAINE (WITH OR WITHOUT AURA)</th>
<th>CLUSTER HEADACHE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>aged 12–17 years</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frequency of headache</td>
<td>&lt;15 days per month</td>
<td>≥15 days per month for more than 3 months</td>
<td>1 every other day to 8 per day,† with a continuous remission§ &gt;1 month in a 12 month period</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Episodic tension-type headache</td>
<td>Episodic migraine (with or without aura)</td>
<td>Episodic cluster headache</td>
</tr>
<tr>
<td></td>
<td>Chronic tension-type headache†</td>
<td>Chronic migraine§ (with or without aura)</td>
<td>Chronic cluster headache</td>
</tr>
</tbody>
</table>

* Headache pain can be felt in the head, face or neck
† The frequency of recurrent headaches during a cluster headache bout
‡ The pain-free period between cluster headache bouts
¶ Chronic migraine and chronic tension-type headache commonly overlap. If there are any features of migraine, diagnose chronic migraine
§ NICE has developed technology appraisal guidance on Botulinum toxin type A for the prevention of headaches in adults with chronic migraine (headaches on at least 15 days per month of which at least 8 days are with migraine)
Diagnosis

Tension-type headache, migraine (with or without aura) and cluster headache

- Diagnose tension-type headache, migraine or cluster headache according to the headache features in the table

Migraine with aura

- Suspect aura in people who present with or without headache and with neurological symptoms that:
  - are fully reversible and
  - develop gradually, either alone or in succession, over at least 5 minutes and
  - last for 5–60 minutes
- Diagnose migraine with aura in people who present with or without headache and with one or more of the following typical aura symptoms that meet the criteria (see above):
  - visual symptoms that may be positive (for example, flickering lights, spots or lines) and/or negative (for example, partial loss of vision)
  - sensory symptoms that may be positive (for example, pins and needles) and/or negative (for example, numbness)
  - speech disturbance
- Consider further investigations and/or referral for people who present with or without migraine headache and with any of the following atypical aura symptoms that meet the criteria (see migraine with aura):
  - motor weakness or
  - double vision or
  - visual symptoms affecting only one eye or
  - poor balance or
  - decreased level of consciousness

Menstrual-related migraine

- Suspect menstrual-related migraine in women and girls whose migraine occurs predominantly between 2 days before and 3 days after the start of menstruation in at least 2 out of 3 consecutive menstrual cycles
- Diagnose menstrual-related migraine using a headache diary for at least 2 menstrual cycles

Medication overuse headache

- Be alert to the possibility of medication overuse headache in people whose headache developed or worsened while they were taking the following drugs for 3 months or more:
  - triptans, opioids, ergots or combination analgesic medications on 10 days per month or more
  - paracetamol, aspirin or an NSAID, either alone or in any combination, on 15 days per month or more

Medication

All headache disorders

- Consider using a headache diary:
  - to record the frequency, duration and severity of headaches
  - to monitor the effectiveness of headache interventions
  - as a basis for discussion with the person about their headache disorder and its impact
- Consider further investigations and/or referral if a person diagnosed with a headache disorder develops any of the features
- Do not refer people diagnosed with tension-type headache, migraine, cluster headache or medication overuse headache for neuroimaging solely for reassurance

**Information and support for people with headache disorders**

- Include the following in discussions with the person with a headache disorder:
  - a positive diagnosis, including an explanation of the diagnosis and reassurance that other pathology has been excluded and
  - the options for management and
  - recognition that headache is a valid medical disorder that can have a significant impact on the person and their family or carers
- Give the person written and oral information about headache disorders, including information about support organisations
- Explain the risk of medication overuse headache to people who are using acute treatments for their headache disorder

**Tension-type headache**

**Acute treatment**

- Consider aspirin, paracetamol or an NSAID for the acute treatment of tension-type headache, taking into account the person’s preference, comorbidities and risk of adverse events
- Do not offer opioids for the acute treatment of tension-type headache

**Prophylactic treatment**

- Consider a course of up to 10 sessions of acupuncture over 5–8 weeks for the prophylactic treatment of chronic tension-type headache

**Migraine with or without aura**

**Acute treatment**

- Offer combination therapy with an oral triptan and an NSAID, or an oral triptan and paracetamol, for the acute treatment of migraine, taking into account the person’s preference, comorbidities and risk of adverse events.
- For people who prefer to take only one drug, consider monotherapy with an oral triptan, NSAID, aspirin (900 mg) or paracetamol for the acute treatment of migraine, taking into account the person’s preference, comorbidities and risk of adverse events
- When prescribing a triptan start with the one that has the lowest acquisition cost; if this is consistently ineffective, try one or more alternative triptans
- Consider an anti-emetic in addition to other acute treatment for migraine even in the absence of nausea and vomiting
- Do not offer ergots or opioids for the acute treatment of migraine
- For people in whom oral preparations (or nasal preparations in young people aged 12–17 years) for the acute treatment of migraine are ineffective or not tolerated:
  - offer a non-oral preparation of metoclopramide
Prophylactic treatment

- Discuss the benefits and risks of prophylactic treatment for migraine with the person, taking into account the person's preference, comorbidities, risk of adverse events and the impact of the headache on their quality of life
- Offer topiramate or propranolol for the prophylactic treatment of migraine according to the person's preference, comorbidities and risk of adverse events. Advise women and girls of childbearing potential that topiramate is associated with a risk of fetal malformations and can impair the effectiveness of hormonal contraceptives. Ensure they are offered suitable contraception if needed
- Consider amitriptyline for the prophylactic treatment of migraine according to the person's preference, comorbidities and risk of adverse events
- Do not offer gabapentin for the prophylactic treatment of migraine
- If both topiramate and propranolol are unsuitable or ineffective, consider a course of up to 10 sessions of acupuncture over 5–8 weeks according to the person's preference, comorbidities and risk of adverse events
- For people who are already having treatment with another form of prophylaxis and whose migraine is well controlled, continue the current treatment as required
- Review the need for continuing migraine prophylaxis 6 months after the start of prophylactic treatment
- Advise people with migraine that riboflavin (400 mg once a day) may be effective in reducing migraine frequency and intensity for some people

Combined hormonal contraceptive use by women and girls with migraine

- Do not routinely offer combined hormonal contraceptives for contraception to women and girls who have migraine with aura

Menstrual-related migraine

- For women and girls with predictable menstrual-related migraine that does not respond adequately to standard acute treatment, consider treatment with frovatriptan (2.5 mg twice a day) or zolmitriptan (2.5 mg twice or three times a day) on the days migraine is expected

Treatment of migraine during pregnancy

- Offer pregnant women paracetamol for the acute treatment of migraine. Consider the use of a triptan or an NSAID after discussing the woman’s need for treatment and the risks associated with the use of each medication during pregnancy
- Seek specialist advice if prophylactic treatment for migraine is needed during pregnancy

Cluster headache

Acute treatment

- Discuss the need for neuroimaging for people with a first bout of cluster headache with a GP with a special interest in headache or a neurologist
- Offer oxygen and/or a subcutaneous or nasal triptan for the acute treatment of cluster headache
- When using oxygen for the acute treatment of cluster headache:
  - use 100% oxygen at a flow rate of at least 12 litres per minute with a non-rebreathing mask and a reservoir bag and
  - arrange provision of home and ambulatory oxygen
- When using a subcutaneous or nasal triptan, ensure the person is offered an adequate supply of triptans calculated according to their history of cluster bouts, based on the manufacturer’s maximum daily dose
• Do not offer paracetamol, NSAIDS, opioids, ergots or oral triptans for the acute treatment of cluster headache

**Prophylactic treatment**
• Consider verapamil for prophylactic treatment during a bout of cluster headache. If unfamiliar with its use for cluster headache, seek specialist advice before starting verapamil, including advice on electrocardiogram monitoring
• Seek specialist advice for cluster headache that does not respond to verapamil
• Seek specialist advice if treatment for cluster headache is needed during pregnancy

**Medication overuse headache**
• Explain to people with medication overuse headache that it is treated by withdrawing overused medication
• Advise people to stop taking all overused acute headache medications for at least 1 month and to stop abruptly rather than gradually
• Advise people that headache symptoms are likely to get worse in the short term before they improve and that there may be associated withdrawal symptoms, and provide them with close follow-up and support according to their needs
• Consider prophylactic treatment for the underlying primary headache disorder in addition to withdrawal of overused medication for people with medication overuse headache
• Do not routinely offer inpatient withdrawal for medication overuse headache
• Consider specialist referral and/or inpatient withdrawal of overused medication for people who are using strong opioids, or have relevant comorbidities, or in whom previous repeated attempts at withdrawal of overused medication have been unsuccessful
• Review the diagnosis of medication overuse headache and further management 4–8 weeks after the start of withdrawal of overused medication

**References**

DIABETES MELLITUS

Diabetes can be classified into four clinical categories:

- Type 1 diabetes (due to β-cell destruction, usually leading to absolute insulin deficiency)
- Type 2 diabetes (due to a progressive insulin secretory defect on the background of insulin resistance)
- Other specific types of diabetes due to other causes, e.g., genetic defects in β-cell function, genetic defects in insulin action, diseases of the exocrine pancreas (such as cystic fibrosis), and drug- or chemical-induced (such as in the treatment of HIV/AIDS or after organ transplantation)
- GDM (diabetes diagnosed during pregnancy that is not clearly overt diabetes)

Some patients cannot be clearly classified as type 1 or type 2 diabetic.

Criteria for the diagnosis of diabetes
FPG ≥126 mg/dL (7.0 mmol/L). Fasting is defined as no caloric intake for at least 8 h.*

or

2-h PG≥200 mg/dL (11.1mmol/L) during an OGTT. The test should be performed as described by the WHO, using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water.*

or

A1C ≥6.5% (48 mmol/mol). The test should be performed in a laboratory using a method that is NGSP certified and standardized to the DCCT assay.*

or

In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose ≥200 mg/dL (11.1 mmol/L).

*In the absence of unequivocal hyperglycemia, results should be confirmed by repeat testing.

Type 2 diabetes mellitus consists of an array of dysfunctions characterized by hyperglycemia and resulting from the combination of resistance to insulin action, inadequate insulin secretion, and excessive or inappropriate glucagon secretion.

Signs and symptoms
Many patients with type 2 diabetes are asymptomatic. Clinical manifestations include the following:

- Classic symptoms: Polyuria, polydipsia, polyphagia, and weight loss
- Blurred vision
- Lower-extremity paresthesias
- Yeast infections (eg, balanitis in men)

However, many patients with type 2 diabetes are asymptomatic, and their disease remains undiagnosed for many years.
Complications
• Acute Complications
  a) Hypoglycaemia
  b) Hyperglycaemia
• Chronic Complications
  a) Macrovascular (e.g. cardiovascular, cerebrovascular, peripheral vascular diseases)
  b) Microvascular (e.g. nephropathy, neuropathy and retinopathy)

MANAGEMENT OF TYPE 2 DIABETES MELLITUS

Diabetes management involves lifestyle modification, medications and patient education to encourage selfcare and empowerment.

The overall aims of management are to improve quality of life, reduce complications and prevent premature death. Patient and family members should be counselled by identifying and addressing concerns which may cause distress thus adversely affecting management.

Short term:
• Relief of symptoms and acute complications

Long term:
• Achievement of appropriate glycaemic levels
• Reduction of concurrent risk factors
• Identification and treatment of chronic complications

Metformin
Metformin is the preferred initial agent for monotherapy and is a standard part of combination treatments. Advantages of metformin include the following:
  • Efficacy
  • Absence of weight gain or hypoglycemia
  • Generally low level of side effects
  • High level of patient acceptance
  • Relatively low cost

The dose of metformin is titrated over 1-2 months to at least 2000 mg daily, administered in divided doses (during or after meals to reduce gastrointestinal [GI] side effects). Exercise increases metformin levels and interferes with its glucose-lowering effect.

Dual-drug therapy

If the patient fails to safely achieve or sustain glycemic goals within 2-3 months, another medication should be added. The choice should be guided by patient characteristics (eg, a DPP-4 inhibitor if both postprandial and fasting glucose levels are elevated; a GLP-1 agonist if postprandial glucose levels are strongly elevated; a TZD if the patient has metabolic syndrome and/or nonalcoholic fatty liver disease)
# Treatment of Type 2 Diabetes Mellitus

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*for symptomatic patients, may initially use secretagogue or insulin to rapidly decrease glucose
**exenatide not approved for use with glitazone

## Simplified Scheme for Insulin Therapy

- Bedtime NPH or glargine with pills, start 10-15 units and titrate to fasting glucose <120 mg/dL
- For twice daily insulin, start ~0.5 U/kg with 2/3 in morning and 2/3 as NPH; titrated by SMBG results (meals and insulin administration must be consistent from day to day—give insulin about 20-30 min pre-meal
- For multiple daily injections, start with 50% basal (glargine or ultralente) and pre-prandial rapid acting (20% pre-breakfast, 15% pre-lunch, 15% pre-supper; titrate by SMBG results
- For 2 or more injections daily, add metformin (titrate to 2 g daily) or glitazone (4 mg rosiglitazone or 30 mg pioglitazone) if total insulin dose >1-2 U/kg
**Triple-drug therapy**

If 2 drugs prove unsuccessful after 2-3 months, the next step is triple therapy. The third drug may be an oral agent from a third class of antidiabetic drugs, basal insulin (typically at bedtime), or the injectable drug exenatide. The expense and adverse effect profile of TZDs make their use in an oral triple therapy approach less desirable.

**Dietary Modifications**

For most patients, the best diet is one consisting of the foods that they are currently eating. Attempts to calibrate a precise macronutrient composition of the diet to control diabetes, while time-honored, are generally not supported by the research. Caloric restriction is of first importance. After that, individual preference is reasonable. Modest restriction of saturated fats and simple sugars is also reasonable.

**Weight loss**

Modest weight losses of 5-10% have been associated with significant improvements in cardiovascular disease risk factors (ie, decreased HbA1c levels, reduced blood pressure, increase in HDL cholesterol, decreased plasma triglycerides) in patients with type 2 diabetes mellitus.

**Activity Modifications**

Most patients with type 2 diabetes mellitus can benefit from increased activity. Aerobic exercise improves insulin sensitivity and may improve glycemia markedly in some patients. Structured exercise training of more than 150 minutes per week is associated with greater HbA1c reduction; however, physical activity helps lower HbA1c only when combined with dietary modifications.

**TYPE 1 DIABETES MELLITUS**

Type 1 diabetes is a chronic illness characterized by the body’s inability to produce insulin due to the autoimmune destruction of the beta cells in the pancreas. Onset most often occurs in childhood, but the disease can also develop in adults in their late 30s and early 40s.

**Signs and symptoms**

The classic symptoms of type 1 diabetes are as follows:

- Polyuria
- Polydipsia
- Polyphagia
- Unexplained weight loss

Other symptoms may include fatigue, nausea, and blurred vision. The onset of symptomatic disease may be sudden. It is not unusual for patients with type 1 diabetes to present with diabetic ketoacidosis.

Diagnostic criteria include the following:

- A fasting plasma glucose (FPG) level ≥126 mg/dL (7.0 mmol/L), or
- A 2-hour plasma glucose level ≥200 mg/dL (11.1 mmol/L) during a 75-g oral glucose tolerance test (OGTT), or
- A random plasma glucose ≥200 mg/dL (11.1 mmol/L) in a patient with classic symptoms of hyperglycemia or hyperglycemic crisis.
METFORMIN
Type 2 Diabetes Mellitus

ADULT
Monotherapy or with sulfonylurea
Immediate-release tablet or solution
- Initial: 500 mg PO q12hr or 850 mg PO qDay with meals; increase q2Weeks
- Maintenance: 1500-2550 mg/day PO divided q8-12hr with meal
- No more than 2550 mg/day
Extended-release
- Glucophage XR: 500 mg PO qDay with dinner; titrate by 500 mg/day qWeek; no more than 2000 mg/day
Renal impairment
- Males: Contraindicated if serum creatinine ≥1.5 mg/dL
- Females: Contraindicated if serum creatinine ≥1.4 mg/dL

Not for use in patients >80 years unless normal renal function establishedInitial and maintenance dosing of metformin should be conservative in patients with advanced age due to the potential for decreased renal function in this population

PEDIATRIC
Immediate-release (10-16 years)
- Initial: 500 mg PO q12hr
- Maintenance: Titrate qWeek by 500 mg; no more than 2000 mg/day in divided doses
Immediate-release (≥17 years)
- Initial: 500 mg PO q12hr or 850 mg PO qDay with meals; increase q2Weeks
- Maintenance: 1500-2550 mg/day PO divided q8-12hr with meal
- No more than 2550 mg/day
Extended-release (<17 years)
- Safety and efficacy not established
Extended-release (≥17 years)
- Glucophage XR: 500 mg PO qDay with dinner; titrate by 500 mg/day qWeek; not to exceed 2000 mg/day
- Fortamet: 500-1000 mg PO qDay; titrate by 500 mg/day qWeek; not to exceed 2500 mg/day

Sulfonylureas - Glyburide

ADULT
Regular tablets
- Initial: 2.5-5 mg PO qDay
- Maintenance: 1.25-20 mg PO qDay or q12hr
- Not to exceed 20 mg/day
- Consider administering q12hr for doses >10 mg/day

Renal impairment: If CrCl <50 mL/min; caution advised
**GERIATRIC**
Initial: 1.25 mg/day if nonmicronized tablets
Depending on glucose response, may increase dose by no more than 1.25-2.5 mg (regular)

**Glimepiride**

**ADULT**
Initial: 1-2 mg PO qAM after breakfast or with first meal; may increase dose by 1-2 mg every 1-2 weeks; not to exceed 8 mg/day

**GERIATRIC**
1 mg PO qDay; titrate dose at weekly intervals to avoid hypoglycemia

**Alpha-Glucosidase Inhibitors - Acarbose**
Initially 25 mg PO q8hr, at meals (with first bite)
Can increase to 50 or 100 mg PO q8hr at 4- to 8-wk intervals based on 1 hour postprandial glucose or glycosylated hemoglobin levels, and on tolerance

**Maximum Dose**
<60 kg: 50 mg q8hr
>60 kg: 100 mg q8hr
Type 2 DM, mono treatment or with sulfonylurea

**Thiazolidinediones - Avandia**
Initial 4 mg PO qDay or divided q12hr
If inadequate response after 8-12 weeks, 8 mg PO qDay or divided q12hr
Monitor: ALT at start of treatment, qMonth for 12 months then q3Months thereafter
Coadministration with sulfonylurea: Adjust sulfonylurea dose if hypoglycemia occurs

**Sitagliptin**
100 mg PO qDay
Renal impairment
- CrCl >50 mL/min: Dose adjustment not necessary
- CrCl 30-50 mL/min: 50 mg PO qDay
- CrCl <30 mL/min: 25 mg PO qDay

**Gliclazide**
From 30 to 120 mg once daily.
The recommended starting dose is 30 mg daily

**Rapid-Acting Insulins - Apidra**
Diabetes Mellitus Type I or II
Indicated to improve glycemic control in adults and children with diabetes mellitus
Dosing considerations
- Equipotent to regular human insulin (ie, elicits same glucose lowering effects on unit per unit basis) when administered IV
- Insulin glulisine has a more rapid onset of action and a shorter duration of action than regular human insulin
- Dosage must be individualized; blood glucose monitoring is essential in all patients receiving insulin therapy
- Total daily insulin requirement may vary and is usually between 0.5-1 unit/kg/day
- Insulin requirements may be altered during stress, major illness, or with changes in exercise, meal patterns, or coadministered drugs

4-17 years: May require 0.8-1.2 units/kg/day SC during growth spurts; otherwise use adult dosing (0.5-1 unit/kg/day)

**Humalog**
Rapid-acting human insulin

**Type 1 diabetes mellitus**
- Approximately one third of the total daily insulin requirements SC; rapid-acting or short-acting, premeal insulin should be used to satisfy the remainder of the daily insulin requirements
- Usual daily maintenance range is 0.5-1 unit/kg/day in divided doses; nonobese may require 0.4-0.6 unit/kg/day; obese may require 0.8-1.2 units/kg/day

**Type 2 diabetes mellitus**
- Type 2 diabetes mellitus inadequately controlled with oral medication
- 10 units/day SC (or 0.1-0.2 unit/kg/day) of intermediate- or long-acting insulin given at bedtime generally recommended; as an alternative, rapid-acting formulations, such as insulin lispro, given before meals have also been used; dose must be adjusted carefully.

**Pediatric**
<3 years: Safety and efficacy not established
≥3 years: May require 0.8-1.2 units/kg/day SC during growth spurts; otherwise, use adult dosing (0.5-1 unit/kg/day)

**Regular insulin (Humulin R, Novolin R)**
Regular insulin has a rapid onset of action of 0.5-1 hours and duration of action of 4-6 hours. The peak effects are seen within 2-4 hours. Preparations that contain a mixture of 70% neutral protamine Hagedorn (NPH) and 30% regular human insulin (ie, Novolin 70/30, Humulin 70/30)

**Type 1 Diabetes Mellitus**
Initial: 0.2-0.4 units/kg/day SC divided q8hr or more frequently.
Maintenance: 0.5-1 unit/kg/day SC divided q8hr or more frequently; in insulin-resistant patients (eg, due to obesity), substantially higher daily insulin may be required
Approximately 50-75% of the total daily insulin requirements are given as intermediate- or long-acting insulin administered in 1-2 injections; rapid- or short-acting insulin should be used before or at mealtimes to satisfy the remainder balance of the total daily insulin requirements
**Type 2 Diabetes Mellitus**
Type 2 diabetes inadequately controlled by diet, exercise, or oral medication: Suggested beginning dose of 10 units/day SC (or 0.1-0.2 unit/kg/day) in evening or divided q12hr

Morning
- Give two thirds of daily insulin requirement
- Ratio of regular insulin to NPH insulin 1:2

Evening
- Give one third of daily insulin requirement
- Ratio of regular insulin to NPH insulin 1:1

**Severe Hyperglycemia (Diabetic Ketoacidosis)**
0.1 unit/kg IV bolus (some argue against bolus), THEN, 0.1 unit/kg/hr IV continuous infusion; if serum glucose does not fall by 50 mg/dL in the first hour, check hydration status; if possible, double the insulin hourly until glucose levels fall at the rate of 50-75 mg/dL/hr; decrease infusion to 0.05-0.1 unit/kg/hour when blood sugar reaches 250 mg/dL

**Hyperkalemia**
5-10 units IV insulin in 50 mL D50W (25 g) infused over 15-30 min

**Administration**
Administer within 15 minutes before a meal or immediately after a meal

Making dose adjustments
- Adjust only 1 insulin dose at a time
- Correct hypoglycemia first
- Correct highest blood sugars next
- If all blood sugars are high (within 2.75 mmol/L [50 mg/dL]): Correct morning fasting blood glucose first
- Change insulin doses in small increments: Type 1 diabetes (1-2 unit change); type 2 diabetes (2-3 unit change)

**Pediatric**
**Type 1 Diabetes Mellitus**
Initial: 0.2-0.4 unit/kg/day SC divided q8hr or more frequently.
Maintenance: 0.5-1 unit/kg/day SC divided q8hr or more frequently; in insulin-resistant patients (eg, due to obesity), substantially higher daily insulin may be required

Adolescents: May require up to 1.5 mg/kg/day during puberty

The average total daily insulin requirement for prepubertal children varies from 0.7-1 unit/kg/day but may be much lower

**Insulin NPH (Humulin N, Novolin N)**
Insulin neutral protamine Hagedorn (NPH) has an onset of action of 3-4 hours. The peak effect occurs within 8-14 hours, and its usual duration of action is 16-24 hours.

**Type 1 Diabetes Mellitus**
Suggested guidelines for beginning dose
• Usual daily maintenance range is 0.5-1 unit/kg/day SC in divided doses; nonobese may require 0.4-0.6 unit/kg/day; obese may require 0.8-1.2 units/kg/day

**Type 2 Diabetes Mellitus**

Suggested guidelines for beginning dose: 0.2 unit/kg/day

**Morning**
• Give two thirds of daily insulin SC
• Ratio of regular insulin to NPH insulin 1:2

**Evening**
• Give one third of daily insulin SC
• Ratio of regular insulin to NPH insulin 1:1

**Blood glucose adjustments**
• Adjust only 1 insulin dose at a time
• Correct hypoglycemia first
• Correct highest blood sugars next
• If all blood sugars are high (within 2.75 mmol/L [50 mg/dL]): Correct morning fasting blood glucose first
• Change insulin doses in small increments: Type 1 diabetes (1-2 unit change); type 2 diabetes (2-3 unit change)

**Pediatric**

**Type 1 Diabetes Mellitus**

<12 years: Safety and efficacy not established

>12 years: Suggested dose is 0.5-1 unit/kg/day SC; use adult dosing; usual daily maintenance range in adolescents is ≤1.2 units/kg/day during growth spurts

**Insulin Glargine (Lantus)**

Insulin glargine has an onset of action of 4-8 hours and a duration of action of 24 hours. Peak effects occur within 16-18 hours.

**Type 1 or 2 Diabetes Mellitus**

Lantus (long-acting) and Toujeo (ultra long-acting) are recombinant human insulin analogues indicated to improve glycemic control in adults with type 1 or 2 diabetes mellitus

**Dosing Considerations**

Indicated for once-daily SC administration; exhibits relatively constant glucose-lowering profile over 24 hr

May be administered at any time during the day; should be administered SC once daily at the same time every day

Dose must be individualized based on clinical response; blood glucose monitoring is essential in all patients receiving insulin therapy

Patients adjusting the amount or timing of dosage should do so only under medical supervision with appropriate glucose monitoring

In patients with type 1 diabetes, insulin glargine must be used in regimens with short-acting insulin

Should not be administered IV or via an insulin pump; IV administration of the usual SC dose could result in severe hypoglycemia
Initial dose

- Type 1 diabetes mellitus: Starting dose should be approximately one third of the total daily insulin requirements; short-acting, premeal insulin should be used to satisfy the remaining two thirds of the daily insulin requirements; insulin glargine should be used in combination with a short-acting or rapid-acting insulin; usual daily maintenance range is 0.5-1 unit/kg/day in divided doses; nonobese may require 0.4-0.6 unit/kg/day; obese may require 0.6-1.2 units/kg/day
- Type 2 diabetes mellitus: Starting dose in patients who are not currently treated with insulin is 10 units (or 0.2 unit/kg) once daily
- Dosage should be individualized under the supervision of a healthcare provider in accordance with the needs of the patient and adjusted according to blood glucose measurement

Pediatric

Type 1 Diabetes Mellitus
Lantus is a long-acting human insulin analogue indicated to improve glycemic control in children with type 1 diabetes mellitus; Toujeo is not approved for use in children
<6 years: Safety and efficacy not established
≥6 years: Approximately one third of the total daily insulin requirements SC; rapid-acting or short-acting, premeal insulin should be used to satisfy the remaining two thirds of the daily insulin requirements; usual daily maintenance range in adolescents is ≤1.2 units/kg/day during growth spurts
**DIABETES MELLITUS AND PREGNANCY**

GDM is defined as glucose intolerance of variable degree with onset or first recognition during pregnancy.

**Screening for diabetes mellitus during pregnancy**

**Gestational diabetes**

The following 2-step screening system for gestational diabetes is currently recommended in the United States:

- 50-g, 1-hour glucose challenge test (GCT)
- 100-g, 3-hour oral glucose tolerance test (OGTT) - For patients with an abnormal GCT result

**Type 1 diabetes**

- The disease is typically diagnosed during an episode of hyperglycemia, ketosis, and dehydration
- It is most commonly diagnosed in childhood or adolescence; the disease is rarely diagnosed during pregnancy
- Patients diagnosed during pregnancy most often present with unexpected coma - Early pregnancy may provoke diet and glycemic control instability in patients with occult diabetes

**Type 2 diabetes**

According to the American Diabetes Association’s "Standards of Medical Care in Diabetes--2010," the presence of any one of the following criteria supports the diagnosis of diabetes mellitus:

- Hemoglobin A1C (HbA1C) = 6.5%
- Fasting plasma glucose = >126 mg/dL (7.0 mmol/L)
- A 2-hour plasma glucose level = 200 mg/dL (11.1 mmol/L) during a 75-g OGTT
- A random plasma glucose level = 200 mg/dL (11.1 mmol/l) in a patient with classic symptoms of hyperglycemia or hyperglycemic crisis

In the absence of unequivocal hyperglycemia, a diagnosis based on any of the first 3 criteria should be confirmed by repeat testing on a different day.

**Management**

**Diet**

The goal of dietary therapy is to avoid single large meals and foods with a large percentage of simple carbohydrates. The diet should include foods with complex carbohydrates and cellulose, such as whole grain breads and legumes.

**Insulin**

The goal of insulin therapy during pregnancy is to achieve glucose profiles similar to those of nondiabetic pregnant women. In gestational diabetes, early intervention with insulin or an oral agent is key to achieving a good outcome when diet therapy fails to provide adequate glycemic control.

**Glyburide and metformin**

The efficacy and safety of insulin have made it the standard for treatment of diabetes during pregnancy. Diabetic therapy with the oral agents glyburide and metformin, however, has been gaining in popularity. Trials have shown these 2 drugs to be effective, and no evidence of harm to the fetus has been found, although the potential for long-term adverse effects remains a concern.
**Insulin Therapy**

The goal of insulin therapy during pregnancy is to achieve glucose profiles similar to those of nondiabetic pregnant women. Given that healthy pregnant women maintain their postprandial blood sugar excursions within a relatively narrow range (70-120 mg/dL), reproducing this profile requires meticulous daily attention by both the patient and clinician. Insulins lispro (HUMALOG), aspart (NOVOMIX), regular (HUMALOG), and neutral protamine hagedorn (NPH) are well-studied in pregnancy and regarded as safe and effective. Insulin glargine is less well-studied, and given its long pharmacologic effect, may exacerbate periods of maternal hypoglycemia. Insulin detemir (LEVEMIR) is safe and comparable to NPH insulin in pregnancy.
POSSIBLE COMPLICATIONS OF ABORTION

Bleeding in early pregnancy or history of recent abortion

Rapid assessment

□ Signs of shock?
  (pallor, fast weak pulse, cool moist skin)
  YES
  Probable septic abortion
  Stabilize, start intravenous fluids, give first dose of antibiotics, refer woman urgently to hospital

□ Signs of infection?
  (fever >38 °C, foul-smelling discharge)
  YES
  Possible incomplete abortion
  Start antibiotics, perform MVA or refer for management

□ Signs of incomplete abortion?
  (cervix open, uterus enlarged and soft)
  NO

Possible complete abortion (or other cause of bleeding). Observe closely and refer if no improvement

Note: Ergometrine (0.2 mg) or oxytocin (10 IU) intramuscularly or by slow intravenous infusion is recommended for control of heavy bleeding.
PRELABOUR RUPTURE OF MEMBRANES (Consider immediate referral or hospitalization)

1. Prelabour ROM
   - Signs of infection? (fever > 38 °C, foul-smelling discharge)
     - YES: Give IV/IN antibiotics, Refer urgently to hospital
     - NO
   - Term (>37 weeks)?
     - YES: >18 hours since rupture of membranes?
       - YES: Treat
       - NO
     - NO: Delivery imminent?
       - YES: Treat, monitor and deliver Refer if active labour does not begin within 24 hours
       - NO: Treat and refer
HYPOTHERMIA

Hypothermia describes a state in which the body's mechanism for temperature regulation is overwhelmed in the face of a cold stressor. Hypothermia is classified as accidental or intentional, primary or secondary, and by the degree of hypothermia.

Primary hypothermia is due to environmental exposure, with no underlying medical condition causing disruption of temperature regulation. Secondary hypothermia is low body temperature resulting from a medical illness lowering the temperature set-point.

The body's core temperature is tightly regulated in the "thermoneutral zone" between 36.5°C and 37.5°C, outside of which thermoregulatory responses are usually activated. The body maintains a stable core temperature through balancing heat production and heat loss.

Hypothermia affects virtually all organ systems. Perhaps the most significant effects are seen in the cardiovascular system and the CNS. Hypothermia results in decreased depolarization of cardiac pacemaker cells, causing bradycardia. Since this bradycardia is not vagally mediated, it can be refractory to standard therapies such as atropine. Mean arterial pressure and cardiac output decrease, and an ECG may show characteristic J or Osborne waves.

Osborne (J) waves (V3) in a patient with a rectal core temperature of 26.7°C

Atrial and ventricular arrhythmias can result from hypothermia; asystole and ventricular fibrillation have been noted to begin spontaneously at core temperatures below 25-28°C. Hypothermia progressively depresses the CNS, decreasing CNS metabolism in a linear fashion as the core temperature drops. At core temperatures less than 33°C, brain electrical activity becomes abnormal; between 19°C and 20°C, an electroencephalogram (EEG) may appear consistent with brain death. The term "core temperature after drop" refers to a further decrease in core temperature and associated clinical deterioration of a patient after rewarming has been initiated. The current theory of this documented phenomenon is that as peripheral tissues are warmed, vasodilation allows cooler blood in the extremities to circulate back into the body core.

Very young and elderly persons are at increased risk and may present to the emergency department with symptoms that are not clinically obvious or specific for hypothermia, such as altered mental status.
Clinical Presentation

A patient's companions often note initial symptoms in the field. Symptoms can include mood change, irritability, poor judgment, and lassitude. Companions may note the patient to demonstrate paradoxical undressing (a severely hypothermic person removes clothing in response to prolonged cold stress) or rhythmic or repeated motions such as rocking. Slurred speech and ataxia may mimic a stroke, alcohol intoxication, or high-altitude cerebral edema. Similarly, profound hypothermia may present as coma or cardiac arrest.

A special low-reading thermometer can be used orally or rectally, but it may not reflect a true core temperature. Care should be taken not to rely on a temperature from a rectal thermometer lodged in stool because an inaccurately low core temperature can be recorded. Obtaining a core temperature may help prevent erroneous diagnosis for patients with an altered mental status due to stroke, drug overdose, alcohol intoxication, or mental illness. Standard temperature measuring devices commonly used for triage may lack the capability to report unusually low temperature; obtain a core temperature reading for any patient suspected of being significantly hypothermic.

Mild hypothermia (32-35°C)
Most people shiver vigorously, usually in all extremities.
As the temperature drops below 34°C, a patient may develop altered judgment, amnesia, and dysarthria. Respiratory rate may increase.
At approximately 33°C, ataxia and apathy may be seen. Patients generally are stable hemodynamically and able to compensate for the symptoms.
In this temperature range, the following may also be observed: hyperventilation, tachypnea, tachycardia, and cold diuresis as renal concentrating ability is compromised.

Moderate hypothermia (28-32°C)
Oxygen consumption decreases, and the CNS depresses further; hypventilation, hyporeflexia, decreased renal flow, and paradoxical undressing may be noted.
Most patients with temperatures of 32°C or lower present in stupor.
As the core reaches temperatures of 31°C or below, the body loses its ability to generate heat by shivering.
At 30°C, patients develop a higher risk for arrhythmias. Atrial fibrillation and other atrial and ventricular rhythms become more likely. The pulse continues to slow progressively, and cardiac output is reduced. J wave may be seen on ECG in moderate hypothermia.
Between 28°C and 30°C, pupils may become markedly dilated and minimally responsive to light, a condition that can mimic brain death.

Severe hypothermia (< 28°C)
At 28°C, the body becomes markedly susceptible to ventricular fibrillation and further depression of myocardial contractility.
Below 27°C, 83% of patients are comatose.
Pulmonary edema, oliguria, coma, hypotension, rigidity, apnea, pulselessness, areflexia, unresponsiveness, fixed pupils, and decreased or absent activity on EEG are all seen.
Medical complications from hypothermia often result and necessitate admission to the hospital in moderate and severe hypothermia. Severely hypothermic patients should be admitted to an intensive care unit where their respiratory and cardiac function and temperature may be closely monitored.

Prehospital Care

Prehospital management focuses on preventing further heat loss, rewarming the body core temperature, and avoiding precipitating ventricular fibrillation or another malignant cardiac rhythm. This should be the preeminent concern. Conscious patients can develop ventricular fibrillation suddenly; prehospital workers, particularly those operating in remote search-and-rescue operations, should avoid inadvertent jerky movement of severely hypothermic patients. Patients who develop hypothermia-induced dysrhythmia in the field may be beyond resuscitation. Patients developing hypothermia from cold-water immersion appear to be at high risk of fibrillation; rescuers probably are justified in instructing such patients to minimize motion and to await careful extrication.

RED FLAG - Caution
Both cardiac pacing and atropine are generally ineffective for bradyarrhythmia. Lidocaine is ineffective in preventing hypothermia-induced ventricular dysrhythmias.

Cardiac dysrhythmias begin to develop at a core temperature of 30°C. Ventricular fibrillation susceptibility is greatest below the core temperature of 22°C.

To prevent cardiac dysrhythmia with continued hypothermia, rescuers or paramedics should attempt rewarming in the field. (A notable exception would be isolated frostbite injury in which limb rewarming would preclude self-rescue because of pain.) Note the following:

- Gently place patients in an environment most favorable to reducing further heat loss from evaporation, radiation, conduction, or convection.
- Remove wet clothing, and replace it with dry blankets or sleeping bags.
- Initiate active external rewarming with heat packs (eg, hot water bottles, chemical packs) placed in the axillae, on the groin, and on the abdomen.
- Be aware of the risk of causing body surface burns from exuberant active external rewarming.
- In dire circumstances, rescuers may provide skin-to-skin contact with patients when heat packs are unavailable and such therapy would not delay evacuation.

Ventricular fibrillation in a cold patient is a desperate event. Generally, defibrillation is ineffective at hypothermic core temperatures and when equipment for heroic attempts at resuscitation is unavailable. In such circumstances followed by extended CPR until rescuers can begin active rewarming and perform successful defibrillation.

Emergency Department Care

Patients with respiratory failure should be endotracheally intubated and placed on a mechanical ventilator. Intubation and insertion of vascular catheters should not be delayed but performed gently while closely monitoring cardiac rhythm for ventricular fibrillation.
Determine whether a cold patient is profoundly or mildly hypothermic. Profoundly hypothermic patients present with stupor or cardiac dysrhythmia (regardless of the recorded temperature) and a core temperature of 30°C or lower. Mildly hypothermic patients may be rewarmed in any available manner (eg, warm blankets, removal of cold, wet clothing) since their risk for cardiac dysrhythmia is low. Surface rewarming is adequate in these cases, but it is ineffective in very low body temperatures and carries an additional risk of temperature after drops and shock secondary to peripheral vasodilation.

Remove any wet clothing, and replace it with warm, dry materials. Profound hypothermia is a true emergency, warranting the same resource-intensive resuscitation as myocardial infarction. Direct treatment at maintaining or restoring cardiac perfusion; maximizing oxygenation is indicated for a prolonged period of time until the core temperature is at least 32°C. Do not attempt resuscitation on the patient with a frozen chest where compressions are not possible. Gingerly handle patients identified with profound hypothermia, and take immediate measures to prevent degeneration of cardiac activity into malignant dysrhythmia. Profoundly hypothermic patients who demonstrate cardiac ectopy may be ideal candidates for bretylium, if available. Administer an initial dose of 5 mg/kg IV (repeated at 10 mg/kg, as needed) to prevent ventricular fibrillation. Lidocaine is ineffective for treatment of hypothermia-induced dysrhythmias.

**Initiate warmed, humidified oxygen; provide heated intravenous saline; and place warmed blankets or heat lamps around a hypothermic patient.**

Emergency departments that routinely treat hypothermia can keep blankets and intravenous fluid bags in a shared heater. In urgent situations, intravenous fluids that contain no dextrose or blood can be heated in a microwave oven. Once these simple measures have been applied, consider more difficult rewarming therapies. A patient who is not becoming progressively colder, is conscious, and has a perfusing cardiac rhythm may not require intensive intervention beyond the methods already discussed.

Optimal rewarming techniques depend on a patient’s condition, the capabilities of providers, and the availability of in-hospital care and warming devices. If core body temperature does not respond to warming efforts, underlying infection or endocrine derangements must be considered. **Slow rewarming methods** include IV solutions heated to 45°C (17 kcal/h); heated, humidified oxygen by mask (30 kcal/h or 0.7°C/h); warmed blankets (0.9°C/h); and heated, humidified oxygen via endotracheal tube (1.2°C/h). If intact, a patient's endogenous physiologic mechanisms (other than shivering) provide similar rates of rewarming (30 kcal/h).

**Moderate rewarming methods** provide heat at approximately 3°C/h. Methods include warmed gastric lavage (2.8°C/h), intravenous solutions heated to 65°C (2.9°C/h), and peritoneal lavage with 45°C fluid at 4 L/h (70 kcal/h or 3°C/h).

Initiate CPR for hypothermic patients who deteriorate into ventricular fibrillation. These patients also warrant immediate weight-based defibrillation (2 J/kg), along with prompt administration of high-dose bretylium (10 mg/kg).
RED FLAG - Caution

A reasonable approach is to initiate resuscitation on all hypothermic patients unless a patient presents with a frozen chest or other obvious nonsurvivable injuries. A patient can be warmed aggressively and resuscitated until the core temperature rises above 32°C. At that juncture, if no signs of life are present and the patient is not responding to advanced cardiac life support measures, termination of resuscitation may be indicated.
EMERGENT MANAGEMENT OF FROSTBITE
Frostbite is a cold-related injury characterized by freezing of tissue.

Prehospital Care
Address life-threatening conditions, such as hypothermia or major trauma, first. Replace wet clothing with dry, soft clothing to minimize further heat loss, and initiate rewarming of the affected area as soon as possible. However, do not attempt rewarming if there is a danger of refreezing. Avoid rubbing the affected area with warm hands or snow, as this can cause further injury. If the affected body part is an extremity, wrap it in a bulky dressing or blanket for mechanical protection during transport. Also, remove jewelry from affected extremities. Oral hydration and administration of ibuprofen may improve outcome, if feasible. Avoid alcohol or sedatives, which can enhance heat loss and impair shivering.

Emergency Department Care
Address life-threatening conditions first. Fluid resuscitation, especially in persons with mountain frostbite, enhances blood flow and tissue perfusion. Rapidly rewarm the affected body part, avoiding further trauma. Tetanus prophylaxis should be administered if the patient's vaccination status is not current or unknown.

An appropriate warming technique is the use of a whirlpool bath or tub of water at 38-40°C. Mild antibacterial soap may be added. Avoid warmer temperatures or dry heat because of the risk of thermal injury.

If a tub is not available, use warm, wet packs at the same temperature.

Administer analgesics, such as morphine sulfate, as needed for pain. Ibuprofen should also be administered.

Debride clear blisters to prevent thromboxane-mediated tissue injury. Leave hemorrhagic blisters intact to reduce the risk of infection. Apply topical aloe vera gel every 6 hours with dressing changes.

RED FLAG - Caution
The only indication for early surgical intervention is debridement of blisters or necrotic tissue and fasciotomy in the case of compartment syndrome.
HYPERTHERMIA

Heat illness should be thought of as a spectrum of disease from heat cramps to heatstroke. Medication-related hyperthermic conditions such as malignant hyperthermia, serotonin syndrome, and neuroleptic malignant syndrome (NMS) need to be specifically recognized, as the treatment of these diseases requires adjunctive pharmacotherapy.

Effective thermoregulation, controlled by the hypothalamus, is critical for proper function of the human body, with normal temperature exhibiting diurnal variation between 36-37.5°C.

Hyperthermia is defined as elevated core temperature of greater than 38.5°C.

Patients at risk for heat illness include the following:

- Athletes exercising strenuously in hot climates
- Elderly patients (because of decreased efficacy of thermoregulation, comorbid illness or medications, lack of fans or air conditioning, inappropriate dress)
- Infants and small children (because of high ratio of surface area to weight, inability to control fluid intake)
- Patients with cardiac disease or those taking beta-blockers (because of inability to increase cardiac output sufficiently for vasodilation)
- Patients who are dehydrated because of poor fluid intake, gastroenteritis, and diuretic or alcohol use (Dehydration increases demand on ATPase pumps, which contribute 25-45% of basal metabolic rate.)
- Patients prone to higher endogenous heat production (eg, infection, thyrotoxicosis)
- Patients taking medications that inhibit sweat production or increase heat production (eg, anticholinergics, antidepressants, antihistamines, neuroleptics, zonisamide, sympathomimetics, lithium, alpha- and beta-blockers)

Symptoms of heat exhaustion include the following:

- Normal to slightly elevated core temperature
- Fatigue or malaise
- Orthostatic hypotension, tachycardia
- Clinical signs of dehydration
- Nausea, vomiting, diarrhea (due to splanchnic and renal vasoconstriction)
- Intact mental status
- Responsive to cool environment, fluid and electrolyte replacement

Symptoms of heatstroke include the following:

- Elevated core temperature, usually greater than 40.5°C
- Vague prodrome of weakness, nausea, vomiting, headache
- CNS symptoms including confusion, ataxia, coma, seizures, delirium
- Hot, dry skin
- Hyperdynamic cardiovascular response (high central venous pressure [CVP], low systemic vascular resistance [SVR], tachycardia)
- Elevation of hepatic transaminases, usually in the tens of thousands range
- Coagulopathy
- Rhabdomyolysis and renal failure
**Noninvasive external cooling**

See the list below:

- Ice packs
- Tepid (15°C) water
- Fan
- Cooling blanket
- Ice bath (eg, bathtub, decontamination tub, child’s wading pool)
- Crystalloid intravenous fluids

**Technique**

**Noninvasive external cooling**

*Evaporative cooling*

This is a fast and efficient noninvasive technique for cooling moderate hyperthermia. It was reported in volunteers that it reduces core body temperature by approximately 0.3°C per minute; however, in heatstroke patients, it reduced core body temperature significantly slower: from 0.05°C to 0.09°C per minute.

Remove all of the patient’s clothing.

Mist over patient *constantly*, using spray bottles filled with tepid (15°C) water.

Place large fans to circulate warm room air (ideally 40°C) directed at the patient.

*Whole-body ice packing*

This technique has the advantage of not requiring constant supervision. It can reduce core temperature approximately 0.03°C per minute.

Remove all of the patient’s clothing.

Position the patient on plastic sheets or in a child’s plastic pool.

Cover the patient’s chest and extremities with crushed ice.

Remove the patient once core temperature reaches 39°C.

*Strategic ice packing*

This is a commonly used technique, often used in conjunction with evaporative cooling, that reduces core temperature approximately 0.02-0.03°C per minute.

Remove all of the patient’s clothing.

Place ice packs in the patient’s groin, in the axillae, and around the anterior neck.

Remove ice packs once core temperature reaches 39°C.

**RED FLAG - Caution**

Antipyretics are not effective in treating environmental hyperthermia.

Consider using short-acting benzodiazepines to reduce agitation and shivering during initial cooling as well as to treat hyperthermia due to sympathomimetic ingestion.

Consider a trial of glucose in any patient with altered mental status.

Avoid rapid replacement of free water, as hyponatremia and cerebral edema may develop.
MINOR SURGICAL PROCEDURES
Superficial and Deep Wounds

Acute Wound: is the result of tissue damaged by trauma. This may be deliberate, as in surgical wounds of procedures, or be due to accidents caused by blunt force, projectiles, heat, electricity, chemicals or friction. An acute wound is by definition expected to progress through the phases of normal healing, resulting in the closure of the wound.

Infection

- Wound infection may be defined as the presence of bacteria or other organisms, which lead to a host reaction. A host reaction can present with one or a combination of the following local and systemic clinical indicators:
  - Local indicators
    - Redness (erythema or cellulitis) around the wound
    - Increased amounts of exudate
    - Change in exudates colour
    - Malodour
    - Localised pain
    - Localised heat
    - Delayed or abnormal healing
    - Wound breakdown
  - Systemic indicators
    - Increased systemic temperature
    - General malaise
    - Increased leucocyte count
    - Lymphangitis

Wound cleansing

Requires the application of fluid to clean the wound and optimise the healing environment. The goal of wound cleansing is to:

- Remove visible debris and devitalised tissue
- Remove dressing residue
- Remove excessive or dry crusting exudates

Principles:

- Use Aseptic Technique procedure
- Wound cleansing should not be undertaken to remove 'normal' exudate
- Cleansing should be performed in a way that minimises trauma to the wound
- Wounds are best cleansed with sterile isotonic saline or water
- The less we disturb a wound during dressing changes the lower the interference to healing
- Fluids should be warmed to 37°C to support cellular activity
- Skin and wound cleansers should have a neutral pH and be non-toxic
- Avoid alkaline soap on intact skin as the skin pH is altered, resistance to bacteria decreases
- Avoid delipidising agents as alcohol or acetone as tissue is degraded
- Antiseptics are not routinely recommended for cleansing and should only be used sparingly for infected wounds
Wash Your Hands
• Rub hands with soap and water for 15 to 30 seconds.
• Be sure to wash between fingers and under your nails.
• Rinse well and dry thoroughly.

GET YOUR SUPPLIES
• Have everything you need ready before you begin

Wash/Irrigate the Wound (Sore)
• Always follow your doctor’s instructions for your wound care.
• If you are instructed to use a syringe with a solution, your nurse will show you how to use this device.
• To clean the open wound, pour enough solution to dampen the gauze, and then wipe your wound using circular motions from the center of the wound outward. Be sure to clean at least 1cm beyond the wound margins.
• Make sure you use a new gauze each time you wipe and discard the soiled one in a plastic bag.
• Dry surrounding skin by patting with new gauze.

Are there likely to be other injuries? (eg. head / cervical spine in falls, eye in facial trauma or teeth with mouth injuries).
Is the wound likely to be contaminated by dirt or foreign bodies?
Is there injury to deeper structures (eg. tendons, nerves)? In the face, remember facial nerve, parotid / lacrimal ducts, medial canthus of the eye. If a deep laceration cannot be examined adequately to exclude damage to such structures, general anaesthesia may be required.
Is blood supply impaired? If a flap or area of soft tissue distal to the laceration appears dusky or poorly perfused, the wound requires specialist assessment. Areas with end-arteriolar supply (extremities such as the tip of the nose, finger tips, and ear lobes) require special care. Do not use local anaesthesia with adrenaline on such wounds.

Adequate anaesthesia is necessary for complete examination, cleansing and repair of wounds.
1. Topical anaesthesia
   ALA (adrenaline/lignocaine) 0.1 ml/kg.
   EMLA or AnGel applied to the wound (most effective on limb wounds)
2. Local anaesthesia
   eg. 1% lignocaine with adrenaline slowly infiltrated into the wound, (care should be taken not to use adrenaline on finger tips)
3. Regional block
   eg. infiltrate nerve proximal to injury (ring block digits - use plain lignocaine, no adrenaline)
   Nitrous oxide may facilitate a more comfortable injection.

Tissue adhesive ("Dermabond glue")
• Can be used on wounds which have clean edges, do not require deep sutures and are not under tension.
• Best for wounds of less than 3cm in length with edges easily held together.
- Do not use on mucosal surfaces.
- If gluing the forehead or in the vicinity of the eye, the eye should be padded to avoid any glue dripping into the eye or onto the eyelashes.
- Oppose the edges of the wound and apply very small amount of glue to the surface, holding the edges together for 30 secs.
- Do not allow glue to enter wound itself (non-absorbable - acts as foreign body). Generates heat (may be uncomfortable if applied too thickly).
- Care should be taken not to apply too much tissue glue and to avoid placement over currently bleeding wounds as the polymerisation is exothermic and the patient will notice a heat sensation. The tensile strength of the bond will be reduced also.
- Does not require removal; comes off in 1-2 weeks.

Adhesive strips ("Steristrips")

- May be adequate in simple lacerations which require opposition of slightly separated wound edges.
- They do not remain in place for long periods, and should not be used if there is movement or tension across the wound.
- Prepare skin with tincture of benzoic compound to aid adhesion.
- Place strips with sufficient space between each to allow drainage of fluid from the wound to avoid infection.
- Keep dry for 72 hours.

Surgical

NB: Young or anxious children will require sedation prior to wound repair

Scalp

- Bleeding may be profuse, but usually ceases with firm digital pressure along the margins of wound. Comb hair out of wound (vaseline often helps). It is not usually necessary to shave much hair.
- Close in 2 layers:
  - GALEA 3/0-5/0 Chromic Cat Gut (CCG) or PDS (absorbable)
  - SCALP 4/0-5/0 Nylon (Removal of sutures [ROS] ~7 days)

Forehead

- Minimal debridement. Do not shave eyebrow
- Superficial scratches should be cleaned only and left to epithelialise (± steristrips)
- Sutures 5/0, 6/0 Nylon (ROS 5-7 days) or Fast gut or Vicryl absorbable sutures

Cheek

- Check for fractures (zygoma, blow out of orbit) and involvement of facial nerve and muscle.
- Ophthalmology opinion if hyphaema or "closed eye with swelling".
- Close as for forehead

Eyelids

- If involving the lid margin then refer to Ophthalmology.
- Look for tarsal plate involvement - refer Ophthalmology
- Simple lacerations can be glued or sutured under low tension. Use 6/0 Vicryl or Fast Gut absorbable sutures.
Lips
- Superficial lacerations can be closed in Emergency by person with appropriate experience if the child is cooperative. Otherwise will need GA and Plastic surgical repair.
- NB: Need accurate approximation of vermilion border and skin. Sutures: skin - 6/0 Nylon (ROS 5 days) or Fast Gut (absorbable); mucosa and muscle - 4/0 CCG, Vicryl
- Lacerations of the inner lip rarely need any intervention.
- Lacerations of the gum margin (e.g. degloving injury) need referral to Dental or Facio-Maxillary.

Limbs
- Immobilise area of laceration and joint above and below, following repair eg. plaster slab or sling.
- Upper Limbs: May require arterial tourniquet control. 4/0, 5/0 Nylon. Deep sutures 4/0 PDS.
- Lower Limbs: Debridement important. Do not close if under undue tension especially pretibial,
- ROS 7-10 days.

Trunk
- Debridement can be more generous. Fat layer: 3/0 PDS. Skin: 4/0, 5/0 Nylon. ROS 10-14 days.

Digits & Hand
- Subungual Haematoma:
  - Usually caused by blunt trauma to the finger tip.
  - If < 50% of nail bed - treat with ice and analgesia only
  - If > 50% and significant pain - then burn hole in nail to relieve the pressure
  - Small lacerations of finger tips with skin loss are very common.
  - Areas of skin loss up to 1 cm² are treated with dressings and heal with good return of sensation. Any greater degree of tissue loss should be referred for plastic surgical opinion.
  - Partial-amputation / crush injury.
  - Need to assess the integrity of the nail bed - if damaged needs plastic surgery repair. X-ray to look for fracture of distal phalynx. A fracture implies damage to the nail bed. Discuss management with Plastics.

Palm:
- Be careful in assessing wound especially in very young children as deeper structures (eg nerves and tendons) may be involved. If in doubt consult Plastics.
- Compound injuries (i.e. fracture and laceration) should have antibiotic cover.

Antibiotics
Antibiotics are not indicated for simple lacerations. They are usually given for bites and wounds with extensive tissue damage, or massive contamination, but are secondary in importance to the initial decontamination of the wound. Recommended antibiotics augmentin for 5 days.
BURN

Burn Depth
Burned patients’ survival is related to the following factors: burn size/depth, age, presence of inhalation injury, and patient comorbidity. Depth of burn injury is usually classified according to degrees.

First-degree burns
In first-degree burns, minor epithelial damage of the epidermis exists. Redness, tenderness, and pain are the hallmarks of this injury. Blistering does not occur, and 2-point discrimination remains intact.

Second-degree burns
Superficial partial-thickness and deep partial-thickness burns are the 2 types of second-degree burns. In these burn injuries, some portion of the skin appendages remains viable, allowing epithelial repair of the burn wound without skin grafting. Superficial partial-thickness burn involves the epidermis and superficial (papillary) dermis, often resulting in thin-walled, fluid-filled blisters. These burns appear pink, moist, and soft and are exquisitely tender when touched by a gloved hand. They heal in approximately 2-3 weeks.

Third-degree burns
Third-degree burns are full-thickness burns that destroy both epidermis and dermis. The capillary network of the dermis is completely destroyed. Burned skin has a white or leathery appearance with underlying clotted vessels and is anesthetic. Unless a third-degree burn is small enough to heal by contraction (< 1 cm), skin grafting always is necessary to resurface the injured area. Immersion scalds, flame burns, and chemical and high-voltage electrical injuries cause third-degree burns.

Fourth-degree burns
Fourth-degree burns cause full-thickness destruction of the skin and subcutaneous tissue, with involvement of the underlying fascia, muscle, bone, or other structures. These injuries require extensive debridement and complex reconstruction of specialized tissues and invariably result in prolonged disability. Fourth-degree burns result from prolonged exposure to the usual causes of third-degree burns.

Severity of burn injury depends on
(1) extent, depth, and location of burn injury;
(2) age of patient;
(3) etiologic agents involved;
(4) presence of inhalation injury; and
(5) coexisting injuries or preexisting illnesses.
Prehospital care
Remove the person from the source of the burn without endangering rescue personnel. After extrication, initial care of the burn victim should follow the basic principles of trauma resuscitation (ie, airway, breathing, and circulation [ABCs]), as follows:
- Perform a rapid primary survey and immediately correct any problems found
- Remove any constricting clothing and jewelry
- When ventilatory and circulatory competence have been restored, perform a secondary survey
- Concurrently with airway and circulatory management, make an effort to stop the burning process

Airway and breathing support
- Administer humidified oxygen via a nonrebreathing reservoir mask or endotracheal tube at a rate of 10-12 L/min if signs of inhalation injury are present
- A patient who is not breathing should be intubated and ventilated with 100% oxygen.

Treatment of burn shock
- Elevate the patient’s legs 12 in. off the ground and administer humidified oxygen
- Administer IV fluid if transport time will be longer than 30 min and fluid resuscitation is indicated

Fluid resuscitation
- Begin resuscitation immediately with warmed fluid if possible
- The arm is the preferred site for cannulation
- Catheters may be placed through burned skin if unburned skin is unavailable
- If IV access is not possible, consider interosseous access methods
- Fluid resuscitation is not recommended for children at the scene of the accident
- In adults, administer lactated Ringer solution or normal saline without glucose
- IV flow rates are determined according to the patient’s clinical status

Cooling of burned tissue
- Remove charred clothing
- Immerse the burn wound in cold (1-5°C) water for about 30 minutes if transport cannot be undertaken immediately
- Do not use ice water or apply ice directly to the burn wound
- Local cooling of burns of less than 9% of TBSA can be continued longer than 30 min to relieve pain
- Minor burns can be cooled with running tap water and dressed after more life-threatening issues have been addressed

Transfer to burn center
- If the vehicle with advanced life support capability can transport the burn patient to a specialized burn treatment facility within 30 minutes, the patient should be taken directly to this facility
- If the transport time to the specialized burn treatment facility is longer than 30 minutes, the patient should be transported instead to the nearest ED with advanced life support capability
**Calculation of fluid needs**

All patients with a major burn injury must receive fluid resuscitation that is influenced by the percentage of the TBSA covered by the burn, as well as by the presence of inhalation injury. Adequate resuscitation is evidenced by a normal sensorium, stable vital signs, and a normal urinary output, as follows:

- Children younger than 2 years: 1 mL/lb/h
- Older children: 0.5 mL/lb/h
- Adults: ≥ 30-40 mL/h

**Criteria for transfer to a burn center**

- Any partial-thickness burn larger than 20% of total body surface area (TBSA) in a patient of any age or larger than 10% of TBSA in children younger than 10 years or adults older than 50 years
- Third-degree burns covering more than 5% of TBSA
- Second-degree or third-degree burns involving critical areas (eg, hands, feet, face, perineum, genitalia, or major joints)
- Burns with associated inhalation injury
- Electrical or lightning burns
- Severe burns complicated by coexisting trauma - If traumatic injuries pose a higher risk to the patient than the burn injuries, the patient may have to be sent first to a trauma center
- Preexisting disease that could complicate management of the burn injury
- Chemical burns with threat of cosmetic or functional compromise
- Circumferential burns on the extremities or the chest

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<th>INFECTION</th>
<th>DIAGNOSTIC POINTS</th>
<th>TREATMENT STRATEGIES</th>
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| Burn Impetigo                  | • Loss of epithelium from previously epithelialized surface  
• Not related to local trauma | • Regular cleaning of debris and exudate  
• Topical antistaphylococcal antibiotics  
• Grafting of chronically unstable areas of epithelium |
| Burn-related surgical wound infection | • Infection in surgically created wound which has not yet epithelialized  
• Includes loss of any overlying graft or membrane | • Regular cleaning of debris and exudate  
• Systemic and topical antistaphylococcal antibiotics  
• Grafting of unstable areas of epithelium |
| Burn wound cellulitis          | • Infection occurs in uninjured skin surrounding a wound  
• Signs of local infection progress beyond what is expected form burn related inflammation | • Systemic antibiotics directed against Streptococcus pyogenes  
• Proper treatment of primary wound |
| Invasive burn wound infection  | • Infection occurs in unexcised burn and invades viable underlying tissue  
• Diagnosis may be supported by histologic examination or quantitative cultures | • Systemic antibiotics directed against presumed pathogen.  
• Wound excision, with biologic closure when possible |

**Pain relief**

Immediate, effective analgesia should be provided
Anxiety disorders are common psychiatric disorders. Anxiety disorders appear to be caused by an interaction of biopsychosocial factors, including genetic vulnerability, which interact with situations, stress, or trauma to produce clinically significant syndromes.

**Panic disorder**

Patients with panic disorder frequently present to the ED with chest pain or dyspnea, fearing that they are dying of myocardial infarction. They typically report a spontaneous sudden onset of fear or discomfort, typically reaching a peak within 10 minutes. Attacks are associated with a constellation of systemic symptoms, including the following:

- Palpitations, pounding heart, or accelerated heart rate
- Sweating
- Trembling or shaking
- Shortness of breath or feeling of smothering
- Choking sensation
- Chest pain or discomfort
- Nausea or abdominal distress
- Feeling dizzy, unsteady, lightheaded, or faint
- Derealization (ie, feeling of unreality) or depersonalization (ie, being detached from oneself)
- Fear of losing control or going crazy
- Fear of dying
- Paresthesias (ie, numbness or tingling sensations)
- Chills or hot flashes

During the episode, patients have the urge to flee or escape and have a sense of impending doom (as though they are dying from a heart attack or suffocation). Other symptoms may include headache, cold hands, diarrhea, insomnia, fatigue, intrusive thoughts, and ruminations.

**Generalized anxiety disorder**

This disorder is characterized by excessive anxiety and worry. Worrying is difficult to control. Anxiety and worry are associated with at least 3 of the following symptoms:

- Restlessness or feeling keyed-up or on edge
- Being easily fatigued
- Difficulty concentrating or mind going blank
- Irritability
- Muscle tension
- Sleep disturbance
- Although not a diagnostic feature, suicidal ideation and completed suicide have been associated with generalized anxiety disorder

**Social anxiety disorder (social phobia)**

A person with social phobia will typically report a marked and persistent fear of social or performance situations, to the extent that his or her ability to function at work or in school is impaired. Exposure to social or performance situation always produces anxiety. Social or performance situations are avoided...
or endured with intense anxiety. Avoidance behavior, anticipation, or distress in the feared social or performance setting produces significant impairment in functioning.

**Agoraphobia**
Inquire about any intense anxiety reactions that occur when the patient is exposed to specific situations such as heights, animals, small spaces, or storms. Other areas of inquiry should include fear of being trapped without escape (eg, being outside the home and alone; in a crowd of unfamiliar people; on a bridge, in a tunnel, in a moving vehicle).

**Specific (simple) phobia**
If specific phobias are suspected, specific questions need to be asked about irrational and out of proportion fear to specific situations (eg, animals, insects, blood, needles, flying, heights). Phobias can be disabling and cause severe emotional distress, leading to other anxiety disorders, depression, suicidal ideation, and substance-related disorders, especially alcohol abuse or dependence.

**Treatment & Management**
Treatment usually consists of a combination of pharmacotherapy and/or psychotherapy.

The outcome of treatment is determined by several factors, including the following:
- Severity of diagnosis
- Level of functioning prior to onset of symptoms
- Degree of motivation for treatment
- Level of support (eg, family, friends, work, school)
- Ability to comply with medication and/or psychotherapeutic regimen

**Acute anxiety**
Patients with significant discomfort from their anxiety can benefit from emergency anxiolytic treatment, primarily with a benzodiazepine. In addition to ED treatment, patients in an acute anxious state of such severity that they pose a danger to themselves or to others should have a psychiatric consultation.

Indications for hospitalization include the following
- Severe functional impairment (cannot meet own daily needs)
- Suicide or homicide risk
- Social skills deficits (eg, the person is so preoccupied that he or she is unaware that his or her actions and behaviors have the potential to provoke others to cause harm)

**Alprazolam (Xanax)**
For management of anxiety attacks. Binds receptors at several sites within the central nervous system, including the limbic system and reticular formation.

**Anxiety**
0.25-0.5 mg PO q6-8hr; titrate to effect q3-4Days; not to exceed 4 mg/day

**Panic Disorder**
- 0.5 mg PO q8hr; may increase q3-4Days by ≤1 mg/day
- Average dose: 5-6 mg/day PO
**Anxiety Associated With Depression**

1-4 mg/day PO divided q8hr

**Diazepam**

Modulates postsynaptic effects of GABA-A transmission, resulting in an increase in presynaptic inhibition. Appears to act on part of the limbic system, the thalamus, and hypothalamus, to induce a calming effect. Also has been found to be an effective adjunct for the relief of skeletal muscle spasm caused by upper motor neuron disorders.

**Anxiety**

2-10 mg PO q6-12hr, OR 2-10 mg IV/IM q6-12hr; no more than 30 mg/8 hours
INSOMNIA

Insomnia is defined as repeated difficulty with sleep initiation, maintenance, consolidation, or quality that occurs despite adequate time and opportunity for sleep and that results in some form of daytime impairment.

A social history should be obtained, addressing the following:

- Transient or short-term insomnia: Recent situational stresses
- Chronic insomnia: Past stresses or medical illnesses
- Use of tobacco, caffeinated products, alcohol, and illegal drugs

The medication history should be reviewed, focusing on agents that commonly cause insomnia, such as the following:

- Beta blockers
- Clonidine
- Theophylline (acutely)
- Certain antidepressants (eg, protriptyline, fluoxetine)
- Decongestants
- Stimulants
- Over-the-counter and herbal remedies

Sedative-hypnotics are the most commonly prescribed drugs for insomnia. Though not usually curative, they can provide symptomatic relief when used alone or adjunctively. Such agents include the following:

- Short- and intermediate-acting benzodiazepines (eg, triazolam, temazepam)
- Zolpidem

The following general precautions should be taken when sedative-hypnotics are used:

- Start with a low dose, and maintain at the lowest effective dose
- Avoid continued nightly use; encourage patients to use them only when truly necessary
- Avoid using for more than 2-4 weeks if possible
- Counsel patients to allow for at least 8 hours of sleep
- Be aware that impairment can be present despite a feeling of being fully awake
- When the problem is falling asleep, prefer hypnotics with a rapid onset of action (zolpidem)
- When the problem is staying asleep, consider a hypnotic with a slower rate of elimination (eg, temazepam)
- If the patient is depressed, consider an antidepressant with sedative properties (eg, trazodone, mirtazapine, amitriptyline) in preference to a hypnotic
- Never use hypnotics with alcohol
- Avoid using in pregnant patients
- Avoid using benzodiazepines in patients with known or possible sleep apnea
- Use lower doses in elderly patients
AGITATED PATIENT

Agitation is defined as an abnormal increase in psychological or motor hyperactivity.

All emergency medicine personnel need to be aware of the signs and symptoms of potential violence. Any patient can become violent, but patients with organic disorders such as dementia, delirium, and chemical intoxication have a higher incidence of violence, as do functional disorders such as mania and schizophrenia. The following is a list of “early warning signs” of violence:

1. Patient exhibits or threatens violence.
2. Patient makes ED staff anxious or fearful.
3. Behavior alternates between shouting and dozing, and between cooperation and belligerence.
4. Patient expresses fear of losing control.
5. Patient is uncooperative, hostile, agitated and unable to sit still.
6. Patient is intoxicated with alcohol or other chemicals or withdrawing from drugs.
7. Patient has a past history of violence. He is a “Frequent flyer” known to police or ED staff for violence or impulsive behavior.
8. Patient has tense, rigid posture, is easily startled and suspicious.

Primary Survey
- Appearance
- Current medical status
- Psychiatric History (history of violence)
- Current medication
- Oriented (time, place, person)

Physiological indications for impending aggression
- Flushing of skin
- Dilated pupils
- Shallow rapid respirations
- Excessive perspiration

Many patients who become violent are fortunately not violent from the moment they come through the door. This allows the staff to prepare but it also allows for alternative measures to restraints to be employed. A doctrine called “the least restrictive method of restraint” should be employed when dealing with the potentially violent patient. This means that a patient should be provided alternatives to correct inappropriate behavior in order to maintain a good working doctor/patient relationship and to maintain the dignity of the patient.

These methods of “talking a patient down” include:
1. Avoid eye contact with patient.
2. Do not block exits and leave the door to the exam room open.
3. Maintain a good distance from potentially violent patient; do not invade the patient’s “space”.
4. Adopt passive, non-confrontational posture and attitude, and allow patient to ventilate his feelings.
5. Treat the patient as you expect him to behave.
6. Offer the patient food or drink.
7. Do not make challenging, provocative, or belligerent remarks.
8. If the patient acts out, tell the patient directly “your behavior is frightening others and we cannot allow such behavior”.

9. Do not turn your back on potentially violent patient. 10. Never underestimate the potential for violence.

Once the use of less restrictive methods of modifying the patient’s behavior, such as “talking them down” have failed then the use of restraints may become necessary. The doctrine of “the least restrictive method of restraint” applies here as well. Providing the patient with options for modifying his/her behavior allows a patient/doctor relationship to be maintained. A patient may choose one method of restraint over another. If it is not possible to engage the patient, and have him/her participate in their treatment, and the situation presents a risk of injury to the patient or staff then it becomes necessary to use force to restrain the patient. This should be done with a team approach that is well rehearsed, in which all the participants understand their role. This can be done in the following manner: Placing 4-5 security officers in clear view of the patient, but 10-15 feet distant. The ED physician should then notify the patient in a firm, but not threatening voice, that the continuation of the patient’s uncontrolled and disruptive behavior will not be allowed, and that the patient will be restrained by “the team” unless he lies down now on the ED cart and cooperates with the medical staff.

Physical restraints should be used if, in the ED physician’s medical opinion, the patient is a danger to themselves, other patients or the staff. Also, the ED physician can use “good faith” restraints to allow evaluation and treatment of an uncooperative incompetent patient (such as a patient with dementia). If physical restraints are to be used, they should be used properly and restraints must be adequate.

The order in which restraints are used does not need to be physical and then chemical. If the patient is willing to take medication prior to the use of physical restraints then give him/her the medication. Often patient just want to get back on their medications in order to feel better (stop the voices) and so will take medication with better cooperation than physical restraints. Chemical restraints can also be used after physical restraints if the patient continues to struggle against the restraints and shows a persistence of uncontrolled behavior.

**Sedation:**

- Remember you are generally treating the undifferentiated patient, with limited access to past medical history.
- These patients are generally reluctant to take oral medications, IV access needs to be obtained, or IM or SL sedation can be given while attempting IV canulation,
- Once you choose to start chemical sedation, you have full responsibility to maintain the patient’s airway, breathing, circulation, provide bladder care, hydration, and general nursing care to that patient.
- Benzodiazepines are preferred in the ED, as have prompt onset of action, and a good safety profile.
- Antipsychotic’s have a role when patient is not responding to benzodiazepines, and as an adjunct to the benzo’s to achieve sedation.

**Midazolam:**

- Start with 2.5-5mg IV or IM increments and work upwards
- Short acting medication that provides rapid sedation, in titrated doses
- Maximum effect in 10mins, and last up to 2 hours.
Diazepam:
- Start with 5-10 PO or IV increments and work upward
- Longer acting than Midazolam, works well for managing withdrawal symptoms
- IV administration causes short lived stinging sensation, do not dilute dose to prevent this

Lorazepam:
- 1-2mg PO
- Patient needs to be willy to take oral medication
- Provides sedation up to 4-6 hours

Antipsychotics:

Olanzapine:
- Start with 5-10mg PO or SL, or 10mg IM
- Newer atypical antipsychotic
- Risk of hypotension after IM injection
- Maximum dose 30mg in 24 hour period

Haloperidol:
- 2.5-10mg IV or IM
- Older conventional antipsychotic
- Avoid in patients with QT prolongation as increases risk of torsades de points
- Risk of dystonic drug reaction

Risperidone:
- 0.25-2mg PO/SL
- Newer atypical antipsychotic
- Works very well in elderly, and combative dementia patients.
- Orthostatic hypotension common early in treatment

Complications of sedation and restraining patients:
- Respiratory depression and pulmonary aspiration
- Sudden cardiac death/Excited delirium
- Hypotension
- Deep venous thrombosis & pulmonary embolus
- Rhabdomyolysis
- Dystonic reactions
- Neuroleptic malignant syndrome
- Anticholinergic effects
- Delirium
- Lactic acidosis
- Lowered seizure threshold
- Special problems in the elderly
DELIRIUM

Delirium is defined as a transient, usually reversible, cause of cerebral dysfunction and manifests clinically with a wide range of neuropsychiatric abnormalities. It can occur at any age, but it occurs more commonly in patients who are elderly and have compromised mental status.

Signs and symptoms
The main symptoms of delirium include the following:
- Clouding of consciousness
- Difficulty maintaining or shifting attention
- Disorientation
- Illusions
- Hallucinations
- Fluctuating levels of consciousness
- Symptoms tend to fluctuate over the course of the day, with some improvement in the daytime and maximum disturbance at night. Reversal of the sleep-wake cycle is common.

Neurological symptoms may include the following:
- Dysphasia
- Dysarthria
- Tremor
- Asterixis in hepatic encephalopathy and uremia
- Motor abnormalities

Diagnosis
The diagnosis of delirium is clinical

Diagnostic criteria for delirium is as follows:
- Disturbance in attention (ie, reduced ability to direct, focus, sustain, and shift attention) and awareness.
- Change in cognition (eg, memory deficit, disorientation, language disturbance, perceptual disturbance) that is not better accounted for by a preexisting, established, or evolving dementia.
- The disturbance develops over a short period (usually hours to days) and tends to fluctuate during the course of the day.
- There is evidence from the history, physical examination, or laboratory findings that the disturbance is caused by a direct physiologic consequence of a general medical condition, an intoxicating substance, medication use, or more than one cause.

Management
The goal of treatment is to determine the cause of the delirium and stop or reverse it. Components of delirium management include supportive therapy and pharmacologic management.
Psychiatric consultation may be indicated for management of behavioral problems such as agitation or aggressive behavior. Mental confusion requires hospitalisation.
**Lorazepam**

Preferable because it is short acting and has no active metabolites. In addition, can be used in both IM and IV forms. When patient needs to be sedated for longer than 24 h, this medication is excellent. Commonly used prophylactically to prevent delirium tremens.

Initial: 2-3 mg PO q8-12hr PRN; not to exceed 10 mg/day

Maintenance: 2-6 mg/day PO divided q8-12hr
POST-TRAUMATIC STRESS DISORDER

Posttraumatic stress disorder (PTSD) is a mental illness that can develop after a person is exposed to one or more traumatic events, such as sexual assault, warfare, traffic collisions, terrorism or other threats on a person's life.

Most people who have experienced a traumatizing event will not develop PTSD. People who experience interpersonal trauma (e.g., sexual assault, child abuse) are more likely to develop PTSD, as opposed to people who experience non-assault based trauma such as accidents, natural disasters and witnessing trauma. Children are less likely to develop PTSD after trauma than adults, especially if they are under ten years of age.

In the typical case, the individual with PTSD persistently avoids trauma-related thoughts and emotions, and discussion of the traumatic event, and may even have amnesia of the event. However, the event is commonly relived by the individual through intrusive, recurrent recollections, flashbacks, and nightmares.

The diagnostic criteria for PTSD, stipulated in the International Statistical Classification of Diseases and Related Health Problems, may be summarized as:

- Exposure to a stressful event or situation (either short or long lasting) of exceptionally threatening or catastrophic nature, which is likely to cause pervasive distress in almost anyone.
- Persistent remembering, or "reliving" the stressor by intrusive flash backs, vivid memories, recurring dreams, or by experiencing distress when exposed to circumstances resembling or associated with the stressor.
- Actual or preferred avoidance of circumstances resembling or associated with the stressor (not present before exposure to the stressor).
- Either (1) or (2):
  1. Inability to recall, either partially or completely, some important aspects of the period of exposure to the stressor
  2. Persistent symptoms of increased psychological sensitivity and arousal (not present before exposure to the stressor) shown by any two of the following:
     - difficulty in falling or staying asleep
     - irritability or outbursts of anger
     - difficulty in concentrating
     - hyper-vigilance
     - Exaggerated startle response.

PTSD causes biochemical changes in the brain and body, that differ from other psychiatric disorders such as major depression.

Psychotherapy is the "gold standard" of treatment for PTSD.
DEPRESSION

Depression is a common mental disorder, characterized by sadness, loss of interest or pleasure, feelings of guilt or low self-worth, disturbed sleep or appetite, feelings of tiredness and poor concentration. Major depressive disorder has significant potential morbidity and mortality, contributing to suicide.

Signs and symptoms
Most patients with major depressive disorder present with a normal appearance. In patients with more severe symptoms, a decline in grooming and hygiene may be observed, as well as a change in weight. Patients may also show the following:
- Psychomotor retardation
- Flattening or loss of reactivity in the patient’s affect (ie, emotional expression)
- Psychomotor agitation or restlessness

Major depressive disorder
Among the criteria for a major depressive disorder, at least 5 of the following symptoms have to have been present during the same 2-week period (and at least 1 of the symptoms must be diminished interest/pleasure or depressed mood):
- Depressed mood: For children and adolescents, this can also be an irritable mood
- Diminished interest or loss of pleasure in almost all activities (anhedonia)
- Significant weight change or appetite disturbance: For children, this can be failure to achieve expected weight gain
- Sleep disturbance (insomnia or hypersomnia)
- Psychomotor agitation or retardation
- Fatigue or loss of energy
- Feelings of worthlessness
- Diminished ability to think or concentrate; indecisiveness
- Recurrent thoughts of death, recurrent suicidal ideation without a specific plan, or a suicide attempt or specific plan for committing suicide
Self-report screening instruments for depression include the following:

<table>
<thead>
<tr>
<th>Over the last 2 weeks, how often have you been bothered by any of the following problems?</th>
<th>Not at all</th>
<th>Several Days</th>
<th>More than half the days</th>
<th>Nearly every day</th>
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<tr>
<td>1. Little interest or pleasure in doing things</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2. Feeling down, depressed, or hopeless</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<td>3. Trouble falling or staying asleep, or sleeping too much</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<td>4. Feeling tired or having little energy</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>5. Poor appetite or overeating</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>6. Feeling bad about yourself – or that you are a failure or have let yourself or your family down</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>7. Trouble concentrating on things, such as reading the newspaper or watching television</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>8. Moving or speaking so slowly that other people could have noticed. Or the opposite – being so fidgety or restless that you have been moving around a lot more than usual</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>9. Thoughts that you would be better off dead, or of hurting yourself in some way</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

**Total Score:** 1-4 Minimal depression; 5-9 Mild depression; 10-14 Moderate depression; 15-19 Moderately severe depression; 20-27 Severe depression

**Management**
In all patient populations, the combination of medication and psychotherapy generally provides. Psychiatric consultation may be indicated for management.
Anaphylaxis is a severe, life-threatening, generalised or systemic hypersensitivity reaction. This is characterised by rapidly developing life-threatening airway and/or breathing and/or circulation problems usually associated with skin and mucosal changes.

Time course for fatal anaphylactic reactions
When anaphylaxis is fatal, death usually occurs very soon after contact with the trigger. From a case series, fatal food reactions cause respiratory arrest typically after 30–35 minutes; insect stings cause collapse from shock after 10–15 minutes; and deaths caused by intravenous medication occur most commonly within five minutes. Death never occurred more than six hours after contact with the trigger.

A diagnosis of anaphylactic reaction is likely if a patient who is exposed to a trigger (allergen) develops a sudden illness (usually within minutes of exposure) with rapidly progressing skin changes and life-threatening airway and/or breathing and/or circulation problems. The reaction is usually unexpected.

Anaphylaxis is likely when all of the following 3 criteria are met:
• Sudden onset and rapid progression of symptoms
• Life-threatening Airway and/or Breathing and/or Circulation problems
• Skin and/or mucosal changes (flushing, urticaria, angioedema)

The following supports the diagnosis:
• Exposure to a known allergen for the patient

Remember:
• Skin or mucosal changes alone are not a sign of an anaphylactic reaction
• Skin and mucosal changes can be subtle or absent in up to 20% of reactions (some patients can have only a decrease in blood pressure, i.e., a Circulation problem)
• There can also be gastrointestinal symptoms (e.g. vomiting, abdominal pain, incontinence)

Life-threatening Airway and/or Breathing and/or Circulation problems
Patients can have either an A or B or C problem or any combination. Use the ABCDE approach to recognise these.

Airway problems:
• Airway swelling, e.g., throat and tongue swelling (pharyngeal/laryngeal oedema). The patient has difficulty in breathing and swallowing and feels that the throat is closing up.
• Hoarse voice.
• Stridor – this is a high-pitched inspiratory noise caused by upper airway obstruction.

Breathing problems:
• Shortness of breath – increased respiratory rate.
• Wheeze.
• Patient becoming tired.
• Confusion caused by hypoxia.
• Cyanosis (appears blue) – this is usually a late sign.
• Respiratory arrest.
Circulation problems:
• Signs of shock – pale, clammy.
• Increased pulse rate (tachycardia).
• Low blood pressure (hypotension) – feeling faint (dizziness), collapse.
• Decreased conscious level or loss of consciousness.
• Anaphylaxis can cause myocardial ischaemia and electrocardiograph (ECG) changes even in individuals with normal coronary arteries.
• Cardiac arrest.

Treatment of an anaphylactic reaction
The specific treatment of an anaphylactic reaction depends on:
1. Location.
2. Training and skills of rescuers.
3. Number of responders.
4. Equipment and drugs available.

Location
Treating a patient with anaphylaxis in the community will not be the same as in an acute hospital. Out of hospital, an ambulance must be called early and the patient transported to an emergency department.

Training of rescuers
All clinical staff should be able to call for help and initiate treatment in a patient with an anaphylactic reaction. Rescuers must use the skills for which they are trained. Clinical staff who give parenteral medications should have initial training and regular updates in dealing with anaphylactic reactions. The Health Protection Agency recommends that staff who give immunisations should have annual updates.34

Number of responders
The single responder must always ensure that help is coming. If there are several rescuers, several actions can be undertaken simultaneously.

Equipment and drugs available
Resuscitation equipment and drugs to help with the rapid resuscitation of a patient with an anaphylactic reaction must be immediately available in all clinical settings. Clinical staff should be familiar with the equipment and drugs they have available and should check them regularly.

Patients having an anaphylactic reaction in any setting should expect the following as a minimum:
1. Recognition that they are seriously unwell.
2. An early call for help.
3. Initial assessment and treatments based on an ABCDE approach.
4. Adrenaline therapy if indicated.
5. Investigation and follow-up by an allergy specialist.

Patient positioning
All patients should be placed in a comfortable position. The following factors should be considered:
• Patients with Airway and Breathing problems may prefer to sit up as this will make breathing easier.
• Lying flat with or without leg elevation is helpful for patients with a low blood pressure (Circulation problem). If the patient feels faint, do not sit or stand them up - this can cause cardiac arrest.
• Patients who are breathing and unconscious should be placed on their side (recovery position).
• Pregnant patients should lie on their left side to prevent caval compression

**Remove the trigger if possible**
Removing the trigger for an anaphylactic reaction is not always possible.
• Stop any drug suspected of causing an anaphylactic reaction (e.g., stop intravenous infusion of a gelatin solution or antibiotic).
• Remove the stinger after a bee sting. Early removal is more important than the method of removal.
• After food-induced anaphylaxis, attempts to make the patient vomit are not recommended.
• Do not delay definitive treatment if removing the trigger is not feasible.

**Cardiorespiratory arrest following an anaphylactic reaction**
Start cardiopulmonary resuscitation (CPR) immediately and follow current guidelines. Rescuers should ensure that help is on its way as early advanced life support (ALS) is essential. Use doses of adrenaline recommended in the ALS guidelines. The intramuscular route for adrenaline is not recommended after cardiac arrest has occurred.
ANAPHYLAXIS ALGORITHM

Anaphylactic reaction?

Airway, Breathing, Circulation, Disability, Exposure

Diagnosis - look for:
- Acute onset of illness
- Life-threatening Airway and/or Breathing and/or Circulation problems
- And usually skin changes

Call for help
- Lie patient flat
- Raise patient’s legs

Adrenaline

When skills and equipment available:
- Establish airway
- High flow oxygen
- IV fluid challenge
- Chlorphenamine
- Hydrocortisone

Monitor:
- Pulse oximetry
- ECG
- Blood pressure

1 Life-threatening problems:
Airway: swelling, hoarseness, stridor
Breathing: rapid breathing, wheeze, fatigue, cyanosis, SpO₂ < 92%, confusion
Circulation: pale, clammy, low blood pressure, faintness, drowsy/coma

2 Adrenaline (give IM unless experienced with IV adrenaline)
IM doses of 1:1000 adrenaline (repeat after 5 min if no better)
- Adult: 500 micrograms IM (0.5 mL)
- Child more than 12 years: 500 micrograms IM (0.5 mL)
- Child 6 - 12 years: 300 micrograms IM (0.3 mL)
- Child less than 6 years: 150 micrograms IM (0.15 mL)
Adrenaline IV to be given only by experienced specialists
Titrated: Adults 50 micrograms; Children 1 microgram/kg

3 IV fluid challenge:
Adult: 500 – 1000 mL
Child: crystalloid 20 mL/kg
Stop IV colloid if this might be the cause of anaphylaxis

4 Chlorphenamine
(IM or slow IV)
- Adult or child more than 12 years: 10 mg
- Child 6 - 12 years: 5 mg
- Child 6 months to 6 years: 2.5 mg
- Child less than 6 months: 250 micrograms/kg

5 Hydrocortisone
(IM or slow IV)
- Adult or child more than 12 years: 200 mg
- Child 6 - 12 years: 100 mg
- Child 6 months to 6 years: 50 mg
- Child less than 6 months: 25 mg
IM route is the best for most individuals who have to give adrenaline to treat an anaphylactic reaction. Monitor the patient as soon as possible (pulse, blood pressure, ECG, pulse oximetry). This will help monitor the response to adrenaline. The IM route has several benefits:
- There is a greater margin of safety.
- It does not require intravenous access.
- The IM route is easier to learn.

The best site for IM injection is the anterolateral aspect of the middle third of the thigh. The needle used for injection needs to be sufficiently long to ensure that the adrenaline is injected into muscle.

**IM (intramuscular) route for adrenaline is the route of choice for most healthcare providers. There is a much greater risk of causing harmful side effects by inappropriate dosage or misdiagnosis of anaphylaxis when using IV adrenaline. This is why the IM route is recommended for most healthcare providers.**

This section on IV adrenaline only applies to those experienced in the use and titration of vasopressors in their normal clinical practice.

**FOR SPECIALIST USE ONLY**

Ensure patient is monitored

**Adrenaline IV bolus dose – adult:**

Titrate IV adrenaline using 50 microgram boluses according to response. If repeated adrenaline doses are needed, start an IV adrenaline infusion. The pre-filled 10 mL syringe of 1:10,000 adrenaline contains 100 micrograms/mL. A dose of 50 micrograms is 0.5 mL, which is the smallest dose that can be given accurately.

**Do not give the undiluted 1:1000 adrenaline concentration IV.**

**Adrenaline IV bolus dose – children:**

IM adrenaline is the preferred route for children having an anaphylactic reaction. The IV route is recommended only in specialist paediatric settings by those familiar with its use (e.g., paediatric anaesthetists, paediatric emergency physicians, paediatric intensivists) and if the patient is monitored and IV access is already available. There is no evidence on which to base a dose recommendation – the dose is titrated according to response. A child may respond to a dose as small as 1 microgram/kg. This requires very careful dilution and checking to prevent dose errors.

**Adrenaline infusion**

An infusion of adrenaline with the rate titrated according to response in the presence of continued haemodynamic monitoring is an effective way of giving adrenaline during anaphylaxis. Use local guidelines for the preparation and infusion of adrenaline.

**Auto-injectors** are often given to patients at risk of anaphylaxis for their own use. At the time of writing, there are only two doses of adrenaline auto-injector commonly available: 0.15 and 0.3 mg. The more appropriate dose for an auto-injector should be prescribed for individual patients by allergy specialists. Healthcare professionals should be familiar with the use of the most commonly available auto-injector devices. The dose recommendations for adrenaline in this guideline are intended for healthcare providers treating an anaphylactic reaction.

**Oxygen (give as soon as available)**

Initially, give the highest concentration of oxygen possible using a mask with an oxygen reservoir. Ensure high flow oxygen (usually greater than 10 litres min⁻¹) to prevent collapse of the reservoir during inspiration. If the patient’s trachea is intubated, ventilate the lungs with high concentration oxygen using a self-inflating bag.
**Fluids (give as soon as available)**
Large volumes of fluid may leak from the patient’s circulation during an anaphylactic reaction. There will also be vasodilation, a low blood pressure and signs of shock. If there is intravenous access, infuse intravenous fluids immediately. Give a rapid IV fluid challenge (20 mL/kg in a child or 500-1000 mL in an adult) and monitor the response; give further doses as necessary. There is no evidence to support the use of colloids over crystalloids in this setting. Consider colloid infusion as a cause in a patient receiving a colloid at the time of onset of an anaphylactic reaction and stop the infusion. 0.9% saline are suitable fluids for initial resuscitation. A large volume of fluid may be needed.

**Antihistamines (after initial resuscitation)**
Antihistamines are a second line treatment for an anaphylactic reaction. The evidence to support their use is weak, but there are logical reasons for them. Antihistamines (H1-antihistamine) may help counter histamine-mediated vasodilation and bronchoconstriction. They may not help in reactions depending in part on other mediators but they have the virtue of safety. Used alone, they are unlikely to be lifesaving in a true anaphylactic reaction. Inject chlorphenamine slowly intravenously or intramuscularly.

*The dose of chlorphenamine depends on age:*
- >12 years and adults: 10 mg IM or IV slowly
- >6 – 12 years: 5 mg IM or IV slowly
- >6 months – 6 years: 2.5 mg IM or IV slowly
- <6 months: 250 micrograms/kg IM or IV slowly
There is little evidence to support the routine use of an H2-antihistamine (e.g., ranitidine, cimetidine) for the initial treatment of an anaphylactic reaction.

**Steroids (give after initial resuscitation)**
Corticosteroids may help prevent or shorten protracted reactions. In asthma, early corticosteroid treatment is beneficial in adults and children. There is little evidence on which to base the optimum dose of hydrocortisone in anaphylaxis. In hospital patients with asthma, higher doses of hydrocortisone do not seem to be better than smaller doses. Inject hydrocortisone slowly intravenously or intramuscularly, taking care to avoid inducing further hypotension.

*The dose of hydrocortisone for adults and children depends on age:*
- >12 years and adults: 200 mg IM or IV slowly
- >6 – 12 years: 100 mg IM or IV slowly
- >6 months – 6 years: 50 mg IM or IV slowly
- <6 months: 25 mg IM or IV slowly

**Bronchodilators**
The presenting symptoms and signs of a severe anaphylactic reaction and life threatening asthma can be the same. As well as the drugs listed above, consider further bronchodilator therapy with salbutamol (inhaled or IV), ipratropium (inhaled), aminophylline (IV) or magnesium (IV). Remember that intravenous magnesium is a vasodilator and can cause hot flushes and make hypotension worse.
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