IAP TEXTBOOK OF

Pediatric
ICU Protocols
SECOND EDITION

Editor-in-Chief
Dr Praveen Khilnani
Head, Pediatric Intensive Care and Pediatric Pulmonology
BLK Superspeciality Hospital
New Delhi, India

Editors
Dr Deepak Ugra
Consultant Pediatrician and
IAP President 2010
Mumbai, Maharashtra, India

Dr Bala Ramachandran
Head, Pediatric Intensive Care
KKCT Hospital
Chennai, Tamil Nadu, India

Dr Soonu Udani
Head, Pediatric ICU
PD Hinduja Hospital
Mumbai, Maharashtra, India

Dr Suchita Khadse
Pediatric Intensivist
Child Hospital
Nagpur, Maharashtra, India

Co-ordinating Editor
Dr Sailesh Gupta
Honorary Secretary General
Indian Academy of Pediatrics
Kailash Darshan, Kennedy Bridge
Mumbai, Maharashtra, India

Foreword
Dr CP Bansal
IAP President 2013

Dr Rohit Agrawal
IAP President 2012

IAP National Publication House, Gwalior

JAYPEE BROTHERS MEDICAL PUBLISHERS (P) LTD
New Delhi • London • Philadelphia • Panama
Amit Gupta
Consultant Neonatologist and Intensivist
MAX Hospital
Noida, Uttar Pradesh, India

Anil Sachdev
Senior Consultant and Associate Director
Pediatric Intensive Care
SGR Hospital
New Delhi, India

Anjul Dayal
Consultant Pediatric Intensivist
Lotus Children’s Hospital
Hyderabad, Andhra Pradesh, India

Anupam Sehgal
Consultant Pediatric Intensivist
PD Hinduja Hospital
Mumbai, Maharashtra, India

Bala Ramachandran
Head
Pediatric Intensive Care
KKCT Hospital
Chennai, Tamil Nadu, India

Bhaskar Saikia
Consultant Pediatric Intensivist
BLK Superspeciality Hospital
New Delhi, India

Deepak Ugra
Consultant Pediatrician and
IAP President 2010
Mumbai, Maharashtra, India

Deepika Singhal
Senior Consultant Pediatric Intensivist
Pushpanjali Crosslay Hospital
Ghaziabad, Uttar Pradesh, India

Dheeraj More
Consultant Pediatric Intensivist
KKCT Hospital
Chennai, Tamil Nadu, India

Dhiren Gupta
Senior Consultant Pediatric Intensivist
SGR Hospital
New Delhi, India

Dinesh Chirla
Senior Consultant and Head
Pediatric Intensive Care
Rainbow Children’s Hospital
Hyderabad, Andhra Pradesh, India

Farhan Shaikh
Consultant Pediatric Intensivist
Rainbow Children’s Hospital
Hyderabad, Andhra Pradesh, India

I Santhanam
Head
Emergency Department
Institute of Child Health
Chennai, Tamil Nadu, India

Jyotinder Kaur
HOD of Pediatrics
Apollo Hospital
Ahmedabad, Gujarat, India

K Chugh
Director, Institute of Child Health and
Head, Pediatric Intensive Care
SGR Hospital
New Delhi, India

KG Ravikumar
Consultant Pediatric Intensivist
KKCT Hospital
Chennai, Tamil Nadu, India
Contributors

K Senthil Kumar
Consultant Pediatric Intensivist
KKCT Hospital
Chennai, Tamil Nadu, India

LN Taneja
Senior Consultant of Pediatrics
National Convener
Indian Academy of Pediatrics
Pediatric Advanced Life Support
MAX Superspeciality Hospital
New Delhi, India

Mahesh Mohite
Consultant Pediatric Intensivist
Panvel, Maharashtra, India

Maninder Dhaliwal
Consultant Pediatric Intensivist
Medanta Medicity
Gurgaon, Haryana, India

M Jaishree
Associate Professor
Pediatrics and Pediatric Intensive Care
Postgraduate Institute of Medical Education and Research
Chandigarh, India

Mritunjay Pao
Consultant Pediatric Intensivist
Sanjivani Hospital
Jorhat, Assam, India

Nameet Jerath
Consultant Pediatric Intensivist
IP Apollo Hospital
Delhi, India

Nitesh Singhal
Consultant Pediatric Intensivist and Fellow
Hospital for Sick Kids
Toronto, Canada

Pankaj Vohra
Senior Consultant
Pediatric Gastroenterologist
MAX Superspeciality Hospital
New Delhi, India

Prashant Mitharwal
Consultant Pediatric Intensivist
KKCT Hospital
Chennai, Tamil Nadu, India

Prasad Nayak
Consultant
Pediatric Intensive Care
AJ Institute of Medical Sciences
Mangalore, Karnataka, India

Preetha Joshi
Consultant Pediatric Intensivist
KB Ambani Hospital
Mumbai, Maharashtra, India

Prashant Pruthi
Consultant Pediatric Intensivist and Fellow
Pediatric Emergency Care
Sydney, Australia

Parthasarathi Bhattacharya
Senior Consultant and Head
Pediatric Intensive Care
Apollo Gleneagles Hospital
Kolkata, West Bengal, India

Prabhat Maheshwari
Senior Consultant Pediatric Intensivist
Artemis Health Care Institute
Gurgaon, Haryana, India

Praveen Khilnani
Head
Pediatric Intensive Care and Pediatric Pulmonology
BLK Superspeciality Hospital
New Delhi, India

Pradeep Sharma
Associate Consultant and PICU Fellow
BLK Superspeciality Hospital
New Delhi, India

Rachna Sharma
Consultant Pediatric Intensivist
BLK Superspeciality Hospital
New Delhi, India
Contributors

**Rajiv Uttam**
Director Pediatric Intensive Care
MAX Superspeciality Hospital
New Delhi, India

**Rakesh Lodha**
Associate Professor and
Pediatric Intensivist
All India Institute of Medical Sciences
New Delhi, India

**Sajith Kesavan**
Consultant Pediatric Intensivist
KKCT Hospital
Chennai, Tamil Nadu, India

**Shipra Gulati**
Pediatric Intensivist
MAX Superspeciality Hospital
New Delhi, India

**Sunit Singh**
Professor and HOD
Department of Pediatrics
Postgraduate Institute of Medical Education and Research
Chandigarh, India

**S Deopujari**
Senior Consultant
Child Hospital
Nagpur, Maharashtra, India

**Suchitra Ranjit**
Head
Pediatric Intensive Care
Apollo Children’s Hospital
Chennai, Tamil Nadu, India

**Sagar Lad**
Consultant Pediatric Intensivist
Jehangir Apollo Hospital
Pune, Maharashtra, India

**Soonu Udani**
Head, Pediatric ICU
PD Hinduja Hospital
Mumbai, Maharashtra, India

**Santosh Soans**
HOD of Pediatrics
AJ Institute of Medical Sciences
Mangalore, Karnataka, India

**Samith Alva**
Pediatrics Intensivist
AJ Institute of Medical Sciences
Mangalore, Karnataka, India

**Suchita Khadse**
Pediatric Intensivist
Child Hospital
Nagpur, Maharashtra, India

**Uma Ali**
Consultant Pediatric Intensivist and
Nephrologist
Lilavati Hospital
Mumbai, Maharashtra, India

**Vikram Gagneja**
Pediatric Intensivist
BLK Superspeciality Hospital
New Delhi, India

**Vinayak Patki**
Consultant Pediatric Intensivist
Wanless Hospital
Miraj, Maharashtra, India

**VSV Prasad**
Senior Consultant Pediatric Intensivist and CEO
Lotus Children’s Hospital
Hyderabad, Andhra Pradesh, India

**Vikas Taneja**
HOD and Consultant Pediatric Intensivist
Columbia Asia Hospital
Gurgaon, Haryana, India

**Vinay Joshi**
Consultant Pediatric Intensivist
KB Ambani Hospital
Mumbai, Maharashtra, India

**V Buche**
Consultant Pediatrics
Child Hospital
Nagpur, Maharashtra, India

**Vinay Aggarwal**
Senior Consultant Pediatric Nephrologist
BLK Superspeciality Hospital
New Delhi, India

**Yogesh Waikar**
Consultant Pediatric Gastroenterologist
PD Hinduja Hospital
Mumbai
Maharashtra, India
Dear Reader,

The book that you hold is the fulfilment of the dreams of the doyens of Indian Academy of Pediatrics (IAP). For many years, the need for good Indian books in every specialty of pediatrics was felt. The Indian Academy of Pediatrics has no dearth of great teachers and writers in the various subspecialties to author these books. Their dedicated and diligent labor has created the beautiful and eminently readable book that you hold. An Indian book by Indian authors will appropriately suit the needs of the readers in India and in countries with similar geographical and sociocultural milieus. Although the first editions of the IAP subspecialty series were published in 2006, we proudly present to you a second, completely revised and updated edition.

The IAP specialty series books serve the purpose of providing evidence based, authentic and uniform information to IAP members, other pediatricians, and students of pediatrics in the country. Guidelines and established protocols on disease management will be very helpful for pediatricians in their everyday practice.

Creating a book is such as the birth of a baby. Right from conception to delivery, there is a long and complex process. It is very labor intensive and time-consuming work that involves considerable financial expense too. To streamline the entire process from writing to editing to publishing to distribution and sales of books, it was envisioned to have an additional wing of IAP, and which is established as "IAP National Publication House (IAP NPH)" at Gwalior.

Knowledge has no limits and seekers of knowledge can access the subject from anywhere in the world. We understand that books published by IAP NPH will be read and referred not only in India but also in many parts of the world. Objective of IAP NPH, therefore, is to provide standardized content and world class quality. With this objective, printed books are to be made available throughout the globe and distribution will also be done through online editions. Publishing 7 books at a time is a mammoth task and for this we collaborated with the second largest medical publisher in the world, i.e. M/s Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, India.

What you are reading, the world is also reading. Our writers are getting worldwide exposure and readers are getting world class books at reasonable cost.
It needs to be mentioned here that all authors and editors have dedicated the royalty from sale of books to IAP and have thereby done a selfless service for our mother organization. By buying this book, you are also contributing to IAP in a significant manner.

Finally, we express our pride and happiness in being associated with this project and in reaching this valuable book to you. We wish you a happy and contented reading.

CP Bansal
IAP President 2013

Rohit Agrawal
IAP President 2012
It has been long felt that textbooks of pediatric critical care tend to be too exhaustive in pathophysiology and somewhat difficult to read and understand for an average pediatrician, essentially a nonintensivist.

It has also been strongly felt that ready availability of a manual or a handbook describing an easy-to-implement protocol-based approach to commonly seen potentially life-threatening pediatric emergencies would be of great use to residents, pediatric consultants and even physicians taking care of critically ill or injured children.

Under the action plan of IAP, this book entitled IAP Textbook of Pediatric ICU Protocols was conceived to be edited by national experts in this vast field of pediatric intensive care. Contributors have been many intensivists and academicians from all over the country.

It is our sincere hope that this manual IAP Textbook of Pediatric ICU Protocols would be available in all emergency rooms and pediatric intensive care units as well as in various medical colleges, and would be used to improve the outcomes of critically ill children. Evidence-based and practice-oriented protocols have been used in an easy-to-read format with a flow diagram after every chapter as much as feasible.

Praveen Khilnani
Deepak Ugra
CP Bansal
Bala Ramachandran
Soonu Udani
Suchita Khadse
This book entitled *IAP Textbook of Pediatric ICU Protocols* is intended to be a practical current state of the art resource for pediatric residents, pediatricians and physicians involved in care of neonatal and pediatric emergency, and intensive care patients commonly seen in the Indian scenario.

A practice-oriented protocol-based approach to common conditions such as acute pneumonia, severe asthma, acute respiratory distress syndrome (ARDS), cardiorespiratory failure, shock, sepsis, coma, raised intracranial pressure, status epilepticus protocol, cardiac failure, rhythm abnormalities, including postoperative cardiac surgery in critically ill children has been discussed. It has been the effort to include as much evidence-based protocols as feasible.

Basic mechanical ventilation details have been emphasized including airway management, rapid sequence intubation, initial ventilator settings, maintenance, monitoring and ongoing adjustment of mechanical ventilation, arterial blood gas analysis and weaning from the ventilator.

Besides a discussion of basic conventional ventilation, advanced ventilation modes, high frequency oscillatory ventilation (HFOV) and noninvasive ventilation protocols have also been included. Wherever feasible, practical case scenarios of commonly seen neonatal and pediatric conditions involving acute renal failure, fulminant hepatic failure, inborn errors of metabolism, acid-base, and fluid and electrolyte disturbances have also been included.

Finally, a brief description of commonly seen poisonings, multiple trauma, standard basic life support (BLS), pediatric advanced life support (PALS) protocols, sedation protocols, pediatric intensive care unit (PICU) drug dosages as well as PICU procedures have been covered with a view of having that information readily available to the reader.

Hope this protocol book finds important place in emergency rooms and PICUs at various institutions facilitating delivery of appropriate and timely emergency and intensive care at right time by all residents, fellows and consultant pediatricians.

Praveen Khilnani
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Approach to Sick Child

Praveen Khilnani

Recognize Sick Child: “Early and promptly”
(Crying unconsolable child, sick looking, refusing feeds, lethargic, comatose, respiratory distress, shock, convulsion)

Office/clinic

Minimum equipment for pediatric clinic
- Oxygen, Ambu bag and mask, oral and nasal airway laryngoscope, ET tubes, IV cannula, Intraosseous needles, IV fluids, suction apparatus, Pulse oximeter and Nebulizer, BP apparatus

Rapidly assess ABC
- Airway: Patency, secretions obstruction, foreign body
- Breathing: Respiratory rate/depth, grunting, retractions, accessory muscle use, see-saw movements
- Circulation: Pulse, blood pressure, capillary refill time

Emergency room
- Start oxygen

Recognize
“Compensated shock” CNS: Lethargy, coma, neck stiffness, convulsions, GCS < 8

Rapidly stabilize

Once a sick child is recognized:
- Do not waste time in detailed examination and investigation
- Regardless of diagnosis, give oxygen

Respiratory distress/failure
- Ensure airway patency: Open airway with jaw thrust or chin lift
- Suspected trauma: Stabilize cervical spine
- If airway compromised: Intubate and start manual ventilation
- If difficult/unable to intubate: Bag and mask ventilation is effective
- Foreign body: Back blows, chest thrusts and Hambright maneuver

Shock
- Establish IV if access >90 seconds
- Establish intraosseous access
- Push bolus of isotonic fluid 20 ml/kg
- If known hypovolemia:
  - Sepsis/dengue: 40-80 ml/kg

Reassess
- Reassess after each
  - intervention
    - Airway: Patency
    - Breathing: Rate, distress, SpO₂
    - Circulation: Capillary, refill, color, heart rate, blood pressure, mental status and urine output

Supportive treatment
- Convection: IV benzodiazepine
- Fever: Antipyretics
- Vomiting: Antiemetics
- Pain: Analgesics
- Anaphylaxis/allergic reaction: Antihistaminic/steroid/adrenaline
- Bronchospasm: Nebulize salbutamol
- Fluid unresponsive shock: Dopamine via peripheral IV

After initial stabilization and ensuring-ABC under reasonable control with IV access established, transfer/transport the child to higher level facility for admission in PICU

Abbreviations: ABC, airway, breathing and circulation; GCS, Glasgow Coma Scale; ET, endotracheal; IV, intravenous; BP, blood pressure; SpO₂, saturation of oxygen; PICU, pediatric intensive care unit
Approach to Collapsed Child

Praveen Khilnani, Rachna Sharma

Abbreviations: CPR—cardiopulmonary resuscitation; AED—automated external defibrillator; PALS—pediatric advanced life support.
Approach to Respiratory Distress

Praveen Khilnani, Bhaskar Saikia
Airway Management Protocol

K Sajith, KG Ravikumar, Bala Ramachandran

**Airway Compromise**
- Stridor
- Severe retractions (mainly suprasternal)
- Paradoxical respirations
- Hoarse or absent sounds
- Arching of the neck
- Drooling and inability to swallow

**Humidified oxygen**: Administer oxygen by simple face masks, nasal cannulae or nonrebreathing masks. Foreign body? follow BLS protocol

**Positioning**: Neck should be slightly flexed on the chest and head should be extended at the atlanto-occipital joint (sniffing position). Because of the large head in infants a shoulder pad may be necessary

**Suction and clear airway**: Suction force of 80–120 cm water is usually necessary if airway is obstructed with secretions

**Maintain airway**: By head tilt chin lift or jaw thrust maneuver.

- Triple airway maneuver: With fingers behind jaw, mandible is displaced downward, forward and finally upward until mandible and lower incisors are anterior to the maxilla.

**Nebulization with adrenaline**: 0.5 ml/kg, maximum 5 ml can be tried if there is stridor due to probable edema, but maintaining airway and saturation

- Injection dexamethasone 0.6 mg/kg IM or IV can be given in croup

**Bag and Mask Ventilation**
- Give positive pressure ventilation if the child is apnoic or the spontaneous respiratory efforts are inadequate to maintain gas exchange
- **Sellick’s maneuver**: Cricoid pressure should be applied to prevent aspiration if child is not empty stomach or time of last feed is not known
- Watch for adequate chest rise and improvement in saturations and heart rate

*Contd...*
Laryngeal Mask Airway
This is a recently introduced technique of securing airway by relatively inexperienced medical personnel such as nurses and paramedics, who may not succeed in placing an endotracheal tube in a pediatric patient. The main principle behind its use is to place the tip of LMA in the beginning of the upper esophagus to occlude it, so that the portion of LMA superior to the upper end of esophagus outlines the glottis thereby placing a mask on the larynx (Figs 4.1A and B). It can be used to maintain airway, provide bag ventilation, used for passage of a flexible bronchoscope as well as passing a suction catheter for suction of copious secretions. The laryngeal mask allows maintenance of airway patency but does not resolve all ventilatory problems in children. The use in neonate and infant has to be cautious and more clinical experience needs to be acquired before widespread use can be recommended. The LMA should not be used as a substitute when endotracheal intubation is necessary.

Many children with severe facial malformations that make tracheal intubation hazardous, or even impossible, greatly benefit from its use, since a tracheal tube
can be introduced through the tube and advanced blindly into the trachea. This technique has led the manufacturer to develop a variant of the laryngeal mask (Fastrach) designed to allow insertion of a tracheal tube through the laryngeal mask, the mask is removed and the tracheal tube is then connected to ventilatory bag.

Benefits are avoiding tracheal intubation, fiberoptic tracheal intubation and reduced hemodynamic variations compared to tracheal intubation.

Disadvantages are difficult insertion and easy displacement, partial or complete airway obstruction, laryngospasm in cases of insufficient sedation and anesthesia, uvular, pharyngeal and laryngeal trauma.

The laryngeal mask is not indicated in the presence of vomiting, regurgitation and full stomach, in emergency, in chronic lung disease and in patients with large quantities of secretions, in hypertrophic tonsils, in patients with malformation of oral cavity.

Various adult and pediatric sizes are available (Table 4.1). It is contraindicated in patients with full stomach and in patients requiring intubation due to airway obstruction due to inflammation or edema.

**Table 4.1: Available sizes of Laryngeal Mask Airway**

<table>
<thead>
<tr>
<th>LMA size</th>
<th>Patient selection information</th>
<th>Maximum inflation volume (ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>LMA-Classic™</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Neonates/infants up to 5 kg</td>
<td>4</td>
</tr>
<tr>
<td>1½</td>
<td>Infants 5–10 kg</td>
<td>7</td>
</tr>
<tr>
<td>2</td>
<td>Infants/children 10–20 kg</td>
<td>10</td>
</tr>
<tr>
<td>2½</td>
<td>Children 20–30 kg</td>
<td>14</td>
</tr>
<tr>
<td>3</td>
<td>Children 30–50 kg</td>
<td>20</td>
</tr>
<tr>
<td>4</td>
<td>Adult 50–70 kg</td>
<td>30</td>
</tr>
<tr>
<td>5</td>
<td>Adult 70–100 kg</td>
<td>40</td>
</tr>
<tr>
<td>6</td>
<td>Large adult over 100 kg</td>
<td>50</td>
</tr>
<tr>
<td><strong>LMA-Flexible™</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Neonates/infants up to 5 kg</td>
<td>4</td>
</tr>
<tr>
<td>1½</td>
<td>Infants 5–10 kg</td>
<td>7</td>
</tr>
<tr>
<td>2</td>
<td>Infants/children 10–20 kg</td>
<td>10</td>
</tr>
<tr>
<td>2½</td>
<td>Children 20–30 kg</td>
<td>14</td>
</tr>
<tr>
<td>3</td>
<td>Children 30–50 kg</td>
<td>20</td>
</tr>
<tr>
<td>4</td>
<td>Adult 50–70 kg</td>
<td>30</td>
</tr>
<tr>
<td>5</td>
<td>Adult 70–100 kg</td>
<td>40</td>
</tr>
<tr>
<td>6</td>
<td>Large adult over 100 kg</td>
<td>50</td>
</tr>
<tr>
<td><strong>LMA-Unique™</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Children 30–50 kg</td>
<td>20</td>
</tr>
<tr>
<td>4</td>
<td>Adult 50–70 kg</td>
<td>30</td>
</tr>
<tr>
<td>5</td>
<td>Adult 70–100 kg</td>
<td>40</td>
</tr>
</tbody>
</table>
Predictors of Difficult Intubation
- History of previous difficult intubation
- History or clinical features of upper airway obstruction
- History of snoring and sleep apnea
- Midface hypoplasia
- Small mouth
- Micro- or retrognathia
- Severe obesity
- Midline clefts
- Limited neck mobility
- Facial trauma or edema
- Oropharyngeal mass
- Airway bleeding
- Limited temporomandibular (TM) joint mobility.

Algorithm for Difficult Airway

<table>
<thead>
<tr>
<th>Recognized (Best method is to intubate in operation theater under inhaled anesthesia)</th>
<th>Unrecognized (If unable to intubate after giving sedation and neuromuscular agents)</th>
</tr>
</thead>
<tbody>
<tr>
<td>If unstable to be shifted or either awake, intubation or sedation + half dose of neuromuscular blocker can be tried</td>
<td>Try bag and mask ventilation till help arrives</td>
</tr>
<tr>
<td>Check equipment (get anesthesiology and ENT back up)</td>
<td>If not able to ventilate, choose LMA</td>
</tr>
<tr>
<td>If unsuccessful, invasive airway access—cricothyrotomy and tracheostomy</td>
<td>LMA failed, get invasive airway access—cricothyrotomy and retrograde intubation or tracheostomy</td>
</tr>
</tbody>
</table>

Alternative noninvasive intubation: Fiberoptic intubation, bronchoscopy assisted intubation, light wand, intubating stylet, intubating LMA

Contents of Cricothyrotomy Kit
Size 3 endotracheal tube adapter and 16 G cannula.
Rapid sequence intubation (RSI) is a technique used to secure the airway in the patient who presents with a full (or presumed full) stomach, where even moderate preintubation gastric insufflation by bag-mask ventilation may cause gastric regurgitation and pulmonary aspiration.

Rapid sequence intubation is contraindicated in patients who cannot be orally intubated. Rapid sequence intubation should be avoided in patients with laryngotracheal abnormalities caused by tumors, infection, edema or a history of cervical radiation therapy. In all such difficult airway scenarios, do not paralyze at the time of intubation—sedate, intubate and then paralyze (known as “awake intubation”).

The main purpose of RSI is to avoid positive pressure ventilation by bag and mask, and prevent gastric inflation in patients at risk of aspiration.

However, bag-valve-mask (BVM) ventilation may be necessary in apneic patients or those with ineffective spontaneous breathing. In such patients, Sellick’s maneuver is performed to prevent air entering into stomach and gentle Ambu bagging can be done.

Table 5.1: The eight “P”s of rapid sequence intubation (RSI)

<table>
<thead>
<tr>
<th>Time</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zero minus 10 minutes</td>
<td>Preparation</td>
</tr>
<tr>
<td>Zero minus 5 minutes</td>
<td>Preoxygenation</td>
</tr>
<tr>
<td>Zero minus 3 minutes</td>
<td>Pretreatment</td>
</tr>
<tr>
<td>—Time zero—</td>
<td>Paralysis with induction</td>
</tr>
<tr>
<td>Zero plus 20–30 seconds</td>
<td>Protection and positioning</td>
</tr>
<tr>
<td>Zero plus 45 seconds</td>
<td>Placement</td>
</tr>
<tr>
<td>Zero plus 45 seconds</td>
<td>Proof</td>
</tr>
<tr>
<td>Zero plus 1 minute</td>
<td>Postintubation management</td>
</tr>
</tbody>
</table>
Steps in Rapid Sequence Intubation

Step 1: Preparation

Table 5.2: The suction, oxygen, airway equipment, pharmacy and monitoring equipment (SOAP ME) mnemonic

<table>
<thead>
<tr>
<th>Suction</th>
<th>Suction should be tested and readily available at the bedside</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen</td>
<td>A high-flow oxygen mask and bag-valve ventilation device should be ready for use</td>
</tr>
<tr>
<td>Airway equipment</td>
<td>At least two functioning laryngoscope handles and the appropriately sized and shaped laryngoscope blades should be obtained. The anticipated blade of choice should be clicked into position to ensure that the light functions properly. An endotracheal tube (ETT) is chosen based on the patient’s anatomy, and one smaller size should be prepared as well. In children, the ETT size may be estimated by the formula, ETT size = 4+ (age in years/4). The average adult male will require a 7.5 or 8.0 sized ETT, the average adult female a 7.0 or 7.5. The ETT cuff should be inflated to test for an air leak. A stylet should be inserted within the ETT to shape it into a configuration that will facilitate insertion into the airway. Care must be taken to ensure that the tip of the stylet does not protrude from the end of the ETT or through the small distal side port (Murphy’s eye)</td>
</tr>
<tr>
<td>Pharmacy</td>
<td>The patient should have at least one intravenous (IV), and patency of the line should be verified and ensured. The specific rapid sequence intubation (RSI) medications, proper dosing, and sequence of administration should be determined, and the agents should be drawn up and labeled</td>
</tr>
<tr>
<td>Monitoring</td>
<td>Cardiac monitoring, blood pressure monitoring, and pulse oximetry are mandatory for all patients. If available, an end-tidal carbon dioxide (EtCO₂) monitor should be readied as well. In a wilderness setting, many of these hospital-based amenities may not be available</td>
</tr>
</tbody>
</table>

Step 2: Preoxygenation

Preoxygenation effectively leads to “nitrogen washout”, within the lungs with 100% oxygen. This allows for a prolonged period of apnea without arterial oxygen desaturation. Patient is preoxygenated by 100% oxygen for 5 minutes before paralysis.

Step 3: Pretreatment

The act of laryngoscopy, and the drugs used for sedation and paralysis, can lead to adverse effects, such as bronchospasm, increased intracranial pressure (ICP), increased intraocular pressure, increased intragastric pressure, increased sympathetic discharge and bradycardia. Pretreatment medications may be given to mitigate these adverse effects.
**Table 5.3: Pretreatment medications—lidocaine, opioid, atropine and defasciculation (LOAD) mnemonic**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Indication</th>
<th>Mechanism</th>
<th>Pediatric dose (IV)</th>
<th>Adult dose (IV)</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lidocaine</td>
<td>↑ICP, RAD</td>
<td>↑ Intracranial response to intubation, mitigates bronchospasm in RAD</td>
<td>1.5 mg/kg</td>
<td>1.5 mg/kg</td>
<td></td>
</tr>
<tr>
<td>Opioid (fentanyl)</td>
<td>↑ICP, ischemic heart disease, aortic dissection</td>
<td>Blunts sympathetic response to laryngoscopy</td>
<td>1–3 μg/kg</td>
<td>3–6 μg/kg</td>
<td>Use with caution in young children</td>
</tr>
<tr>
<td>Atropine</td>
<td>Children &lt; 10 years; adults receiving a second dose of SCh</td>
<td>Mitigates bradycardic response to SCh</td>
<td>0.02 mg/kg (minimum dose 0.1 mg)</td>
<td>2.0 mg/kg</td>
<td></td>
</tr>
<tr>
<td>Defasciculation</td>
<td>↑ ICP or globe injury</td>
<td>Defasculates and mitigates ICP response to SCh</td>
<td>0.01 mg/kg</td>
<td>0.01 mg/kg</td>
<td>Only for adults and children &gt; 20 kg</td>
</tr>
</tbody>
</table>

Abbreviations: ICP, intracranial pressure; RAD, reactive airway disease; Sch, succinylcholine

**Step 4A: Paralysis (with Induction)**

The next step in RSI is rapid intravenous (IV) administration of an induction agent to produce complete loss of consciousness, followed immediately by administration of a neuromuscular blocking agent (NMBA) to induce complete motor paralysis.

**Induction Agents**

The choice of agent is based on the patient’s clinical condition and the agent’s own specific attributes.

**Table 5.4: Clinical characteristics of induction agents**

<table>
<thead>
<tr>
<th>Induction agents</th>
<th>Induction dose (IV)</th>
<th>Onset of action</th>
<th>Duration of action</th>
<th>Benefits</th>
<th>Precautions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thiopental</td>
<td>1–5 mg/kg</td>
<td>&lt; 30 sec</td>
<td>5–10 min</td>
<td>↓ ICP</td>
<td>↓ BP, laryngospasm</td>
</tr>
<tr>
<td>Midazolam</td>
<td>0.2–0.3 mg/kg</td>
<td>30–60 sec</td>
<td>15–30 min</td>
<td>Reversible, amnestic, anticonvulsant</td>
<td>Apnea, no analgesia</td>
</tr>
</tbody>
</table>

*Contd...*
Rapid Sequence Intubation

Step 4B: Paralysis: Neuromuscular Blocking Agents
Paralytic agents do not provide any analgesia, sedation, or amnesia. The ideal NMBA should have rapid onset of action, short duration of action, and few adverse side effects. Succinylcholine (SCh), a depolarizing NMBA, comes closest to meeting all these traits. Succinylcholine has its side effects, including muscle fasciculations, bradycardia, hyperkalemia, malignant hyperthermia, and trismus (masseter muscle spasm).

Nondepolarizing NMBAs possess fewer side effects than SCh, but have delayed time to paralysis, prolonged duration of action, or both.

Table 5.5: Neuromuscular blocking agents

<table>
<thead>
<tr>
<th>Neuromuscular blocking agent</th>
<th>Intubating dose (IV)</th>
<th>Onset</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Depolarizing</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Succinylcholine</td>
<td>1.5 mg/kg (adult)</td>
<td>45–60 sec</td>
<td>6–12 min</td>
</tr>
<tr>
<td></td>
<td>2 mg/kg (child)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3 mg/kg (infant)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Nondepolarizing</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rocuronium</td>
<td>1.0 mg/kg</td>
<td>50–70 sec</td>
<td>30–60 min</td>
</tr>
<tr>
<td>Vecuronium</td>
<td>0.15 mg/kg</td>
<td>90–120 sec</td>
<td>60–75 min</td>
</tr>
<tr>
<td>Pancuronium</td>
<td>0.1 mg/kg</td>
<td>100–150 sec</td>
<td>120–150 min</td>
</tr>
</tbody>
</table>

Step 5A: Protection
After sequential administration of the induction and paralytic agents, the patient will lose consciousness and become apneic. Perform Sellick’s maneuver (the application of cricoid pressure), just as the patient is noted to lose consciousness. This application of firm pressure to the cricoid cartilage compresses the esophagus, preventing passive regurgitation of gastric contents and aspiration.
Sellick’s maneuver should be maintained until the endotracheal tube (ETT) has been inserted into the trachea, its position verified, and the cuff inflated.

**Step 5B: Positioning—For Children Below 2 Years Age**

For an infant, the large occiput contributes to flexion of the head and neck and resultant airway obstruction. This is alleviated by elevating the shoulders with a towel roll.

*For children above 2 years:* Towel roll is placed under the head, followed by gentle hyperextension of the head at the atlanto-occipital joint, provides optimal alignment of the airway axes (T = Tracheal axis, P = Pharyngeal axis, O = Oral axis).

**Step 6: Placement**

Complete muscular paralysis can be confirmed by gently grasping the patient’s jaw and checking for flaccidity. With the laryngoscope in the left hand, gently open the mouth with the right hand. Insert the laryngoscope into the right side of the patient’s mouth and sweep the tongue to the left.

The curved (Macintosh) blade is slid into the vallecula while the straight (Miller) blade is positioned below the epiglottis. The handle is pushed along the axis of the handle at a 45° to the patient’s body. If the glottic aperture is not readily visible then perform the backward, upward, rightward pressure (BURP) maneuver. This involves placement of the right hand on the thyroid cartilage, followed by application of BURP to help bring the glottis into view.

When the glottis is visible, the right hand gently inserts the ETT until the cuff is about 2–3 cm past the vocal cords. Once the ETT is in place, remove the stylet and inflate the cuff.

**Step 7: Proof (Confirmation of Endotracheal Tube Placement)**

Sellick’s maneuver should not be released until confirmation of correct ETT placement.

Methods used to detect ETT placement include clinical assessment (visual confirmation of ETT passing through the vocal cords, air entry at lung bases on auscultation, chest rise while Ambu bagging, fogging of the ETT), pulse oximetry, end-tidal carbon dioxide (EtCO$_2$) detection, and aspiration techniques. Chest radiography assesses ETT position but does not confirm ETT placement within the trachea.

**Step 8: Postintubation Management**

After verification of correct placement of the ETT within the trachea, the tube should be secured (taped or tied) in place to ensure it does not move or migrate. The patient’s blood pressure and other vital signs should be repeatedly monitored.
Conclusion

The purpose of RSI is to make emergent intubation easier and safer, thereby, increasing the success rate of intubation while decreasing the complications.

Abbreviations: IV, intravenous; SPO$_2$, oxygen saturation; ECG, electrocardiogram; BP, blood pressure; ETT, endotracheal tube; ETCO$_2$, end-tidal carbon dioxide concentration
Definition
Respiratory failure is defined as inability of respiratory system to adequately deliver oxygen or remove CO₂ or both. Terms such as respiratory distress (mild, severe) and respiratory failure (early, late) represent spectrum of severity.

Etiology of Acute Respiratory Failure
These diseases can be grouped according to the primary abnormality (Table 6.1):

Classification of Respiratory Failure
Respiratory failure may be classified as hypoxemic or hypercapnic.

Table 6.1: Common causes of respiratory failure

<table>
<thead>
<tr>
<th>Common causes of Type I (hypoxemic) respiratory failure</th>
<th>Common causes of Type II (hypercapnic) respiratory failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumonia</td>
<td>Chronic bronchitis and emphysema</td>
</tr>
<tr>
<td>Pulmonary edema</td>
<td>Severe asthma</td>
</tr>
<tr>
<td>Pneumothorax</td>
<td>Drug overdose</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
<td>Poisonings</td>
</tr>
<tr>
<td>Pulmonary arterial hypertension</td>
<td>Myasthenia gravis</td>
</tr>
<tr>
<td>Cyanotic congenital heart disease</td>
<td>Polyneuropathy</td>
</tr>
<tr>
<td>Bronchiectasis</td>
<td>Poliomyelitis</td>
</tr>
<tr>
<td>Acute respiratory distress syndrome (ARDS)</td>
<td>Primary muscle disorders</td>
</tr>
<tr>
<td>Fat embolism syndrome</td>
<td>Head and cervical cord injury</td>
</tr>
<tr>
<td>Obesity</td>
<td>Primary alveolar hypoventilation</td>
</tr>
<tr>
<td></td>
<td>Obesity hypoventilation syndrome</td>
</tr>
</tbody>
</table>
Respiratory Failure

**Hypoxemic Respiratory Failure (Type I)**
This is characterized by a PaO₂ of less than 60 mmHg with a normal or low PaCO₂. This is the most common form of respiratory failure, and it can be associated with virtually all acute diseases of the lung, which generally involve fluid filling or collapse of alveolar units. Some examples of Type I respiratory failure are cardiogenic or noncardiogenic pulmonary edema, pulmonary hemorrhage and pneumonia.

**Hypercapnic Respiratory Failure (Type II)**
This is characterized by a PaCO₂ of more than 50 mmHg. Hypoxemia is common in patients with hypercapnic respiratory failure who are breathing room air. The pH depends on the level of bicarbonate, which, in turn, is dependent on the duration of hypercapnia. Common etiologies include drug overdose, neuromuscular disease, chest wall abnormalities and severe airway disorders, e.g. asthma, chronic obstructive pulmonary disease (COPD).

**Approach to a Child with Acute Respiratory Failure**
For adequate management of a child with acute respiratory failure, proper detailed history, physical examination and relevant investigations are necessary. It must, however, be emphasized that establishing a detailed diagnosis may take up important initial intervention time in order of priority after quick history and rapid cardiopulmonary assessment. For example, after ensuring patent airways, breathing and circulation (ABC), beginning oxygen therapy in a wheezing patient without waiting for a chest radiograph may be appropriate as dictated by the clinical condition.

History of onset and duration of symptoms prior to onset of respiratory distress is important. Respiratory problems at birth such as premature birth and hyaline membrane disease, apnea, stridor, asphyxia and respiratory distress or neuromuscular problems should be enquired into.

While conducting a rapid cardiopulmonary assessment for examination of a child in respiratory failure, the following points must be paid attention to:

- General condition: playful, toxic, drooling or continuously coughing
- Color: pink, pale or cyanosed
- Mental status: agitated, anxious, lethargic, comatose
- Chest deformity/scoliosis
- Hoarse voice, no voice or croupy cough
- Respiratory rate: tachypnea, bradypnea or episodes of apnea
- Audible wheeze
- Accessory muscle use: head bobbing, nasal flaring, sternocleidomastoid prominence, suprasternal retractions, subcostal and intercostal retraction
- Breath sounds: equal, diminished or absent, wheezes, rales (crepitation)
- Tachycardia
- Congenital facial deformity/airway problems, such as choanal atresia, short chin (mandibular hypoplasia), micrognathia or retrognathia
Simultaneous initial intervention and investigations include:

- Placing a pulse oximeter probe in the emergency department (casualty) to check oxyhemoglobin saturation should be a standard of care on all patients in acute respiratory failure.
- Oxygen therapy by mask, nasal cannula or head box should be initiated at the first opportunity.
- Position of comfort should be maintained, such as sitting position or in mother's lap to control child's anxiety. One should avoid forcefully laying down the child for examination of throat, or for a neck or chest radiograph, as this can precipitate severe airway obstruction, cyanosis, bradycardia and cardiac arrest in a child with partial upper airway obstruction. If airway and breathing are maintained, aerosol therapy with a beta stimulant (salbutamol) or adrenaline nebulizer may be initiated depending upon predominant wheezing or stridor respectively.
- On the other hand if the airway is not maintained or respiratory distress is severe, airway should be emergently opened with jaw thrust or head tilt, and chin lift maneuver followed by bag mask ventilation with 100% oxygen and endotracheal intubation.
- Preferably, endotracheal intubation should be performed under controlled situation in the pediatric intensive care unit (PICU) or the operation theater, especially if labile upper airway obstruction is suspected, such as in cases of severe croup/epiglottitis/bacterial tracheitis, with ready availability of tracheostomy set up and an ENT surgeon available as stand by while endotracheal intubation procedure is being performed (endotracheal intubation procedure is described elsewhere).
- Blood for complete blood count (CBC), blood culture and basic metabolic investigations such as sodium, potassium, urea and creatinine may be drawn. Arterial blood gas can also be drawn.
- A good intravenous (IV) line should be established for IV fluid therapy, for drug therapy such as steroids and antibiotics as needed.
- Portable upright chest and airway (neck) anteroposterior and lateral view radiographs may be obtained if patient is relatively stable.
- If history is suggestive of inhalation of foreign body followed by respiratory distress in a previously asymptomatic child, bronchoscopic removal of foreign body may be required under anesthesia. In a child with history of foreign body obstruction, pediatric advanced life support (PALS) protocol comprising of back blows, chest thrusts and Heimlich's maneuver should be followed.

Indices Used to Assess Lung as an Oxygenator (Fig. 6.1)

- \( \text{PaO}_2 \): Normal value in newborn infant at sea level 40–70 mmHg, then increase till adult values of 90–120 mmHg.
  - Hypoxemia: \( \text{PaO}_2 \) lower than the acceptable range for age. In general, for a child, hypoxemia is if \( \text{PaO}_2 \) is less than 60 mmHg.
  - Hypoxia: Inadequate tissue oxygenation.
- \( \text{SaO}_2 \): Aim to maintain saturation of oxygen greater than or equal to 92%.
Respiratory Failure

- Qs/Qt: Normally shunt fraction is less than 10% of total cardiac output. In case of respiratory failure, shunt fraction is greater than 15%.
- PA-PaO₂: Normally less than 20 mmHg in child and less than 50 mmHg in newborn. In respiratory failure, difference is greater than 300 torr with FiO₂ of 100%.
- PaO₂/FiO₂: Normal ratio is greater than 400 mmHg breathing room air at sea level
  Ratio less than 300: acute lung injury
  Ratio less than 200: ARDS.

Indications for Admission to the PICU

In general, all patients too unstable to be managed in ward should be admitted to the PICU. These include:
- Severe respiratory distress, tachypnea and retractions
- Oxygen requirement on the rise greater than 50% to maintain hemoglobin saturations above 90%
- Desaturation below 90% on highest flow of oxygen
- Lethargic child
- Arterial blood gas showing hypoxemia, hypercarbia or metabolic acidosis.

Indications for Mechanical Ventilation

- The indications are mainly clinical. Although blood gases are important for decision, they are not absolutely necessary. The usual indications include:
- PaO₂ less than 55 mmHg or PaCO₂ greater than 60 mmHg despite 100% oxygen therapy

![Flow diagram for evaluation of hypoxemia](image)

**Fig. 6.1**: Flow diagram for evaluation of hypoxemia

*Abbreviations: A-a, alveolar-arterial; V/Q, ventilation/perfusion; GB syndrome, Guillain–Barré syndrome*
Deteriorating respiratory status despite oxygen and nebulization therapy
Anxious, sweaty lethargic child with deteriorating mental status
Respiratory arrest (must be avoided at all cost).

Emergency Management of Few Important Clinical Problems

Upper Airway Obstruction
Nasal and pharyngeal causes such as choanal stenosis or atresia will cause cyanosis at rest. The common symptom of upper airway obstruction is stridor. In infancy, important causes of stridor include laryngomalacia, vocal cord paralysis, laryngeal web, vascular ring, tracheal stenosis, airway hemangiomas, hypocalcemia and hypoparathyroidism.

In older child, common infectious causes causing upper airway obstruction (signs and symptoms of hoarseness, drooling and swallowing difficulty, stridor with or without respiratory distress) include laryngotracheobronchitis, epiglottitis, diphtheria, tonsillitis/peritonsillar abscess, retropharyngeal abscess, bacterial tracheitis and tracheobronchomalacia.

Other causes of upper airway obstruction should be looked into such as airway foreign bodies, airway tumors, postintubation stridor and subglottic stenosis.

The emergency department investigations and management comprise:

- Oxygen for all patients and no sedation
- To assess whether airway maintained and stable.

If the airway is stable:
- Maintain position of comfort
- Do not force to examine the airway
- Do not lay the child forcefully
- Accompany the child for portable soft tissue anteroposterior and lateral neck X-rays.

If the airway is unstable:
- Secure airway first in the best possible location in controlled environment, i.e. operation theater or the PICU with availability of emergency tracheostomy.

Once the airway has been secured:
- Give IV antibiotics and steroids as indicated
- Ceftriaxone/cefuroxime for tracheitis, peritonsillar abscess and epiglottitis
- Adrenaline nebulization and IV methylprednisolone/dexamethasone for laryngotracheobronchitis and hypertrophic tonsils in infectious mononucleosis. For angioedema, subcutaneous adrenaline and IV dexamethasone is indicated.
- After initial stabilization, transfer to PICU.

Acute Respiratory Distress Syndrome/Pneumonia
Acute respiratory distress syndrome and pneumonia are predominantly alveolar diseases with respiratory distress with or without fever affecting oxygenation as well as ventilation.
Emergency department management includes quick history and rapid cardiopulmonary assessment. The following steps should be taken in the emergency department:

- Place pulse oximeter and begin oxygen therapy.
- Consider endotracheal intubation and initiation of mechanical ventilation if indicated by clinical deterioration (or arterial blood gases).
- Portable chest X-ray is done to confirm alveolar disease (diffuse infiltrates/consolidation usually with normal size heart).
- Complete blood count, blood culture, coagulation profile, electrolytes, urea creatinine and liver functions are obtained.
- Intravenous line is secured; if not successful, then intravenous access is obtained.
- Fluid bolus is administered if patient is in shock as indicated by poor capillary refill and other parameters.
- Intravenous antibiotics are administered.
- If hypotension and poor perfusion is seen despite fluid therapy, consider dopamine at 10 µg/kg/min and central venous pressure (CVP) monitoring.
- Initiate transfer/arrange transport to the nearest PICU by discussing with the pediatric intensivist.

**Status asthmaticus (see Chapter 11)**

- High flow oxygen
- Steroids: IV hydrocortisone or IV methylprednisolone
  - Inhaled beta agonist; salbutamol of choice, no added benefit of levosalbutamol clinically (though causes less tachycardia compared to salbutamol)
  - Intravenous and subcutaneous beta agonist—terbutaline
  - Methylxanthines—theophylline (to be used when no response to steroids, inhaled and IV beta agonist)
  - Anticholinergic—inhaled ipratropium bromide
  - Magnesium sulphate
  - Heliox (if available).

**Indications for intubation**

- Cardiorespiratory arrest
- Refractory hypoxemia
- Significant respiratory acidosis unresponsive to pharmacotherapy
- Rapid deterioration in mental status

**Tension Pneumothorax**

This is a medical emergency and should be promptly recognized and treated. Severe respiratory distress with shock occurs due to decreased intrathoracic venous return caused by tamponade effect of air leak from lungs under pressure compressing the heart. These result in acute fall in cardiac output as indicated by hypoxemia, poor perfusion and hypotension. If left untreated, it can result in cardiopulmonary arrest. Clinically, one finds absent or low breath sounds on the affected side as well as muffled heart sounds and shifted apical impulse. Chest
X-ray shows mediastinal shift as well as compression with free air in the pleural cavity depressing the dome of diaphragm. Chest X-ray may take time, therefore one should not wait for chest X-ray before intervention.

Chest needling on the suspected side with 16–18 gauge IV cannula or scalp vein needle may be attempted in second intercostal space anteriorly in midclavicular line to relieve tension pneumothorax.

However, once the chest X-ray is obtained, the patient will require tube thoracostomy on the affected side.

**Neuromuscular Disorders**

The commonly seen disorders are briefly discussed below.

**Central hypoventilation syndrome**: Central hypoventilation may be congenital or acquired. Congenital form (Ondine’s curse) typically presents as cyanosis at birth readily responsive to mechanical ventilation (but not to oxygen therapy alone) with normal chest radiographs but repeated weaning failures. In less severe cases, abnormalities in respiration during sleep, such as periodic breathing, apnea or acute life-threatening episodes are reported. This must be differentiated from reversible systemic processes such as sepsis, hypothermia, electrolyte abnormalities, hypocalcemia and seizures, CNS infections, intracranial hemorrhage and acute hydrocephalus. It also needs to be distinguished from obstructive sleep apnea (OSA) due to hypertrophied tonsils and adenoids, macroglossia (Down’s syndrome), micrognathia (Pierre Robin syndrome), other oral or nasal congenital anomalies, temporomandibular ankylosis, vascular ring and vocal cord paralysis, and post cleft palate surgical repair.

Therapy is immediate ventilatory support if in acute respiratory failure. Doxapram (a central respiratory stimulant) theophylline and caffeine have been tried to get a better apnea free respiratory effort.

Tracheostomy with home mechanical ventilatory assistance during sleep is commonly required. Recently, noninvasive methods, such as use of nasal mask continuous positive airway pressure (CPAP) or noninvasive positive pressure ventilation (NIPPV) have been shown to be very effective, and need for tracheostomy can be averted.

**Guillain–Barré syndrome (acute postinfectious polyneuritis)**: This condition commonly presents as a postviral immune-mediated paralysis affecting skeletal muscles as well as autonomic nervous system leading to profound muscle weakness, ascending in nature with paresthesias.

Diaphragmatic and intercostal muscle weakness leads to neuromuscular respiratory failure requiring mechanical ventilation in 20% of affected children. Therefore, frequent assessment of respiratory reserve is necessary. Concern for respiratory failure if forced vital capacity (FVC) falls below 15–20 ml/kg, maximum negative inspiratory pressure less than 20–30 cm H₂O and pCO₂ greater than 50 mmHg.

Cranial nerve palsy and/or cerebellar ataxia may be first presenting feature. More than 10% patients present with upper extremity weakness. Cerebrospinal
fluid (CSF) protein level is usually elevated greater than 45 mg/100 ml in absence of pleocytosis (cytoalbuminoid dissociation).

Electromyography (EMG) shows evidence of lower motor neuron disease, and nerve conduction velocity is delayed.

Bladder and bowel dysfunction is common with hypertension due to effect on autonomic nervous system. Sinus tachycardia, bradycardia, ST and T wave abnormalities and postural hypotension may be seen.

Early plasmapheresis for removing autoimmune factors within 7 days of onset of disease has been found to be beneficial. Gamma globulin therapy has demonstrated to be more effective when compared to plasmapheresis. However, the mainstay of treatment remains supportive.

Prognosis is usually good with recovery in 3–4 weeks. However, muscle power and neurological recovery may be incomplete and prolonged in a few cases.

**Unilateral phrenic nerve paralysis:** This condition results from birth trauma to phrenic nerve and usually presents with respiratory distress in infancy. Fluoroscopy of the diaphragmatic motion is diagnostic. Diaphragm moves upward with inspiration. Usually adequate gas exchange can be maintained with CPAP alone or institution of mechanical ventilation; however, long-term management will require surgical plication of affected diaphragm.

Other important causes of phrenic nerve injury include direct phrenic nerve injury during open heart surgery and aortopulmonary shunt procedures. Half the cases occur during closed heart surgery such as during pulmonary artery banding and patent ductus arteriosus ligation.

These patients are usually detected when the subject fails to wean from the mechanical ventilation. Transcutaneous phrenic nerve stimulation can be applied in cervical region, and if response is seen, a plication surgery of diaphragm may be avoidable, especially in older child.

**Poliomyelitis:** Poliomyelitis represents an acute viral infection of the CNS that results in widespread muscle paralysis due to involvement of anterior horn cells and secondary respiratory failure.

Minor febrile illness, upper respiratory infections or gastroenteritis begins lasting for 1–2 days. In less than a week later, severe muscle pain, fever, irritability, paresthesias, muscle fasciculation and diminished deep tendon reflexes in affected muscles are seen. In some cases, it rapidly progresses to total paralysis. Cerebrospinal fluid shows mild pleocytosis with polymorphs in early course and mononuclear cells in later phase. Causative virus can be isolated from fecal and oropharyngeal specimens. Serological confirmation is made by identifying specific antibodies to poliovirus.

Bulbar palsy can result in loss of airway control due to pharyngeal muscle paralysis and can lead to airway obstruction and aspiration of pharyngeal secretions. Endotracheal intubation, mechanical ventilation and chest physiotherapy remain the key treatment modalities.

Tracheostomy is often required. Intensive care unit survival is good. However, the pediatric mortality can exceed to 30% if good ICU facilities are not available. Chronic respiratory insufficiency can result from vocal cord paralysis, scoliosis, secondary respiratory restriction and central hypoventilation.
Spinal cord trauma: High cervical injuries (C3 and C5) result in loss of diaphragmatic, intercostal and abdominal muscle function. Accessory muscles in neck and shoulder remain intact. Intubation and ventilation are invariably required. As pointed out earlier, other accompanying chest and lung injuries contribute to severity of respiratory failure. Tracheostomy and long-term mechanical ventilation are usually required. In patients with intact nerve conduction, phrenic nerve radiofrequency electrophrenic pacing has been tried.

Summary

Approach and management of acute respiratory failure is summarized in Figure 6.2. Early recognition and urgent institution of treatment is of paramount importance in children. This is due to low respiratory reserve and propensity to bradycardia in pediatric age group and cardiac arrest is usually secondary to hypoxemia as well as due to increased vagal tone. Airway control and oxygen should be used as a first measure. Indications of instituting mechanical ventilation are clinical and not entirely dependent on blood gases, as blood gases may be normal in early respiratory failure. Noninvasive ventilation should be tried first if available (except in uncooperative comatose patient or a patient with poor airway reflexes as well as in patient with ARDS). Prolonged ventilation in neuromuscular disorders may require tracheostomy. If detected early, acute respiratory failure is a treatable condition with mortality and morbidity related to primary cause and secondary complications as a result of prolonged mechanical ventilation in the PICU.
Respiratory Failure

Fig. 6.2: Management algorithm of respiratory distress/failure

**Abbreviations**: IVIG, intravenous immunoglobulin; FiO₂, fraction of inspired oxygen; ARDS, acute respiratory distress syndrome; PICU, pediatric intensive care unit; ABG, arterial blood gas; BAL, bronchoalveolar lavage
Respiratory Failure: Mechanical Ventilation Algorithm

**Indications for mechanical ventilation**
- Respiratory arrest
  - Inadequate ventilation: $\text{PCO}_2 > 55$
  - Inadequate oxygenation: $\text{PO}_2 < 60$
- Chronic respiratory failure
- Cardiac insufficiency/shock
- Neurologic dysfunction
  - Central hypoventilation
  - Frequent apnea
  - GCS < 8
  - Inability to protect airways

**Initial Ventilator Settings**
- $\text{FiO}_2$: 100%
- Rate: 30–40 for neonates, 20–30 for infants and small children, older child and for adolescents
- Inspiratory time: (1×: 1.2–1.5)
  - Neonates: 0.3–0.4 s
  - Small children: 0.5–0.6 s
  - Older children: 0.7–0.9 s
- PEEP: 3–5 cm $\text{H}_2\text{O}$
- Pressure control
  - PIP
  - Neonates: 16–22 cm $\text{H}_2\text{O}$
  - Children: 18–26 cm $\text{H}_2\text{O}$ (Adequate chest rise)
- Volume control
  - Tidal volume: 6–10 ml/kg (Adequate chest rise)

**Rapid Sequence Intubation**
- Secure the airway rapidly and prevent aspiration of gastric contents.

**Steps**
- Preparation
- Preoxygenation
- Pretreatment
- Paralysis with induction
- Protection and positioning
- Placement with proof
- Positiation management

**Tube Placement** (Use Sellick’s maneuver)
- Confirm tube placement
  - ETCO$_2$/breath sounds
  - Adequate chest rise
  - Secure tube
  - Chest X-ray
- Recheck vital signs
- Heart rate
- Blood pressure
- Medication
  - Continued paralysis
    - Vecuronium: 0.1 mg/kg/hour
  - Sedation
    - Benzodiazepines/opioids

**Adjuncts after intubation**
- Tidal volume or PIP should be sufficient to make the chest move adequately
- Check for adequacy
  - Bilateral air entry
  - Chest rise and SpO$_2$
- Check ABG
  - To improve oxygenation: Adjust $\text{FiO}_2$, PEEP, inspiratory time, PIP, VT (rapidly bring down to lowest tolerable level)
  - To improve ventilation: Adjust respiratory rate, PIP/VT

**Management of Low PAO$_2$**
- Do not just increase $\text{FiO}_2$
  - Increase inspiratory time
  - Increase VT/PIP
  - If O2 woresen, get chest X-ray to rule our air leak/pneumothorax
  - Optimize cardiac output: fluid/inotrope
  - Keep hemoglobin > 9 mg/dl
  - Maintain normothermia
  - Rule out tube obstruction/suction
  - Defer sedation/consider paralysis

**Management of high PACO$_2$**
- Increase PIP/volume
  - Increase frequency
  - Decrease dead space
  - Decrease PEEP
  - Increase cardiac output
- Decrease CO$_2$ production
  - Bring down temperature, sedate, decrease carbohydrate load
- Rule out blocked/misplaced ET tube
- Fix leak in the circuit, endotracheal tube cuff, humidifier

**Measures to Minimize Barotrauma**
- Permissive hypercapnia
  - Tolerate higher pCO$_2$ to limit PIP as long as pH > 7.2
- Permissive hypoxia
  - Tolerate PaO$_2$ of 55–65, SaO$_2$ 88–90% in exchange for limiting FiO$_2$ < 60% and to minimize PEEP requirements as long as there is no metabolic acidosis

**Sudden Desaturations/Patient Ventilator Asynchrony**
- Use “DOPE” mnemonic:
  - D: Displacement of tube
  - O: Obstruction of tube
  - P: Pneumothorax
  - E: Equipment failure
  - Check tube placement if in doubt, take tube out and ventilate manually with 100% $\text{O}_2$
  - Examine patient: is the chest rising? Breathing sounds present and equal?
  - Look for atelectasis, bronchospasm, pneumothorax

**Abbreviations:**
- $\text{PaO}_2$: partial pressure of oxygen
- $\text{PCO}_2$: partial pressure of CO$_2$
- FTT: failure to thrive
- GCS: Glasgow Coma Scale
- ETCO$_2$: end-tidal CO$_2$
- $\text{FiO}_2$: fraction of inspired oxygen
- PEEP: positive end-expiratory pressure
- PIP: peak inspiratory pressure
- SPO$_2$: oxygen saturation
- ABG: arterial blood gas
- VT: tidal volume
- ET: endotracheal tube
- SaO$_2$: arterial oxygen saturation
Indications for Mechanical Ventilation

- **Respiratory failure**: Apnea or pulmonary arterial oxygen tension (PaO₂) < 60 mmHg or
- **PaCO₂ > 50 mmHg** (acute and unresponsive to other measures).
- **Shock**: Usually, requirement of more than 60 ml/kg of fluid boluses or with respiratory failure or with altered sensorium to decrease the work of breathing.
- **Altered sensorium** [Glasgow Coma Scale (GCS) < 8] with central hypoventilation or inability to maintain or protect airway.
- **Raised intracranial pressure** (ICP) unresponsive to first-line measures.

Contraindications for Noninvasive Ventilation

- Unable to protect airway
- No spontaneous breathing
- Severe oronasal bleeding, excessive secretions
- Noncooperative patient, agitation, fighting the NIV mask
- Poor sensorium
- Facial trauma, severe facial deformities that prevent good mask fit
- Morbid obesity
- Hemodynamic instability.

Mode of Ventilation

Controlled mandatory ventilation (CMV) mode/intermittent mandatory ventilation (IMV) or synchronized intermittent mandatory ventilation (SIMV) mode/pressure support ventilation (PSV)-CPAP mode depending on the triggering (who starts), cycling (which ends inspiration) mechanism and synchronization.

**Pressure limited/volume limited**: The flow pattern is different for both modes. Pressure-limited ventilation has a decelerating flow pattern while volume-limited ventilation has rectangular wave flow pattern. For delivering the same tidal volume (Vt), pressure-limited ventilation requires less peak pressure than volume-limited ventilation. Pressure-limited ventilation provides higher MAP.
(improves oxygenation) and maintains a higher mean lung volume compared with volume-limited ventilation.

Pressure-limited ventilation is said to be better for children with abnormal lung as it gives less barotrauma and cause less variations in peak pressures. In case of normal lung, either volume limited or pressure limited can be tried. While using pressure-limited ventilation, adequate Vt should be ensured and while using volume-limited ventilation, care should be taken in limiting plateau pressures.

_Dual mode [pressure-regulated volume control (PRVC) and other modes]_ combines the benefits of both pressure-limited ventilation (decelerating flow) and volume-limited ventilation (ensures Vt by servo feedback) can be used.

**Initial Settings**

**Pressure Limited**
- **FiO2**: Start with one and wean down to get good SpO₂ (> 90%).
- **Optimal positive end expiratory pressure**: To achieve adequate alveolar recruitment so that saturations are maintained at minimum FiO₂ (< 0.6) and compliance is best with no hemodynamic compromise.
- **Respiratory rate**: Normal for age (adjust if there is air trapping).
- **Pressure control above PEEP (positive end expiratory pressure)**: So that Vt equal to 6–8 ml/kg is delivered with adequate chest rise.
- **Inspiratory time**: Normal for age, 0.4 second for neonates, 0.5–0.6 second for infants, 0.6–0.8 second for toddlers and 0.9–1 second for older children [adjust in case of air trapping or severe acute respiratory distress syndrome (ARDS)]

**Volume Limited**
- **Tidal volume**: 6–8 ml/kg, look for adequate chest rise.
- **Inspiratory time**: Normal for age (adjust in case of air trapping or severe ARDS).
- **Optimal positive end expiratory pressure**: Adequate alveolar recruitment so that saturations are maintained at minimum FiO₂ (< 0.6) and compliance is best with no hemodynamic compromise.
- **Respiratory rate**: Normal for age (adjust if there is air trapping).
- **FiO2**: To get good SpO₂ (> 90%).

**Sedation and Analgesia**
- Most patients can be managed with adequate sedation without muscle relaxant. Muscle relaxants should never be started without adequate sedation.
- Midazolam and morphine is used for most of the cases. In case of specific scenarios, other agents can be tried.
- Morphine is not preferred in hemodynamic instability and wheezing. Ketamine is preferred instead.
- Midazolam is not preferred in severe hemodynamic instability and liver failure.
Ketamine is preferred in shock and wheezing. Ketamine is not used in raised ICP. Alternate agents for sedation are thiopentone, phenobarbitone, diphenhydramine, triclofos, oral diazepam, etc.

Changes in Settings according to Arterial Blood Gas

- **Poor oxygenation:** Check Displaced tube (usually right mainstem, pyriform fossa, etc.), Obstruction (kinked or bitten tube, mucous plug, etc.), Pneumothorax (collapsed lung), Equipment failure (DOPE), increase $\text{FiO}_2$, optimize PEEP, increase PIP or Vt if chest rise not adequate, increase inspiratory time (Ti) (may try inverse ratio ventilation if severe ARDS).
- **High $\text{PaCO}_2$:** Ignore if pH acceptable (> 7.2) unless there is increased ICP or severe pulmonary hypertension (permissive hypercapnia). To decrease $\text{PaCO}_2$, increase rate or increase PIP or Vt. In bronchospasm, decrease rate and increase expiratory time to prevent air trapping.
- **High $\text{PaO}_2$:** Decrease $\text{FiO}_2$
- **Low $\text{PaCO}_2$:** Decrease rate, decrease Vt if chest rise excessive.

Disease Specific Ventilation

**Status Asthmaticus**

- Main indications are clinical deterioration despite maximal drug therapy.
- Rising $\text{PaCO}_2$ (40–45 mmHg) from a low $\text{PaCO}_2$ (25–30 mmHg)
- Fatigue, lethargy, deteriorating mental status
- Mixed respiratory and metabolic acidosis.

Initiation of Ventilation

- Controlled intubation, use sedation and muscle relaxation (short-acting muscle relaxant such as succinylcholine)
- Use cuffed endotracheal tube (ETT) if feasible
- Ketamine with midazolam are good sedatives for initiation and maintenance of mechanical ventilation.
- Mechanical ventilation in asthma is associated with high morbidity and mortality.
- Risks involved barotrauma (air leak) due to dynamic hyperinflation, impaired venous return (tamponade) and low cardiac output due to hyperinflation (pulsus paradoxus). Strategies that minimize end expiratory volume, intrinsic positive end expiratory pressure (PEEP), and maximize expiratory time, using lower tidal volumes and respiratory rates with permissive hypercapnia have been shown to be associated with lower mortality.

Ventilation Strategies

**Controlled Hypoventilation using SIMV or Assist Control**

- Volume or pressure limited mode
- Plateau pressure limit must be < 35 cm H$_2$O
- Tidal volume 6–10 ml/kg f 8–14/min, PEEP 3–4 cm H$_2$O.
Support Modes: Volume or Pressure Support

- Patient determines (f) rate and Ti
- Plateau pressure limit set at < 35 cm water
- Tidal volume 6–10 ml/kg
- Patient can determine the respiratory cycle, frequency and flow pattern.
  - There is active exhalation and decreased end expiratory lung volume
- As plateau pressure falls, barotrauma decreases.

Use of Positive End-Expiratory Pressure

In conscious patient not on muscle relaxants, on assist control mode, PEEP can be used in physiological levels (3–4 cm H₂O). Application of extrinsic PEEP (at levels less than intrinsic PEEP) helps decrease work of breathing and easier patient triggering.

Controlled Hypercapnia

Both pressure control or volume control can be used as long as plateau pressures do not exceed 35, rates are at 8–4 [inspiration:expiration (I:E) ratio of 1:3 or more], use of prolonged expiration to avoid intrinsic PEEP and delivered Vt 6–10 ml/kg. In volume control mode PIP may go too high. Advantage of pressure control mode is that decelerating flow delivers volume at a lower inspiratory pressure. Strategies of permissive hypercapnia and permissive hypoxemia are generally acceptable to minimize barotrauma and air leak.

Case Scenario 1

A 10-year-old boy weighing 25 kg comes to the emergency with history of fever and cough for 2 days and has developed respiratory distress for past 3 hours. He is a known case of bronchial asthma on regular fluticasone inhaler but has stopped his inhalers for the past 10 days.

On examination, he is drowsy and lethargic. His heart rate is 130 beats/minute, respiratory rate is 40 breaths/minute with severe intercostal and subcostal retractions with nasal flare. His BP is 100/74 mmHg, peripheral pulses are feeble and capillary fill time (CFT) is 4 seconds. He is dusky and saturating 84% in air which increases to 92% in 100% oxygen. On auscultation, he has bilateral poor air entry and a CXR done of him reveals bilaterally hyperinflated lung fields. He was immediately intubated and ventilated for impending respiratory failure. He was initiated on pressure regulated volume control (PRVC) mode of ventilation with the following settings:

- Tidal volume 150 ml (6 ml/kg)
- FiO₂ 100%
- Rate 12
- I:E ratio 1:3
- PEEP 3
His ABG on the above settings was:
- pH 7.15, pCO₂ 90 mmHg, pO₂ 96 mmHg, HCO₃⁻ 20.1 mEq/L, saturation 99%
- and base excess (BE) –6.

Following this, rate was decreased to 8 and FiO₂ to 80% such that his I:E ratio increased to 1:4. The repeat ABG revealed a pH of 7.26, pCO₂ 72 mmHg, pO₂ 76 mmHg, HCO₃⁻ 23 mEq/L, saturation 96% and BE –2. He was continued on the same settings allowing for some permissive hypercapnia as discussed earlier.

**Ventilation for Acute Respiratory Distress Syndrome**

Indications for ventilation in ARDS are essentially based on clinical evidence of hypoxemia, and include:
- Increasing respiratory distress
- Tachypnea, tachycardia, accessory muscle use (in late stage apnea/bradycardia)
- Increasing oxygen requirement with desaturation on maximal oxygen
- Respiratory fatigue/lethargy
- To reduce work of breathing.

**Goals of Ventilation in Acute Respiratory Distress Syndrome**

Ventilation should be delivered with minimal volutrauma to lungs (using low tidal volumes, i.e. 6–8 ml/kg), minimal tolerable inspired oxygen with PEEP to achieve PaO₂ 55–80 mmHg and maximal tolerable arterial pCO₂ (50–60 mmHg) with arterial pH > 7.25 (permissive hypercapnia) and absence of metabolic (hypoxic) acidosis. Conventional ventilation is the most readily available modality. Earlier standard approach used was volume ventilation with tidal volumes 10–15 ml/kg with PEEP. Adequate filling pressures with use of fluid and good cardiac contractility with inotropic support to prevent low cardiac output are required. Problems with conventional 10–15 ml/kg Vt and PEEP are as follows:
- Barotrauma, volutrauma, air leak (pneumothorax), chronic lung disease, delayed recovery, poor cardiac output, prolonged ventilation and nosocomial infections.

In view of problems with conventional Vt ventilation, low Vt strategy is recommended (Source: NIH ARDS Network Study).

This was a prospective randomized multicenter trial of 240 patients with two groups using 12 ml/kg versus 6 ml/kg Vt, PEEP 5–18 cm of H₂O, FiO₂ 0.3–1, showed 25% reduction in mortality in 6 ml/kg group. In another study, use of higher PEEP with lower tidal volumes (open lung approach) has been used with improved results.Gattinoni et al, in adults and Marraro in pediatrics ARDS patients showed that chest computerized tomography (CT) may be useful to see the extent of pulmonary involvement. Gattinoni studied benefits of prone positioning in patients with ARDS with underventilated posterior zones. Prone positioning is being recommended although transient improvement in oxygenation occurs but no real effect on improving long-term outcomes has been shown. Problems
associated with prone positioning include difficulty in nursing management and monitoring (chances of accidental extubation, especially during X-ray examination, and physiotherapy).

Currently, no good pediatric studies are available on use of prone positioning in ARDS patients and the effect on outcomes.

**Case Scenario 2**

A 5-year-old premorbidly well child weighing 15 kg comes to emergency with 3 days of moderate to high grade fever and cough. He has been lethargic for the past 1 day and is not feeding well. Mother noticed that he is breathing fast since morning and has become dusky and unresponsive for the past 10 minutes. On examination, he is unresponsive with a heart rate of 140 beats/minute, respiratory rate 60 breaths/minute with retractions and head bobbing. He is peripherally cyanosed, saturating 80% in air and saturation slowly increasing to 88% in 100% oxygen. Auscultation reveals bilateral extensive crepitations. The CXR is suggestive of ARDS with bilateral diffuse infiltrates in lungs.

He was intubated and ventilated on PRVC mode. His initial settings were:
- Tidal volume 90 ml (6 ml/Kg)
- FiO₂ 100%
- Rate 25
- I:E ratio 1:2
- PEEP 6

His saturations improved to 85% on the above settings and an ABG done showed pH 7.30, pCO₂ 45 mmHg, pO₂ 47 mmHg, HCO₃ 20.4 mEq/L, BE –5 and saturation 85%. To improve his oxygenation, his PEEP was increased to 8 and I:E ratio to 1:1. Following the intervention, his saturations improved to 95%.

**Air Leak Syndrome**

_Pneumothorax, Bronchopleural Fistula_

Ventilation for air leak syndrome is challenging. Chest tubes are frequently required.

**Ventilation Strategies**

Using low MAP, low peak inspiratory pressures, low PEEP, lower tidal volumes, and lower inspiratory times are needed.

**Other Modes Useful in Air Leak Syndrome**

High frequency oscillatory ventilator delivers small tidal volumes at high frequency with lower peak and mean airway pressures.

Patient has to be muscle relaxed.

Patient cannot be suctioned frequently as disconnecting the patient from the oscillator can result in volume loss in the lung.
Likewise, patient cannot be turned frequently so decubitus ulcers can occur. Patient should be turned and suctioned 1–2 times/day if he/she can tolerate it.

Postoperative Ventilation following Open Heart Surgery

General Principles
One needs to understand the cardiac physiology associated with the lesion and corrective surgery as well as cardiopulmonary interactions in the postoperative period.

Hypoxia and hypercarbia should be avoided to prevent pulmonary hypertension that increases right ventricular afterload/chances of right ventricular failure.

Excessive systemic vasoconstriction should be avoided to prevent increase in left ventricular (LV) afterload.

Volume/pressure limited ventilation: Mode of ventilation has not shown to make any real difference in outcomes.

Excessive PEEP and excessive mean airway pressures should be avoided to prevent tamponade/low cardiac output.

Pulmonary and systemic vascular resistance can increase with pain causing increased afterload on the heart.

Consider nitric oxide in patients with severe preoperative pulmonary hypertension in postoperative period.

Chronic Lung Disease/Neuromuscular Weakness
Tracheostomy is usually performed.

One needs to assess need for day/night/home ventilation

Generally low ventilator settings are needed. LP60 (USA) pressure controlled ventilator can be used.

Noninvasive positive pressure ventilation can also be tried to deliver pressure support (PS) and CPAP via tight-fitting mask (BiPAP). One can set a “back up” rate in case of apnea.

Case Scenario 3
A 13-year-old immunized female child weighing 30 kg was admitted with complaints of sudden onset weakness of lower limbs with inability to stand and bear weight for 2 days. The next day she developed weakness of both upper limbs such that she could only move her arms in the bed. She started to have decreased volume of voice and complained of some tingling sensation in both legs. There was no history of fever, cough, loose stools, trauma and alteration in sensorium or seizures. She had an episode of fever with cough 2 weeks back which lasted for 3–4 days.
On examination, she was alert, conscious and oriented. Her HR was 110 beats/minute and respiratory rate 30 breaths/minute. She had shallow respiratory efforts with paradoxical respiration. CNS examination revealed quadriplegia with power in both lower limbs and upper limbs being 1/5 and 2/5 respectively. She had global areflexia. There was no objective sensory loss and no other focal deficits. She was diagnosed to have Guillain-Barré syndrome with respiratory muscle weakness supported by nerve conduction velocity (NCV) findings. She was ventilated for neurogenic cause of respiratory failure on PRVC mode of ventilation with the following settings:

- Tidal volume 200 (6–7 ml/kg)
- Rate 15
- FiO₂ 40%
- PEEP 3
- I: E ratio 1:2

His ABG on the above settings was within normal limits.

**Raised Intracranial Pressure**

Following points should be kept in mind.

- Avoid ketamine, succinylcholine as these agents raise ICP.
- Midline head up position is ideal.
- Adequate sedation and muscle relaxation is required to prevent coughing and bucking on the ventilator (leads to raised ICP) and adequate analgesia during painful procedures
- Low PEEP (avoidance of excessive PEEP) to prevent ICP from going up.
- Goal of ventilation to keep normal PaO₂ and PaCO₂ 30–35 mmHg; hyperventilation is no longer recommended.

**Weaning**

Ventilatory settings are reduced once the primary pathology or condition that led to ventilation is improving. There are no set protocols for weaning. Different protocols are followed by different institutions. Generally, the following pattern is adopted:

- FiO₂ is weaned first to 0.4, maintaining saturation in acceptable range.
- Mode is changed to SIMV with pressure support mode.
- PEEP is decreased gradually in steps of 2 cm H₂O to 4–5 cm H₂O.
- SIMV rate is decreased to 5–10 breaths/minute.
- Patient is reassessed after each change in the settings and if the oxygen requirement goes up or patient develops respiratory distress or hypercarbic on blood gas, weaning process is paused and support level increased.

Some patients (especially when the lung is normal or short ventilation for neurological indications) can be directly given a spontaneous breathing trial after stopping sedation and extubated without weaning.
Spontaneous Breathing Trial
Spontaneous breathing trial is done before extubation to assess extubation readiness. It can be done with a T-piece after disconnecting ventilator, endotracheal CPAP, or minimal pressure support with CPAP. Usually the pressure support level is adjusted to the size of ETT (6 cm H₂O PS for ETT > 5 mm, 8 for ETT 4–5 and 10 for ETT 3–4). Duration of the trial ranges from 30 minutes to 2 hours. Following are the criteria for terminating a spontaneous breathing trial (SBT):
- Inability to maintain gas exchange (needing more than 0.5 FiO₂ for saturations greater than 95%)
- Inability to maintain effective ventilation (measured exhaled Vt < 5 ml/kg; PaCO₂ > 50 mmHg or increase > 10 mmHg)
- Increased work of breathing (tachypnea or use of accessory muscles or paradoxical breathing pattern)
- Other signs of distress (diaphoresis, anxiety, rise in HR, change in mental status, hypotension).

If the patient tolerates the SBT, we can proceed to extubate.

Extubation
The following criteria to be met before extubation:
- Presence of airway reflexes, manageable secretions.
- Minimal oxygen requirement < 0.4 and PEEP < 5 with saturations above 94%
- Good spontaneous Vt with minimal pressure support (5–10 above PEEP depending on the tube size) during SBT.
- Alert or easily arousable.
- Nil orally for at least 4 hours before extubation.
- Hemodynamically stable (dopamine requirement < 5 µg/kg/min)
- PaCO₂ < 50 mm Hg
- pH 7.3–7.47
- Core temperature below 38.5°C.
- Leak around the ETT is good but not a prerequisite for extubation
- No major metabolic derangements.

Injection dexamethasone 0.2 mg/kg q 6 hours can be given prior to extubation, the first dose given 12 hours before extubation. It can be continued 48 hours after extubation. It decreases postextubation stridor.

Indications for HFOV
- Requiring MAP > 20 mmHg
- Not maintaining saturations with PEEP > 14 mmHg, FiO₂ > 0.6
- Oxygenation index (OI) > 16 (OI = MAP x FiO₂ x 100/PaO₂).
Abbreviations: NIV, noninvasive ventilation; CPAP, continuous positive airway pressure; BiPAP, bi-level positive airway pressure; SpO₂, pulse oximeter oxygen saturation; HR, heart rate; BP, blood pressure; ABG, arterial blood gas; CXR, chest X-ray; HFOV, high-frequency oscillatory ventilation; FiO₂, fraction of inspired oxygen; MAP, mean airway pressure; PaCO₂, arterial carbon dioxide tension.
Further Reading

Weaning is the word used to describe termination of mechanical ventilation (MV). Most children can be easily weaned from the ventilator without either delay or significant problems. Only a small group of children, usually those with underlying chronic pulmonary disease, neurologic disease, or malnutrition are difficult to wean.

Prolong ventilation increases risk of ventilator-associated pneumonia (VAP) and reintubation in view of early extubation is poor prognostic marker. Thus, the value of removing the ventilator as soon as possible must be balanced against the risks of premature withdrawal.

The gradual transition from full or almost full mechanical support to spontaneous breathing may be accomplished by gradually decreasing the mandatory breath rate with synchronized intermittent mandatory ventilation (SIMV), the level of PEEP, and/or the degree of pressure or volume support.

**Weaning Strategy**

**Step 1**

The best approach for all patients is to question (perhaps several times) every day:

- Why are they receiving MV?
- Do they require the current levels of support?
- Do they actually still need to be ventilated?

When a child is on MV the process of weaning starts right after intubation.

- The primary reason for intubation should be sorted out and once sorted the child can be weaned down and extubated (e.g. if the child is intubated for altered sensorium due to drug overdose, manage the drug overdose and once the sensorium improves wean down and extubate).
- If the child is on ventilator for a severe lung injury (e.g. acute respiratory distress syndrome (ARDS)), and is on “high peep and low tidal volume strategy,” continue on optimal PEEP, where adequate recruitment is achieved and start reducing the FiO₂ to the range of 0.5–0.6. Once there, then looking at the
Weaning from Mechanical Ventilation

overall situation and accepting higher arterial carbon dioxide tension (PaCO₂), pH > 7.25, pulse oximeter oxygen saturation (SpO₂) of >88% and pulmonary arterial oxygen tension (PaO₂) of > 60 mmHg, start reducing the pressures.

- If the child is ventilated for low cardiac output states (e.g. myocarditis), start weaning down once the cardiovascular status improves.

**Step 2**

*Relative contraindications for weaning and extubation:*

- Levels of PaO₂: < 60 mmHg, where FiO₂: >0.6
- In addition, significantly increased respiratory rate or reduction in tidal volume (or particularly a combination of both) during spontaneous breathing strongly suggests that the patient is not ready for extubation.

**Step 3**

*Readiness for weaning and extubation:*

Generally speaking, once the child is maintaining PaO₂ of > 60 mmHg at FiO₂ of < 0.6, and requiring PEEP of < 6 cm H₂O, and stable at delivered tidal volume of 4–6 ml/kg, then the child is ready for weaning trial and extubation.

Generally, before discontinuation of MV, patients should be hemodynamically stable, alert, and capable of protecting their airway (i.e. adequate cough and gag) and have no severe metabolic abnormalities that may affect their work of breathing or muscular strength.

**Step 4**

Sedation should be reduced carefully in order not to compromise both the respiratory drive and patient comfort or to precipitate drug withdrawal. During this period, gas exchange and breathing pattern should be assessed.

There are no well-established methods to predict successful extubation in children, and it is unfortunately impossible to mimic breathing without an endotracheal tube (ETT) unless an extubation is performed.

Purely protocol-directed extubation strategies have yielded inconsistent results. However, it appears that, when clinical standards are reasonable, protocol-directed weaning regimens offer no advantage over usual practice for the weaning of either adults or children.

**Step 5**

*Weaning mode of ventilation*

Unfortunately, the optimal mode of MV used during weaning remains controversial.

Daily T-piece trials consistently have been superior to the SIMV mode in weaning, and at least equivalent to pressure support ventilation (PSV) weaning.
Pressure support ventilation provides a progressive unloading of inspiratory muscles compared with SIMV. The results of trials with PSV, however, have been variable.

Noninvasive positive pressure ventilation (NPPV) also has been used as a method to support ventilation following early extubation. A clinical study showed that this technique of postextubation NPPV compared with standard oxygen therapy, averted respiratory failure after extubation and decreased intensive care unit (ICU) mortality among patients at increased risk.

A trial of spontaneous breathing with assessment of the gas exchange and pattern of breathing with minimal pressure support (10 cm H$_2$O for ETT size up to 3.5 mm, 8 cm H$_2$O for ETT size up to 4.5 mm and support of 6 cm H$_2$O for ETT size 5 mm and above) or T-tube without pressure support appears to be equally useful approaches in order to evaluate readiness for extubation.

**Step 6**

Although there is lot of heterogeneity in the study groups and more studies are required to prove conclusively, but it is becoming clear that children and neonates with high risk of postextubation stridor (those with difficult intubation, ventilated for more than 5–7 days, neck surgeries, failed one extubation, etc.) benefit by use of steroids (dexamethasone) starting 12–24 hours before extubation.

The administration of inhaled racemic epinephrine or heliox or application of NPPV for a short period of time may decrease the rate of reintubation in cases of postextubation stridor.

Weaning failure is failure of the patient to maintain ventilation and oxygenation when the ventilatory support is reduced. Extubation failure assumes that the patient has in fact been successfully weaned from ventilatory support and that it is the extubation (i.e. later phase) not the weaning (i.e. earlier phase) that has not been successful. In general, a need for reintubation for any reason within 24 hours after elective extubation is termed extubation failure.

For adults, the rates of reintubation range from 2% to 20% and are similar for children.

Major risk factors associated with extubation failure in children are:

- Young age (i.e. < 3 years)
- Duration of ventilation
- Severity of underlying lung disease
- Oxygenation impairment (i.e. oxygenation index > 5)
- Intravenous sedation.
### Suggested Weaning Strategy from Mechanical Ventilation

#### Treat the primary cause for ventilation (sepsis, poisoning, myocarditis, etc.)

If child is ventilated on high PEEP, do not wean PEEP first as it can derecruit alveoli.

Reduce FiO₂ slowly keeping SpO₂ (> 88%), pH (> 7.25, PaO₂ (> 60 mmHg)

At 0.5–0.6 FiO₂, start reducing PEEP and ventilator rate accepting higher PaCO₂ keeping pH (> 7.25

At FiO₂ 0.4–0.5, and PEEP of < 6 cm H₂O reduced sedation, no paralysis, and shift over to SIMV-pressure support mode

#### Start spontaneous breathing trial (SBT):
Stop any feeds, stop sedation, after a good chest physiotherapy and endotracheal (ET) suction, place the child on PSV mode or on T-piece trial. At FiO₂ of 0.3–0.5, as smaller size ETT causes more airway resistance, use appropriate pressure support (10 cm H₂O for ET size up to 3.5, 8 for ET size up to 4.5 and support of 6 cm H₂O for ET size 5 and above

Every morning stop sedation for an hour (sedation holiday) and check if the following criteria are achieved.

- SpO₂ > 92% at PEEP < 6 cm H₂O and FiO₂ < 0.4–0.5
- Delivered tidal volume 4–6 ml/kg

#### Hemodynamic stability
- Heart rate and blood pressures appropriate for the age
- Minimum or no vasopressor requirement

Patient initiates spontaneous inspiratory efforts

Absence of fever

Patient performs the following simple commands:
- Opens and closes eyes, moves limbs against gravity
- Good cough and gag

Meets all the above criteria

Yes

Resume the earlier ventilation and reassess for extubation readiness after 24 hours

No

Continue PSV or T-piece trial (SBT). After 60 minutes assess whether: quite and stable breathing without tachypnea, distress or shallow breathing.

SpO₂ > 92%, change in heart rate (HR) < 20%, change in systolic blood pressure (SBP) < 20%

Patient is not agitated. Patient is obeying commands.

After making sure that stomach is empty, chest clear on auscultation, keep the same size and one size smaller ETT ready for possible reintubation along with laryngoscope and AMBU bag and mask.

Extubate the child and keep in high flow oxygen/continuous positive airway pressure (CPAP). Close vitals monitoring, watch for color perfusion, HR, respiratory rate, work of breathing, level of alertness, any stridor, postextubation blood gas and chest X-ray.

Dexamethasone starting 12–24 hours prior to extubation in children at risk of postextubation stridor, and continue for 1 day after extubation and then stop. Racemic epinephrine nebulizations and CPAP also reduce postextubation stridor in at “at risk” children. (Difficult intubation, neck surgeries, failed one extubation, prolonged ventilation, etc.)
Noninvasive Ventilation

Rajiv Uttam, Shipra Gulati

Ventilatory support provided without invasive airway control
Nasal mask or nasal CPAP cannula or nasal oral mask

Key differences between NIV and IV
Advantages of NIPPV
- Allows patient to maintain normal functions
  - Speech
  - Eating
- Helps Avoids the risks and complications related to
  - Intubation
  - Sedation
  - Less ventilator associated pneumonia

Clinical Uses of NIV in Intensive Care
- Cardiogenic pulmonary edema
- Hypoxic respiratory failure
- Other possible indications
  - Weaning
  - Post surgery
  - Asthma
  - Extubation failure
  - Neuromuscular respiratory weakness

NIV can be applied in two ways

NINPV
- Supports ventilation by exposing chest wall to subatmospheric pressure inspiration
- Expiration occurs as pressure around chest wall allowed to return to atmospheric level (Iron lung)

NIPPV
- Delivery of positive pressure ventilation to lungs without invasive airway:
  It can be given by:
  - Pressure controlled ventilator
  - Pressure support ventilation
  - CPAP devices
  - Bilevel positive airway pressure ventilator

Contd...
### Initiating NIPPV
Initial settings:
- Spontaneous trigger mode with backup rate
- Start with low pressures
  - IPAP 8–12 cm H₂O
  - PEEP 3–5 cm H₂O
- Adjust inspired O₂ to keep saturation > 90%
- Increase IPAP gradually to
  - Decrease respiratory rate
  - Increase tidal volume

### Success and Failure Criteria for NIPPV
Improve in pH and PCO₂ occurring within 2 hours predict eventual success of NIPPV

**Indication for Early Discontinuation of NIPPV**
- Worsening sensorium
- Extreme distress and anxiety
- Hemodynamic instability
- Worsening oxygenation

*Abbreviations:* ETT, endotracheal tube; NIV, noninvasive ventilation; IV, invasive ventilation; NIPPV, noninvasive positive pressure ventilation; NINPV, noninvasive negative pressure ventilation; CPAP, continuous positive airway pressure; IPAP, inspiratory positive airway pressure; PEEP, positive end-expiratory pressure.
High Frequency Ventilation

- Basic concept is to use smaller than physiological tidal volumes (tidal volume ≤ dead space) at supraphysiological rates to support ventilation and oxygenation
- Underlying benefit: Less barotrauma and volutrauma

Types of HFV

- **High Frequency Positive Pressure Ventilation**
  - O₂ supplied from high pressure gas source
  - Gas delivery and flow provided by a pneumatic valve system

- **High Frequency Jet Ventilation**
  - Ventilation delivered by low compliance, low volume tubing and jet catheter inserted or built into ET tube, which is attached to the ventilator

- **High Frequency Oscillating Ventilation**
  - CPAP system with piston displacement of gas
  - Rates 180–900 breaths per minute
  - Tidal volume less than anatomic dead space

Indications for HFOV

- Inadequate oxygenation that cannot safely be treated without potentially toxic ventilatory settings, thus increase risk of VALI
- Objectively defined by:
  - PIP > 30–35 cm H₂O
  - FiO₂ > 0.6 or inability to wean
  - Mean airway pressure > 15 cm H₂O
  - PEEP > 10 cm H₂O

Clinical Goals

- Reasonable oxygen to limit oxygen toxicity
  - SaO₂ 86–92%
  - PaO₂ 55–90 mmHg
- Permissive hypercapnia

Contd...
**Oxygenation**

Two primarily variables that control oxygenation are:
- $\text{FiO}_2$
- Paw

**Oxygenation: Clinical Tips**

- Initiate HFOV with
  - $\text{FiO}_2$: 1.0
  - Paw: 5–8 cm H$_2$O more than Paw on CMV
- Increase Paw by 1–4 cm H$_2$O to achieve optimal lung volume
- Optimal lung volume is determined by increase in $\text{SaO}_2$ allowing the $\text{FiO}_2$ to be weaned
- Diaphragm is at ~T9 on chest radiograph
- Maintain Paw and wean $\text{FiO}_2$ until ≤ 0.6

**Ventilation**

Tidal volume ($\Delta P$ or amplitude)
- Controlled by the force with which the oscillatory piston moves
- Start amplitude in 30’s and adjust until wiggle extends to lower level of patient’s groin
- Adjust in increments of 3–5 cm H$_2$O

**Frequency ($f$)**

Range: 3–15 Hz
- Initial frequency settings
  - Preterm neonates: 10–15 Hz
  - Term Neonates: 8–10 Hz
  - Children: 6–8 Hz
  - Adults: 5–6 Hz

**Improving Ventilation**

- To improve ventilation first increase amplitude
- If no improvement, consider decreasing frequency

*Abbreviations: HFV, high frequency ventilation; CPAP, continuous positive airway pressure; HFOV, high frequency oscillatory ventilation; VALI, ventilator-associated lung injury; PIP, peak inspiratory pressure; $\text{FiO}_2$, fraction of inspired oxygen; MAP, mean airway pressure; PEEP, positive end-expiratory pressure; $\text{PaO}_2$, partial pressure of oxygen; $\text{SaO}_2$, arterial oxygen saturation; Paw, mean airway pressure; CMV, conventional mechanical ventilation*
Case Scenario
A 6-year-old girl developed worsening of her asthma symptoms one early morning. Her mother administered her two puffs of salbutamol with spacer (which she was well trained to have). Not seeing any improvement after 15 minutes she gave her two more puffs and moved her to the neighborhood nursing home.

At arrival there the pediatrician found her to be dyspneic, diaphoretic and unable to talk in full sentences. Auscultation of chest revealed B/L rhonchi. Her pulse oximeter oxygen saturation (SpO₂) was 90%. Keeping her history of pediatric intensive care unit (PICU) admission twice in the past, she was administered two more puffs of salbutamol, 20 mg of prednisolone orally and transferred to a tertiary care hospital in an ambulance. All along the 20 minutes drive she received oxygen (O₂) inhalation. She was also administered two more puffs of salbutamol with spacer on the way.

Steps in Management of Pediatric Status Asthmaticus

Step 1
Assess Airway, Breathing and Circulation (ABC) and take resuscitative measures if necessary.

Step 2
Assess severity of the attack (Table 11.1).
Status asthmaticus is defined as severe asthma that fails to respond to inhaled β₂-agonist agonists, oral or intravenous (IV) steroids, and O₂ and that requires admission to the hospital for treatment.

The rapid assessment of a child with status asthmaticus should focus upon determining the severity of airway obstruction. Wheezing, which reflects turbulent airflow in obstructed airways, is usually equally audible on both hemithoraces. Asymmetric wheezing may imply unilateral atelectasis,
**Table 11.1: Severity of asthma exacerbations**

<table>
<thead>
<tr>
<th></th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
<th>Respiratory arrest imminent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breathing</td>
<td>Walking</td>
<td>Talking</td>
<td>At rest</td>
<td>Infant stops feeding</td>
</tr>
<tr>
<td></td>
<td>Can lie down</td>
<td>Talking: Softer,</td>
<td>Infant: Hunched</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>shorter cry;</td>
<td>forward</td>
<td></td>
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<td></td>
<td></td>
<td>Difficult feeding</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Prefers sitting</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Talking</td>
<td>Sentences</td>
<td>Phrases</td>
<td>Words</td>
<td></td>
</tr>
<tr>
<td>Alertness</td>
<td>May be agitated</td>
<td>Usually agitated</td>
<td>Usually agitated</td>
<td>Drowsy or confused</td>
</tr>
<tr>
<td>Respiratory rate</td>
<td>Increased</td>
<td>Increased</td>
<td>Increased</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Normal rate of</td>
<td>Increased</td>
<td></td>
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<tr>
<td></td>
<td>breathing in awake</td>
<td></td>
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<tr>
<td></td>
<td>children:</td>
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<tr>
<td></td>
<td>Age</td>
<td></td>
<td></td>
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</tr>
<tr>
<td></td>
<td>&lt; 2 months</td>
<td>&lt; 60 breaths/minute</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>2–12 months</td>
<td>&lt; 50 breaths/minute</td>
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<td></td>
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<tr>
<td></td>
<td>1–5 years</td>
<td>&lt; 40 breaths/minute</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>6–8 years</td>
<td>&lt; 30 breaths/minute</td>
<td></td>
<td></td>
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<tr>
<td>Accessory muscles and</td>
<td>Usually not</td>
<td>Usually</td>
<td>Usually</td>
<td>Paradoxical thoracoabdominal</td>
</tr>
<tr>
<td>suprasternal retractions</td>
<td></td>
<td></td>
<td></td>
<td>movement</td>
</tr>
<tr>
<td>Wheeze</td>
<td>Moderate, often only</td>
<td>Loud</td>
<td>Usually loud</td>
<td>Absence of wheeze</td>
</tr>
<tr>
<td></td>
<td>end expiratory</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulse/minute</td>
<td>Mild tachycardia</td>
<td>Moderate tachycardia</td>
<td>Severe tachycardia</td>
<td>Bradycardia</td>
</tr>
</tbody>
</table>

Contd...
Guide to limits of normal pulse rate in children:
- Infants 2–12 months: Normal Rate < 160 beats/minute
- Preschool 1–2 years: < 120 beats/minute
- School age 2–8 years: < 110 beats/minute

Pulsus paradoxus (Can be observed on SpO₂ monitor wave form)
- Absent: < 10 mmHg
- May be present: 10–25 mmHg
- Often present: 20–40 mmHg

PEF (After initial bronchodilator)
- % predicted or % personal best
  - Over 80%
  - Approx. 60–80%
  - < 60% predicted or personal best or response lasts < 2 hours

PaO₂ (on air) and/or PaCO₂
- Normal Test not usually necessary
  - < 45 mm Hg
  - 60 mmHg
  - < 60 mmHg Possible cyanosis
  - 45 mmHg: Possible respiratory failure

SaO₂ % (on air)
- 95%
- 91–95%
- < 90%

Hypercapnia (hypoventilation) develops more readily in young children than adults and adolescents

Abbreviations: SpO₂, pulse oximeter oxygen saturation; PEF, peak expiratory flow; PaO₂, partial pressure of oxygen in arterial blood; PaCO₂, partial pressure of carbon dioxide in the arterial blood; SaO₂, percentage of available hemoglobin that is saturated with oxygen.
pneumothorax or foreign body. Expiratory wheezing alone is found in mild to moderate illness, whereas expiratory plus inspiratory wheezing is present in moderate to severe status asthmaticus. The silent chest is an ominous sign and may indicate either pneumothorax or the complete absence of airflow due to severe airway obstruction and imminent respiratory failure.

Blood gas analyses may support the clinical judgment of severity. An increasing level of carbon dioxide ($CO_2$) is an ominous sign. During a moderate asthma attack, a capillary blood gas analysis may be sufficient; for patients admitted to an intensive care unit, arterial blood gas analyses should be a routine. Sequential measurements are important as respiratory alkalosis with hypocarbia is common during the early phases of an asthma attack, while normalization and a subsequent increase in the partial pressure of carbon dioxide in the arterial blood ($PaCO_2$) may be important indicators of clinical deterioration. Thus, a normal $PaCO_2$ with even borderline low partial pressure of oxygen in the arterial blood ($PaO_2$) indicates a phase of rising $PaCO_2$, hence, need for more intensive therapy.

A chest X-ray may be relevant in the search for underlying complications such as pneumonia or air leakages.

**Step 3**
Take into consideration the treatment that the child may have received in the past few hours. This helps us in deciding where in the treatment algorithm (Figs 11.1A and B) should we start. For example, a child who has received several doses of salbutamol in past 1 hour it may be futile to begin treatment at the top end of the algorithm.

**Step 4**
Follow the algorithm for treatment. Generally children tolerate repeated doses of salbutamol very well and tachycardia as a side effect is less worrisome.

**Step 5**
At all stages, the child should be constantly monitored and escalation or de-escalation of therapy should be done accordingly. For example; a child who is showing signs of exhaustion may have to be straight away intubated even if IV β-agonist or aminophylline has not yet been tried.

**Step 6**
Intravenous ketamine can be tried in children who are not improving on IV β-agonist, IV steroids and supportive therapy. It is a sedative that has bronchodilator properties. Generally, it is started in the dose of 1 mg/kg/hour after a loading dose of 1 mg/kg. The infusion can be increased to 3 mg/kg/hour. However, all preparations should have been made for intubation and ventilation before starting IV ketamine.
Fig. 11.1A: Management of asthma exacerbations in the acute care setting

**Abbreviations:** PICU, pediatric intensive care unit; PEF, peak expiratory flow; PCO₂, partial pressure of carbon dioxide in blood; PO₂, partial pressure of oxygen in blood
**Fig. 11.1B**: Initial management of acute asthma

**Abbreviations**: MDI, metered-dose inhaler; SpO₂, pulse oximeter oxygen saturation; ABC, airway, breathing and circulation IV, intravenous; PICU, pediatric intensive care unit; LABA, long acting β-agonist
**Step 7: Intubation and Ventilation**

Generally, decision to intubate and ventilate an asthmatic child is made on clinical grounds. Thus cardiac arrest, respiratory arrest or severe bradypnea, extreme physical exhaustion and altered sensorium are taken as absolute indications. Blood gas analysis, worsening pulsus paradoxus as assessed on the bedside monitors can be additional parameters in making a decision for intubation and ventilation.

Standard rules of sedation and muscle relaxation are followed with some preferring to use ketamine.

Ventilation is started in the controlled mode. Both pressure and volume controlled modes [or the combined modes like pressure regulated volume control (PRVC)] can be used initially. As soon as possible, the child is shifted to assist/synchronized intermittent mandatory ventilation (SIMV) modes. Experience with pressure support mode in the initial stages of ventilation is very limited in pediatrics.

Non-invasive ventilation for status asthmaticus in children is not generally recommended, although a few units have some experience with this modality also.

**Step 8**

Permissive hypoventilation is an accepted strategy in difficult to ventilate asthmatic children.

**Further Reading**

Brief Protocol of Treatment of Bronchial Asthma in PICU

Praveen Khilnani

Severe Asthma
Rapid cardiopulmonary assessment

No Improvement
• O2 < 90% on air
• Tachycardia
• Fast breathing
• Pallor/Cyanosis
• Altered sensorium
• Monitor SpO₂, HR, RR
• Promptly start oxygen 15 L/min
• Salbutamol nebulisation
  – 3 times every 20 minutes
• Systemic steroid: oral/IV
Reassess after hour

Admit in PICU
• Continue O₂
• Salbutamol nebulization
  – 3 times every 20 minutes/or
  – Continuous nebulization 0.5 mg/kg
  (Max 15 mg)
• Ipratropium Bromide
• IV salbutamol if available
• 50% MgSO₄: 50 mg/kg/dose IV
• Try SC Adrenaline
• IV Aminophylline
• Systemic steroid therapy
• IV fluids

Improved
• RR < 50
• Minimal chest recession
• Minimal use of accessory muscles
• PFR > 50–75% predicted
• Saturation > 90% on air

No Improvement
• Exhausted/Lethargic
• Mixed acidosis
• Silent chest/decreased chest movement
• Worsening SpO₂

Rapid Sequence Intubation
(use ketamine)

Continue
• Hourly inhaled or nebulized Salbutamol
• Ipratropium Bromide 6 hourly
• Ensure adequate fluid intake
• Hourly monitoring
  – HR, RR, SpO₂

Contd...
### Initiate Ventilation
- Low tidal volume/PIP
- PRVC: good option
- Slow rate
- I:E ratio: 1:3–1:4
- Low PEEP
- Keep Pplat < 30
- Permissive hypercapnia (Keep pH > 7.2)

### Consider
- Inhaled anesthetics (Halothane, Isoflurane)
- Heliox
- ECMO for failed conventional ventilation

### Good Improvement
- No resp distress
- PEF > 75%
- Saturation > 95% on air
- No tachypnea

### Discharge on
- Inhaled β₂ agonists
- Oral steroid for 3–10 days

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**Abbreviations:**
- PICU: pediatric intensive care unit
- SC: subcutaneous
- IV: intravenous
- SPO₂: oxygen saturation
- ECMO: extracorporeal membrane oxygenation
- PEEP: positive end-expiratory pressure
- I:E ratio: inspiratory to expiratory ratio
- PRVC: pressure regulated volume control
- PIP: peak inspiratory pressure
- RR: respiratory rate
- HR: heart rate
- PFR: peak flow rate
- PEF: peak expiratory flow

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Introduction

Shock can be defined by clinical variables, hemodynamic variables, oxygen utilization variables, and/or cellular variables; however, the review committee of 2007 chooses to define septic shock by clinical, hemodynamic, and oxygen utilization variables only.

Shock should be clinically diagnosed before hypotension occurs by clinical signs, which include:

- Hypothermia or hyperthermia
- Altered mental status
- Peripheral vasodilation (warm shock) or vasoconstriction with capillary refill > 2 s (cold shock)
- Tachycardia
- Tachypnea out of range for age and level of fever or anxiety.

Decompensated or hypotensive shock with low BP has a poor outcome.

Step 1

This includes fast recognition and action done almost simultaneously

- Zero minutes
- Recognize decreased mental status and perfusion
- Maintain and establish vascular access—use intraosseus if IV fails in 90 s
- 5–15 mins push 20 ml/kg normal saline/colloid X 3 Upto 60 ml/kg.

Assess between each push—correct hypoglycemia and hypocalcemia.

There should be no time wasted in gaining access. If access is not easily obtained in about 90 secs, the intraosseus (IO) route is a must as almost everything can go in by that route including inotropes. Because mortality went up with delay in time to inotrope drug use, the 2007 update now recommends use of peripheral inotropes dopamine and dobutamine (not vasopressors) until central access is attained. Careful observation of the limb and local instillation of phentolamine to prevent necrosis if extravasation occurs, is used.
Optimizing fluids in the first 15 minutes or as soon as possible. Pediatric septic shock is associated with severe intravascular volume depletion, and children frequently respond well to aggressive volume resuscitation.

The continued emphasis is directed to first hour fluid resuscitation and inotrope drug therapy directed to goals of
- Reducing heart rate to threshold level for age
- Getting peripheral pulse to match central pulse volume
- Improving mentation
- Improving urine output to at least 1 ml/kg/hr
- Reducing capillary refill time (CRT) to < 3 s

This assessment is done after each bolus and a quick check for overload in the form of rapid expansion of the liver span, rales and increased work of breathing and enlargement of the cardiac silhouette on chest X-ray is done.

**Step 2**
- 15 mins fluid refractory shock—establish central venous access
- Start dopamine, 10 mcgm /kg/min—establish arterial access
- Continue maintenance fluids 4 ml/kg/hr and boluses of 9NS/colloid as needed
- 30–60 mins have passed—fluid refractory dopamine resistant shock.

Three scenarios:
1. When pediatric patients are normotensive with a low CO and high SVR, initial treatment of fluid-refractory patients consists of the use of an inotropic agent such as dobutamine may be added. Dopamine at 10–15 µg/kg/min would be on at this time. However, the stage of fluid refractory dopamine resistant shock is an important defining step as the mortality changes when the patient fails to respond to fluids and dopamine.

2. When pediatric patients are hypotensive with a low CO and high SVR (cold shock) epinephrine (EPI) is started at 0.1 µg/kg and titrated to effect. When BP improves an inodilator (dobutamine, milrinone, nitroglycerin (NTG)) is added to improve tissue perfusion. This can be done using the same clinical parameters described above or additional laboratory data such as base excess of > -5 or increasing lactate levels.

3. When pediatric patients are hypotensive with a high CO and low SVR (warm shock) then norepinephrine (NE) is the vasopressor of choice. Since the pulse pressure is wide and diastolic pressures are usually low, the need is to increase the MAP. Here to a vasodilator can be added.

There is no magic formula for inotrope or fluid titration. The guidelines are there to give a framework for initiating and adding drugs based on clinical exam and parameter readings of CVP, BP, etc. Many children by now may be on more than three agents including vasopressors and vasodilators. Other agents like vasopressin when there is vasoplegia and poor response are in favor with some and some use phenylephrine which raises the SVR and thus the BP.
**What Does Early Goal Directed Therapy Try To Do (EGDT)?**

It restores the balance between delivery and demand quickly by manipulating preload, afterload and contractility using fluids, inotropes, vasodilators to enhance delivery and packed red blood cells (PRBCs) to deliver more oxygen by increasing $O_2$ content.

**Early Goal Directed Therapy**
- Normal MAP-CVP (> 60) and mixed Ven sat > 70%
- U/O > 1 ml/kg/hr CVP > 8–12 cm H$_2$O$_2$
- All four Goals to be met for success
- Done by:
  - Increasing inotropes/dobutamine and fluids
  - Sedation and ventilation to reduce $O_2$ consumption
  - PRBCs to keep Hb 10

**First Hour Antibiotics and Source Control**
An increasing mortality for every hour of delay in the administration of an appropriate antibiotic has been clearly shown in several pediatric and adult studies. While every attempt should be made to draw appropriate culture/s prior, this should not hold up the administration of the drug. The choice should be as per the site of infection and local patterns and a broad spectrum antibiotic like a 3rd generation cephalosporin should be used. De-escalation can be done later.

Along with this there must be an active search for a source and immediate action for source control taken whenever possible.

**Mechanical Ventilation and Sedation**
There are many reasons to ventilate patients with septic shock. This step should be considered in any patient who is not rapidly stabilized with fluid resuscitation and peripherally administered inotropes. To establish a central venous pressure (CVP) line a quiet nonmoving child is best as bleeding will occur easily with an underlying coagulopathy.

**Steroids When and At What Dose?**
If a child is at risk of absolute adrenal insufficiency (e.g. purpura fulminans, congenital adrenal hyperplasia, prior recent steroid exposure as in asthma or nephrotic syndrome) and remains in shock despite EPI or NE infusion and optimized fluids and inotropes for an hour (catecholamine resistant shock) then hydrocortisone can be administered. Sampling for levels tells us about absolute but not about relative cortisol insufficiency and hence may be redundant. Hydrocortisone may be administered as an intermittent or continuous infusion at a dosage which may range from 1–2 mg/kg/day for stress coverage to 50 mg/kg/day titrated to reversal of shock. At this stage the mortality from shock rises.
**Glucose Control**

Glucose containing fluids D5 or D10 along with insulin should be used for maintenance and insulin titrated to keep blood sugar between 80–150 mg/dL. This prevents catabolism as well as the ill effects of hyperglycemia. Tight glucose control leads to hypoglycemia and this can be brain damaging so should be guarded against. Hyperglycemia should not be treated by reducing fluid concentrations to glucose free fluids and removing insulin as there is poor glucose utilization and insulin is needed.

**Summary**

- Immediate recognition of shock state from decreased perfusion state and altered mental status. ABCs with high flow O₂
- Rapid IV access IO immediately if IV not available
- For 0–15 mins at least 60 ml/kg must go in. Isotonic nonglucose containing fluid
- Clinical evaluation of improvement of shock by decreasing HR, CRT < 2s, improved mental status, improved peripheral pulse—central pulse, improved urine output, warmer extremities and MAP > 60 (age related values)
- Fluid overload to be also evaluated
- Rapid decision to start dopamine/dobutamine by peripheral line not wait for central line
- First hour appropriate antibiotics
- Continue fluid boluses as needed throughout the process. May be 150–200/ kg in first few hours + maintenance fluids with glucose
- Ventilation with sedation and analgesia; if fluid refractory dopamine resistant shock. CVP and arterial lines if possible
- Epinephrine for cold shock norepinephrine for warm shock +/- vasodilators
- Steroids for catecholamine resistant shock at 2 mg/kg/d Q8
- Early goal directed therapy with ScVO₂ 70%, Hb 10, CVP 8–12, MAP > 60 mmHg
- Source control asap
- Glucose control with insulin if needed < 150 mg/dL.

**Shock**

- 0–5 minutes
  - Recognize decreased mental status and poor organ perfusion
  - Maintain airway and establish vascular access (PALS)
- 5–15 minutes
  - Push 20 cc/kg normal saline → reassess 5 end points (Improved sensorium, CRT< 3 s, decreasing or normalized HR, peripheral pulse volume = central, u/o increased to at least 1 ml/kg/hr) if not → give again upto 60 ml/k (correct hypocalcemia and hypoglycemia →
  - Reassess 5 end points after each bolus + look for s/o fluid overload
**Brief Protocol for Managing Shock**

**Fluid refractory shock**
- Establish central venous access—call for help
- Start dopamine 10 micrograms/kg/min by peripheral till CVP in
- Once CVP in, send SvO₂ immediately and note 5 end points again
- If there is no improvement in 5 clinical end points go by CVP and CRT
- Continue fluid boluses if < 8, titrate to 12
- If >14 reduce fluids assess for improvement and volume overload
- If SBP/MAP >= 5th % tile for age and SvO₂ low start dobutamine (10–20)
- If BP still low and s/s of low perfusion titrate with dopamine +– Epi.

**Antibiotic**
Within the first hour appropriate antibiotic as per possible source of infection 3rd gen cephalosporin usually +/- AG. Or coverage as needed 2 cultures if possible.

**Consider Endotracheal Intubation and Ventilation with RSI and Lung Protective Strategies**
- 15-40 mins fluid refractory-dopamine resistant shock
  - Epinephrine for cold shock: cold clammy with narrow pulse press < 20
  - Norepi for warm shock: warm with good pulse volume and wide pulse pressure
  - Recheck ScVO₂, urine output, CRT,
- 60 mins Catecholamine resistant shock (NIBP now unreliable Art line needed)
  - if BP low after at least 1 hour after optimizing fluids and inotropes give 2 mg/kg Hydrocortisone
  - Continue Q8H till shock resolves

<table>
<thead>
<tr>
<th>BP</th>
<th>Normal</th>
<th>Low</th>
<th>Low</th>
</tr>
</thead>
<tbody>
<tr>
<td>cold shock</td>
<td>cold shock</td>
<td>warm shock</td>
<td></td>
</tr>
<tr>
<td>SVCO₂ sat &lt; 70%</td>
<td>SVCO₂ sat &lt; 70%</td>
<td>↓</td>
<td></td>
</tr>
<tr>
<td>↓</td>
<td>↓</td>
<td>↓</td>
<td></td>
</tr>
<tr>
<td>Vasodilator + Volume</td>
<td>Volume + Epi</td>
<td>Vol + Norepi</td>
<td></td>
</tr>
<tr>
<td>Milrinone/nitroglycerine/dobutamine</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Vasopressin (VP) if there is no response or inadequate response to increase of E/NE and signs of MOD with rising lactate, falling TCO₂, etc. are seen PRBCs may also be used to enhance oxygen delivery and raise the hematocrit to 30 if ScVO₂ is < 70% after optimizing fluids and CVP to 12 cm H₂O.

**Glossary**
- ScVO₂: Mixed venous oxygen saturation
- EPI: Epinephrine
- NE: Norepinephrine
- CRT: Capillary refill time
- u/o: Urine output
**Further Reading**


0–5 Min

5–40 Min
Initial resuscitation: Push fluid boluses of 20 ml/kg isotonic saline or Ringer’s lactate up to and over 60 cc/kg until perfusion improves or unless rales or hepatomegaly develop. Correct hypoglycemia. Begin antibiotics (third generation cephalosporin and aminoglycoside)
   Simultaneously establish a second peripheral IV line or if feasible central line.

Shock Not Reversed?

40–60 Min
Fluid refractory shock: Begin inotrope IV/IO. Dopamine up to 10mic/kg/min, dobutamine/10 mg/kg/min. Reverse cold shock by titrating dopamine, or if resistant (normal or low blood pressure) titrate epinephrine (0.05–0.3 µg/kg/min). Reverse warm shock with low blood pressure by titrating norepinephrine (0.05–1.0 µg/kg/min).

Shock Not Reversed?

60 Min
Recognize catecholamine resistant shock
- Begin hydrocortisone (50 mg/m2/dose) if at risk for absolute adrenal insufficiency.
- Consider use of vasodilator (sodium nitroprusside/Trinitroglycerin) or phosphodiesterase inhibitor such as milrinone if cold shock and normal blood pressure(if available).
- Consider vasopressin infusion (0.3–2 milliunits/kg/min: equivalent to 0.0003–0.002 units/kg/min or 0.01–0.12 units/kg/hr) if warm shock with low blood pressure unresponsive to norepinephrine (if available).
**Beyond 60 Min**

Transfer to PICU facility.

Monitor CVP, mean arterial pressure. Titrate fluids and inotropes to attain normal mixed venous oxygen saturation ScVO$_2$ > 70 (other advanced drugs such as levosimendan, enoximone, terlipressin beyond scope of these guidelines)

*Relief of tamponade such as pneumothorax, or pericardial tamponade, increased intra-abdominal pressure due to fluid should be considered at any point.

**Choice of Empirical Antibiotic in Patients with Septic Shock with Respect to Clinical Settings**

<table>
<thead>
<tr>
<th>Clinical setting</th>
<th>Usual pathogens</th>
<th>Preferred therapy</th>
<th>Alternate therapy</th>
</tr>
</thead>
</table>
| Unknown source from the community | *Salmonella typhi/paratyphi*  
*H. influenzae*  
*Enterobacteriaceae*  
*B. fragilis*  
*E. fecalis*  
Think of malaria and dengue | Ceftriaxone plus metronidazole or Piperacillin/tazobactam or Meropenem or Imipenem | Quinolone (cipro/levo) plus either metronidazole or Clindamycin |
| Lung source | *S. pneumoniae*  
*H. influenzae*  
*Staphylococcus aureus*  
*M. pneumoniae*  
Think of malaria and dengue | Ceftriaxone/cefotaxime/amoxicillin and azithromycin or clarithromycin | Substitute new fluoroquinolone (levo/gatifloxacin/moxifloxacin) for macrolide |
| IV line sepsis | *S. epidermidis*  
*S. aureus (MSSA)*  
*Klebsiella*  
*Enterobacter*  
*Serratia* | Vancomycin plus Meropenem or Imipenem or Cefepime or Pip-Tazo | May substitute linezolid for vancomycin  
Add antifungals if fungus suspected |
| Urosepsis | *Enterobacteriaceae* | Ceftriaxone or Cefotaxime or Quinolone | Aztreonam or Ampicillin + Amikacin |
| Meningitis | *S. pneumoniae*  
*H. influenzae*  
*Meningococci* | Ceftriaxone or Cefotaxime | Add vancomycin if drug resistant pneumococci suspected |
| Intrabdominal source | *Enterobacteriaceae*  
*B. fragilis*  
*Enterococci* | Ceftriaxone plus Metronidazole or Piperacillin/tazobactam or Meropenem or Imipenem | Quinolone (cipro/levo) plus either Metronidazole or Clindamycin |
### Doses of Various Antibiotics in Pediatric Septic Shock

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Frequency</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ampicillin</td>
<td>50 mg/kg/dose</td>
<td>6 hourly</td>
<td>IV, IM</td>
</tr>
<tr>
<td>Ampicillin + Sulbactam</td>
<td>50 mg/kg/dose of ampicillin</td>
<td>6 hourly</td>
<td>IV or IM</td>
</tr>
<tr>
<td>Amikacin:</td>
<td>&gt; 10 years: 20 mg/kg on day 1,</td>
<td>OD</td>
<td>IV or IM</td>
</tr>
<tr>
<td></td>
<td>then 15 mg/kg, 1 week–10 year: 25 mg/kg on day 1, then 18 mg/kg,</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Neonates: &lt; 30 weeks: 15 mg/kg on day 1, then 7.5 mg/kg, &gt; 30 weeks–</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Term: 10 mg/kg, term 15 mg/kg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amoxicillin</td>
<td>50 mg/kg/dose</td>
<td>8 hourly, 12 hourly (for babies &lt; 1 week), 6 hourly (2–4 weeks)</td>
<td>IV, IM</td>
</tr>
<tr>
<td>Amoxicillin + Clavulanic acid</td>
<td>Dose same as amoxicillin 4:1</td>
<td>8 hourly</td>
<td>IV, IM</td>
</tr>
<tr>
<td>Amphotericin B</td>
<td>0.5–1.5 mg/kg/day</td>
<td>Infusion with D5W over 4–8 weeks</td>
<td>IV</td>
</tr>
<tr>
<td></td>
<td>Total dose 30–35 mg/kg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amphotericin B Lipid/Liposomal</td>
<td>2–3 mg/kg/day over 1 hr</td>
<td>Infusion with D5W over 2–4 weeks</td>
<td>IV</td>
</tr>
<tr>
<td></td>
<td>Total dose 20–60 mg/kg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fluconazole</td>
<td>12 mg/kg stat, 6–12 mg/kg/dose</td>
<td>OD</td>
<td>IV</td>
</tr>
<tr>
<td>Itraconazole</td>
<td>5 mg/kg/dose</td>
<td>12–24 hourly</td>
<td>IV, oral</td>
</tr>
<tr>
<td>Voriconazole</td>
<td>6 mg/kg/dose stat over 2 hours, repeat after 12 hours, then 4 mg/kg/dose</td>
<td>12 hourly</td>
<td>IV, IV, oral</td>
</tr>
<tr>
<td>Ceftriaxone</td>
<td>50 mg/kg/dose</td>
<td>12 hourly</td>
<td>IV or IM</td>
</tr>
<tr>
<td>Cefepime</td>
<td>50 mg/kg/dose</td>
<td>8 hourly</td>
<td>IM or IV</td>
</tr>
<tr>
<td>Cefotaxime</td>
<td>50 mg/kg/dose</td>
<td>4–6 hourly, 12 hourly for neonates</td>
<td>IV</td>
</tr>
</tbody>
</table>
### IV, Intravenous; IM, Intramuscular

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Frequency</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ceftazidime</td>
<td>50 mg/kg/dose</td>
<td>6–8 hourly</td>
<td>IV, IM</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>&gt;10 years: 7 mg/kg on day 1, then 5 mg/kg/dose, 1 week–10 year: 8 mg/kg on day 1, then 6 mg/kg, Neonates: 5 mg/kg</td>
<td>OD</td>
<td>IV, IM</td>
</tr>
<tr>
<td>Cloxacillin</td>
<td>50–100 mg/kg/dose</td>
<td>4–6 hourly</td>
<td>IV</td>
</tr>
<tr>
<td>Vancomycin</td>
<td>10 mg/kg/dose</td>
<td>6 hourly</td>
<td>IV over 1 hour</td>
</tr>
<tr>
<td>Neonates: 10 mg/kg/dose</td>
<td>8 hourly</td>
<td>IV</td>
<td></td>
</tr>
<tr>
<td>Piperacillin + Tazobactam</td>
<td>Piperacillin: 75–100 mg/kg/dose</td>
<td>6 hourly</td>
<td>IV</td>
</tr>
<tr>
<td>Neonates: 10 mg/kg/dose</td>
<td>8 hourly</td>
<td>IV</td>
<td></td>
</tr>
<tr>
<td>Ticarcillin + Clavulanic acid</td>
<td>50–75 mg/kg/dose</td>
<td>6 hourly</td>
<td>IV</td>
</tr>
<tr>
<td>Cefoperazone sulbactam</td>
<td>50 mg/kg/dose of cefoperazone</td>
<td>8 hourly</td>
<td>IV</td>
</tr>
<tr>
<td>Meropenem</td>
<td>40 mg/kg/dose</td>
<td>8 hourly</td>
<td>IV</td>
</tr>
<tr>
<td>Imipenem + Cilastin</td>
<td>25 mg/kg/dose of imipenem</td>
<td>6 hourly</td>
<td>IV</td>
</tr>
</tbody>
</table>
Dengue viral (DV) infections affect all age groups and produce in decreasing order of frequency, an asymptomatic infection, mild nonspecific symptoms or classic dengue. The more severe manifestations of shock and hemorrhage occur in less than 5% of DV infections.

**Classification and Clinical Course of Dengue**

The new revised dengue classification released by the WHO in 2009 divides dengue cases into just two major categories of severity—dengue (with or without warning signals) and severe dengue (Fig. 15.1).

**Clinical Manifestations and Phases**

Dengue is a systemic and dynamic disease with a wide spectrum of clinical presentations ranging from mild to severe; however, the clinical evolution and outcome may be highly unpredictable.

The course of illness is characterized by three well demarcated phases—the febrile, critical and recovery phase. While most patients recover following a self-limiting non-severe clinical course, a small proportion progress to severe disease which is characterized by plasma leakage with or without hemorrhage.

![Fig. 15.1: New simplified classification of dengue viral infections](Source: WHO 2009)
Clinical Phases of Dengue

After the incubation period, the illness begins abruptly and is followed by the three phases, the febrile, critical and recovery phases as mentioned in Figure 15.2.

The most specific and life-threatening manifestation of the critical phase is an increase in capillary permeability leading to plasma leakage and an equivalent rise in the hematocrit. This phase is short-lived, typically lasting 24–48 hours. Prolonged uncorrected shock, metabolic acidosis and thrombocytopenia may worsen disseminated intravascular coagulation (DIC), which in turn may lead to massive hemorrhage, thus setting off a progressive downward spiral of worse shock and bleeding.

Abbreviations: HCT: Hematocrit, RBC: red blood cells, IVF: intravenous fluids.

Fig. 15.2: Suggested approach to a patient with severe dengue and hypotension
Diagnosis
Laboratory Confirmation of Dengue

There are three main diagnostic methods to diagnose DV infections: serological tests, virological diagnosis and molecular methods including the polymerase chain reaction (PCR). The non-structural protein 1 (NS1) monoclonal antibody that can detect dengue NS1 antigen in blood, the positivity rate of PCR and NS1 decreases after the initial 4–5 days of fever.

Management of Patients with Dengue

For such a complex, dynamic and unpredictable disease, successful outcomes with mortality rates of < 1% can be achieved in the vast majority with surprisingly simple and inexpensive interventions, provided they are early, appropriate and are continually targeted to keep pace with the disease evolution. Conversely, once the window of opportunity of early treatment is missed or inappropriately addressed, the disease can progress extremely rapidly and can result in refractory shock, relentless hemorrhage and florid multi-organ failure where even expensive and sophisticated intensive care resources may be futile.

Indications for hospitalization and intravenous (IV) fluids include “warning signs” (Fig. 15.3) of significant plasma leak of which severe, intense abdominal pain is considered the most important; other warning signs are persistent vomiting, restlessness or lethargy, clinical fluid accumulation, mucosal bleeds or other significant bleeds, lethargy or restlessness and laboratory feature of rise in HCT along with rapid fall in platelet count. Infants and patients with co-morbid conditions such as diabetes, renal failure and obesity may also require admission.

Indications for intensive care unit (ICU) admission include children with severe dengue manifesting with shock, respiratory distress, abnormal bleeding or organ failure, e.g. neurological complications, liver and or renal dysfunction.

The four major management priorities of dengue patients in the critical phase are:

Management Priority 1: Replacement of Plasma Losses

Intravenous rehydration is the single most important intervention that can correct shock and save lives in both severe and non-severe forms of dengue, provided it is timely and appropriate.

Titrating fluid therapy in dengue: Fluid therapy in a patient with dengue shock has two parts: Initial rapid fluid boluses to reverse shock followed by titrated fluid volumes to match ongoing losses. The end-points/targets of fluid administration are normalization of the SBP (if low), pulse pressure > 30 mmHg, a urine output of > 0.5–1 mL/kg/hour with stable vital signs and a gradual fall in the elevated baseline hematocrit.
Probable Dengue

- Lives in/travel to dengue endemic area
- Fever and two of the following criteria:
  - Nausea, vomiting
  - Rash
  - Aches and pains
  - Tourniquet test positive
  - Leukopenia
  - Any warning sign
- Laboratory-confirmed dengue (important when no sign of plasma leakage)

Warning Signs

- Intense abdominal pain or tenderness
- Persistent vomiting
- Clinical fluid accumulation
- Mucosal bleed
- Lethargy, restlessness
- Liver enlargement > 2 cm
- Laboratory: Increase in hematocrit with concurrent rapid decrease in platelet count

Criteria for Severe Dengue

- Severe plasma leakage with rising hematocrit leading to:
  - Shock
  - Fluid accumulation (pleural, ascitic)
  - Respiratory distress
  - Severe bleeding
  - Severe organ involvement
- Liver: Elevated transaminases (AST or ALT >=1000)
- CNS: Impaired consciousness, seizures
- Cardiac or other organ dysfunction

Abbreviations: HCT: Hematocrit, RBC: red blood cells, IVF: intravenous fluids

Fig. 15.3: Volume replacement flow chart for patients with severe dengue and compensated shock
Management Priority 2: Recognition and Management of Hemorrhage

Occult hemorrhage is one of the most important yet preventable causes of death. Suspect hemorrhage in the critical phase of dengue when the hematocrit is “normal” or lower than expected for the degree of shock. The most important intervention in a patient with dengue shock and life-threatening bleeds is restoration of oxygen carrying capacity with fresh whole blood (WB) or packed red blood cell (PRBC) transfusions; this must be done urgently rather than waiting for the hematocrit to fall significantly. The 2009 WHO Dengue Guidelines emphasize that in a bleeding dengue patient, the threshold for PRBC whole blood must be higher than that suggested for septic shock, where a hematocrit < 30% is the usual transfusion threshold. Platelet and other blood products are not usually necessary unless the patient is bleeding.

Abbreviations: HCT: Hematocrit, SBP: systolic blood pressure, PRBC: Packed red blood cells, CVP: central venous pressure, ECHO: echocardiogram, LV: left ventricle, RV: right ventricle, IAP: Intra-abdominal pressure, Rx: treatment

Fig. 15.4: Suggested approach to severe dengue and refractory shock (late presenters)
Management Priority 3: Prevention and Management of Fluid Overload

Intravenous rehydration is the sheet anchor of shock therapy; however, this can cause fluid overload and mandates careful titration of fluids to avoid this complication.

Management Priority 4: Prevention of Iatrogenic Complications

Complications of aggressive invasive ICU interventions such as central lines, drainage of pleural and ascitic fluid and even nasogastric tube insertion may lead to significant bleeding.

Further Reading

New Fever in PICU: Nosocomial Infection

Praveen Khilnani, Vikram Gagneja
**Pediatric HIV Algorithm**

**Abbreviations:** HIV, human immunodeficiency virus; PCR, polymerase chain reaction; LIP, lymphoid interstitial pneumonia.

**HIV suspected expecting mother**
- Perform pretest counseling
- Perform standard HIV testing

**Pediatric/adolescent HIV**
- Suspect HIV when signs and symptoms are suggestive
- Perform HIV DNA PCR in two separate blood samples

**HIV positive**
- Start mother on standard drug regime designed to decrease perinatal transmission

**HIV negative**
- Give routine antenatal care
- Exclude HIV

**HIV negative**
- Investigate neonate for HIV
  - Attend delivery following universal precautions
  - Perform HIV DNA PCR before 48 hours of age at 1–2 months/3–6 months

**HIV negative**
- Repeat HIV DNA PCR and exclude HIV if test is again negative

**HIV positive**
- Repeat HIV DNA PCR and diagnose a HIV +ve and begin treatment

**HIV positive**
- Diagnose a HIV +ve and begin treatment

**Monitor CD4 counts**
- Category 1: No suppression (CD% > 25%)
- Category 2: Moderate suppression (CD% 15–24%)
- Category 3: Severe suppression (CD% < 15%)

**Begin antiretroviral therapy**
- Start any of age-appropriate drug regimes

**Assign clinical category**
- Periodically examine patient and assign appropriate clinical category based on signs/symptoms and look for opportunistic infections

**P. Carinii pneumonia**
- Clinically present with tachypnea, dyspnea, cough and fever
- Fiberoptic bronchoscopy with bronchoalveolar lavage and appropriate staining with Giemsa/methenamine silver is diagnostic method of choice
- Treatment is TMP-SMX @ 20mg/kg X 21 days or pentamidine 4 mg/kg X 21 days

**Other opportunistic infections**
- Viral pneumonias
- Atypical mycobacterial pneumonia
- Lymphoid interstitial pneumonia (LIP)
- Candida sepsis
- Encephalopathy
- Treat symptomatically
Empyema Algorithm

**Simple (Uniloculated)**
- Antibiotics ± chest tube drainage
- Antibiotics

**Multiloculated**

**Antibiotics to be continued till**
- 48–72 hours after patient becomes afebrile and to be followed by oral antibiotics
- WBC count normal
- Chest X-ray showing clearing
- Tube thoracostomy yield <50 ml/day

**Usual duration of antibiotics**
- For *Haemophilus influenzae* and *Streptococcus pneumoniae*: 7–14 days
- For *Staphylococcus aureus*: 3–4 weeks
- For tubercular: ATT

**Indications for chest tube drainage**
If pleural fluid has following characteristics:
- Frank pus
- Smear positive for microorganisms
- Biochemical analysis
  - pH < 7.1
  - Glucose < 40 mg/dl
  - LDH > 1000 IU/L

**Medical**
- Antibiotics + chest tube drainage + thrombolytic therapy
- For thrombolytic therapy:  
  - Streptokinase
  - Urokinase

**Surgical**
- VATS
- Thoracoscopic debridement and irrigation

If above measures fail:
Decortication and open drainage

**Abbreviations:** ATT, antitubercular therapy; LDH, lactate dehydrogenase; VATS, video-assisted thoracoscopic surgery
Comatose Child

Praveen Khilnani, Nameet Jerath

Abbreviations: GCS, Glasgow coma scale; RSI, rapid sequence intubation; ATT, antitubercular therapy.
Fig. 19.1: Physiological correlation of clinical signs related to level of lesion (respiratory, pupillary, motor)
Continual assessment of neurological and cardiorespiratory status is imperative. CT head is part of raised intracranial pressure (ICP) monitoring as is GCS (Glasgow Coma Scale) score.

**Approach to increased ICP in the Neurological Injured Child**

**Abbreviations:** ICP, Intracranial pressure; HOB, Head of bed; CSF, Cerebral spinal fluid; CT, Computerized tomography
Pediatric Head Trauma

Pediatric Head Trauma

21

Praveen Khilnani, Pradeep Sharma

Contd...
Contd...

**Fig. 28.1**: Inter-relationship of PaO₂, PaCO₂, and cerebral perfusion pressure (CPP) with cerebral blood flow

**Abbreviations**: ICP, Intracranial pressure; CPP, Cerebral perfusion pressure
Clinical Signs of Herniation

Central Transtentorial Herniation

Progressive Rostrocaudal Deterioration of Brainstem Function
- Compression of diencephalic structures causes lethargy, apathy, or confusion
- Loss of upward gaze due to compression of the diencephalic pretectal area against the posterior tentorial incisura
- Extensor plantar responses and decortication (above red nucleus)
- Pupils dilate to midposition because of sympathetic and parasympathetic dysfunction
- Hyperventilation
- Decerebrate rigidity (below red nucleus and above vestibulospinal and reticulospinal tracts)
- Autonomic dysregulation
- Tonsillar herniation, common pathway leading to death.

Uncal Herniation (Lateral Transtentorial Syndrome)
- Associated with supratentorial masses and masses in the temporal fossa
  - Subdural or epidural hematoma, large MCA stroke, temporal lobe tumor
- Medial portion of the temporal lobe (uncus) displaces over the tentorial notch
- Causing compression of
  - Ipsilateral oculomotor nerve and brainstem compression.

Classically, A Stepwise Progression
- Early clinical sign is dilatation of the ipsilateral pupil
  - Compression of the parasympathetic fibers traveling on the periphery of the third nerve
  - Loss of the light reflex, ipsilateral ptosis
  - Patient may be surprisingly alert
- Contralateral hemiparesis
  - Compression of the ipsilateral cerebral peduncle against the free edge of the tentorium
  - Rarely ipsilateral hemiparesis (Kernohan's phenomenon)
  - Consciousness deteriorates
- Midbrain compression, sometimes associated hemorrhage, with compromise of the ascending portion of the RAS
  - Bilateral pupillary dilation.

Clinical Presentation

Vomiting Without Nausea
- Lesions involving the floor of the fourth ventricle and direct irritation of the vagus nucleus.
- More common in the morning.
- Changes in the level of consciousness.
- Bradycardia and rise in blood pressure.
- Respiratory changes can occur in the form of periodic breathing or apnea.

**Non-Localizing Signs**
- Stretching of VI nerve - diplopia.
- Compression of III nerve - pupillary dilatation and loss of ocular movements; initially ipsilateral side.
- Compression of the cerebral peduncle due to brain herniation can result in hemiplegia.

**Further Reading**
Status Epilepticus

Status epilepticus may be defined as:
- Any child having a seizure lasting for more than 10 minutes
  Or
- Any child brought with a seizure to hospital
  Or
- Having a series of seizures without return to baseline mental status between attacks.

For the purposes of quick identification and timely treatment to prevent brain damage, the second definition is used as the working one in practice.

**Step 1: Benzodiazepines (BZDs)**

Lorazepam (LRZ) 0.1 mg/kg, maximum 2 mg, slow intravenous (IV) route. The same dose can be given via the intraosseous, per rectal or buccal route.

Or

Diazepam (DZ) or midazolam (MDZ) 0.2 mg/kg can be given, slow IV route. The same dose can be given by intraosseous route. Rectal dose is 0.3–0.5 mg/kg.

Respiratory depression or hypotension can occur and this needs to be monitored closely (out of hospital intranasal or buccal MDZ is an easily available option).

The older protocols advocated repeat of the BZD. However, studies have shown that this does not add much to seizure control and adds to respiratory depression and time wastage. Hence if the seizures do not stop in another 10 minutes, go to step 2.

**Step 2**

- Phenytoin 20 mg/kg IV by slow push over 20 minutes.
  Or
Fosphenytoin 20 mg/kg of phenytoin equivalents IV, given by slow push over 10–15 minutes. This dose can also be given by the intraosseous (IO) route. If seizure does not stop within 5 minutes after the dose is complete, go to step 3.

This is also to be used if diazepam is the first BZD used, but not if LRZ is used.

- Phenobarbitone should be given 20 mg/kg IV. It can cause respiratory depression and hypotension particularly if given with benzodiazepines. If required, secure airway with endotracheal intubation. This would be the first choice in neonates and infants.

If seizures continue, then go to step 4; patient is in refractory status epilepticus (RSE). One of the following drugs could be given depending on the patient’s evaluation and availability of resources.

A general caveat is that it is best to optimize the levels of one drug before adding another and hence a repeat dose of these, may be given.

**Step 3: RSE**

1. IV valproic acid 30–35 mg/kg diluted with Normal saline (N saline). To be administered over 20 minutes as profound hypotension can occur (Newer protocols advocate the use of valproate in place of phenytoin and/or phenobarbitone as showing faster and longer seizure control in adult studies).

   Note: Centers not having the facilities for life support should transfer patients at this stage as the need may arise at any time.

2. Levetiracetam at a dose of 20 mg/kg loading infusion and then 10 mg/kg Q12 h can also be tried where intubation and ventilation need to be deferred for transport.

   However, by now, 1 hour may have passed and the clock is ticking so if seizures are not aborted, coma producing therapies need to be started.

**Step 4: Midazolam Infusion**

Loading dose of 0.2 mg/kg followed by infusion of 2–6 mcg/kg/min. Amount of 3 mg/kg of midazolam (MDZ) is added to 50 cc N saline, when given 1 cc/kg will deliver 1 mcg/kg/min. Start at low dose and increase by 1 mcg/kg/min every 15 minutes until control is achieved. Maximum rate is 20 mcg/kg/min or till hemodynamic instability is a problem to manage. Once control is achieved, maintain the same dose for 24 hours and then wean by 1 mcg/kg/min every 2–3 hours. Transfer to a center capable of long-term life support is needed as the respiratory depression and hemodynamic instability is unpredictable and varies from patient to patient.
Propofol 2–4 mg/kg bolus, then 1–3 mg/kg/h not recommended for children below 12 years of age. Approval for this drug is for usage for 12 hours only so informed consent before usage is advised.

**Thiopentone Infusion**

This general anesthesia is reserved for severe refractory status. Patient should be intubated and ventilated prior to starting the infusion and inotropes kept on standby. Invasive BP monitoring and real-time EEG monitoring will be needed, at least intermittently if not continuously. A separate IV line is needed. The loading dose is 3–5 mg/kg slowly immediately followed by an infusion of 1–5 mg/kg/min. Hemodynamic support in terms of extra fluids, vasopressors and inotropes may be required. Hence this needs to be done within a fully equipped PICU. Burst suppression with 6–8 bursts per minute is the target and hence it is foolhardy to try this without EEG monitoring.

**Step 5: Anesthetic Agents**

Once this stage is reached, the mortality and morbidity of RSE is more than 50%. These agents need to be delivered through a proper circuit and monitoring is done by an anesthetist who understands the drug. Aborting the seizures is usually easy but maintenance and survival are a universal issue.

In addition to these drugs, oral drugs like topiramate can be started. Other drugs that have been tried with success are as follows:

- **Lidocaine** 1.5–2 mg/kg IV over 2 minutes then can give a drip at 3–4 mg/min. This is the same class of drugs as phenytoin and an excellent membrane stabilizer. Neonatal studies have shown this drug to be effective. MDZ infusion as in older children should also be used in neonates because the same principles of quick resolution apply.
- **Pyridoxine** (vitamin B₆) should be given in all neonates and infants with resistant seizures (B₆ responsive seizures). Dose is 100 mg IV.

Tapering of any infusion should only be done after complete electrical seizure freedom for more than at least 24 hours. Very gradual tapering should be done as seizures will return and will often be non-convulsive and only be caught by EEG monitoring. The most toxic drug or last introduced one should be removed first. Hence long-term agents should be on board and all levels are well maintained before new drugs are added or tapering is begun.
IAP Convulsive Status Epilepticus Management Algorithm (2008)

Convulsive status epilepticus
- GTCS more than 5 minutes
- Not regained consciousness between two episodes of seizure

Stabilize
- Airway breathing
- Circulation
  - Get IV access
  - Blood sugar level
  - Take samples

0 Minutes
- Inj. lorazepam IV 0.05–0.1 mg/kg, or
- Diazepam 0.2 mg/kg, or Midazolam 0.2 mg/kg

10 Minutes
- Repeat inj. lorazepam, or Diazepam, or
- Midazolam at same doses

30 Minutes
- Inj. phenytoin 20 mg/kg at the rate of 1 mg/kg/min, or
- Inj. phosphonytioin 30 mg/kg at the rate of 3 mg/kg/min

40 Minutes
- Inj. Phenytoin: 10 mg/kg second dose, or
- Inj. Phosphonytioin: 15 mg/kg second dose

60 Minutes
- Intubate (RSI) put on ventilator
- Start midazolam infusion at the rate of 1–24 mcg/kg/min
- Increase dose every 15 minutes

ABCs stabilized seizures controlled
- Place child in recovery position
- Monitor vitals
- Shift to war I
- Investigate cause EEG

Rule out nonconvulsive status epilepticus

Further options
- Inj. sodium valproate

ICU Setting

Refactory status epilepticus

Contd...
IAP Algorithm for the Management of Neonatal Seizure (2008)

Maintain ABC and temperature

Withdraw blood for biochemistry

Immediate glucose by dextrostix

Correct glucose and calcium

Administer IV, phenobarbital 20 mg/kg

Repeat in 5 mg/kg boluses till a maximum of 40 mg/kg, every 15 minutes if seizure continues

IV phenytoin 15–20 mg/kg diluted in equal volume of normal saline at a maximum rate of 1 mg/kg/min over 35–40 minutes

IV lorazepam (0.05–0.1 mg/kg) or diazepam (0.25 mg/kg bolus or 0.5 mg/kg rectal)

Or

IV midazolam as a continuous infusion (an initial IV bolus of 0.15 mg/kg, followed by continuous infusion (1 µg/kg/min) increasing by 0.5–1 µg/kg/min every 2 minutes until a favorable response or a maximum of 16 µg/kg/min

100 mg pyridoxine IV or oral (if IV not available) should be given
# Cardiovascular Drug Protocol

## Inotropes and Vasopressors

### Adrenergic Receptors

<table>
<thead>
<tr>
<th>Receptor</th>
<th>Physiological response</th>
<th>Agonist</th>
</tr>
</thead>
<tbody>
<tr>
<td>α 1</td>
<td>Vasoconstriction</td>
<td>E &gt; NE &gt; D</td>
</tr>
<tr>
<td>α 2</td>
<td>Vasodilation</td>
<td>E &gt; NE</td>
</tr>
<tr>
<td>β 1</td>
<td>Inotropy, chronotropy</td>
<td>I &gt; E ≥ D ≥ NE</td>
</tr>
<tr>
<td>β 2</td>
<td>Vasodilation, bronchodilation, smooth muscle relaxation</td>
<td>I ≥ E &gt; D &gt; NE</td>
</tr>
<tr>
<td>D1</td>
<td>Smooth muscle relaxation</td>
<td>D</td>
</tr>
</tbody>
</table>

**Abbreviations**: E, epinephrine; NE, norepinephrine; D, dopamine; I, isoproterenol

### Major Hemodynamic Effects of Adrenergic Receptor Activation

<table>
<thead>
<tr>
<th>Agent</th>
<th>α 1</th>
<th>β 1</th>
<th>β 2</th>
<th>D1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dopamine*</td>
<td>Vasoconstriction, ↑ SVR</td>
<td>Inotropy, chronotropy</td>
<td>Vasodilation</td>
<td>Vasodilation (renal)</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>Vasoconstriction, ↑ SVR</td>
<td>Inotropy (minor)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Epinephrine#</td>
<td>Vasoconstriction, ↑ SVR</td>
<td>Inotropy, chronotropy</td>
<td>Vasodilation</td>
<td>—</td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>—</td>
<td>Inotropy</td>
<td>Vasodilation</td>
<td>—</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>—</td>
<td>Inotropy</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Milrinone</td>
<td>—</td>
<td>Nonreceptor mediated inotropy and vasodilation</td>
<td>—</td>
<td></td>
</tr>
</tbody>
</table>

**Abbreviations**: SVR, systemic vascular resistance.

* Dose related: At low infusion rates, D1 receptor effects predominate; at intermediate rates, β receptor effects predominate; and high rates, α receptor effects predominate

# Dose related: At low infusion rates, β receptor effects predominate; at high rates, α receptor effects predominate
**Dopamine**
- Low infusion (1–5 mcg/kg/min)—renal vasodilation (D1)
- Intermediate (6–10 mcg/kg/min)—inotrope (β1)
- High (11–20 mcg/kg/min) vasopressor and inotrope (α 1 and β1)
- Recommended as the first line agent for children with fluid refractory septic shock
- Used to treat cardiogenic or distributive shock with hypotension.

**Epinephrine**
- < 0.3 mcg/kg/min—inotrope and vasodilator (β 1 and β 2)
- > 0.3 mcg/kg/min—vasopressor and inotrope (α 1 and β 1)
- Used to treat shock when hypotension exists with low cardiac output (cold shock) unresponsive to dopamine
- It is also the agent of choice for hypotension or shock following successful treatment of cardiac arrest.

**Norepinephrine**
- Agent of choice in children with low blood pressure and a normal or elevated cardiac index (warm shock) refractory to dopamine
- Elevates SVR (α 1) which causes bradycardia by reflex vagal activity. So it is most valuable in context of tachycardia with shock
- Mainly used in septic and distributive shock (low SVR) with good cardiac output.

**Dobutamine**
- Agent of choice in patients with congestive heart failure (CHF) and a normal or mildly decreased blood pressure
- Can be used in septic shock when primary problem is complicated by myocardial dysfunction. In these patients concomitant use of vasopressors such as norepinephrine may be appropriate.

**Milrinone**
- Phosphodiesterase III inhibitor
- Produce positive inotropic and lusitropic (diastolic improvement) effects as well as vasodilation
- Used in isolated myocardial dysfunction such as CHF and may be alternative to coadministration of dobutamine and an after load reducing agent
- Too rapid infusion during loading dose produces hypotension. This problem is exacerbated in volume depleted patients.

**Vasopressin**
- It is pure vasopressor
- Used mainly in catecholamine refractory vasodilatory shock with good cardiac function
- Should not be used in patients with impaired myocardial function.
Digoxin
Role of digoxin in acute care of critically ill children has diminished due to narrow therapeutic range, slow onset of action, and potential for life threatening complications.

Doses (Infusion Rates in Mcg/Kg/Min)

<table>
<thead>
<tr>
<th>Agent</th>
<th>Inotropic</th>
<th>Pressor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dopamine</td>
<td>2–15</td>
<td>&gt; 10</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>0.05–0.5</td>
<td>0.3–1.0</td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>—</td>
<td>0.05–1.5</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>—</td>
<td>0.02–0.04*</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>2.5–20</td>
<td>—</td>
</tr>
<tr>
<td>Milrinone#</td>
<td>0.25–1.0</td>
<td>—</td>
</tr>
</tbody>
</table>

* Units/kg/min
# Loading dose 50 mcg/kg should be given over 15 min. Withhold loading dose in the event of hypotension. Adjust dose in renal dysfunction

Dilution
For dopamine, dobutamine
- 6 mg/kg in 100 ml of NS/5% dextrose gives 1ml/hr = 1 mcg/kg/min
- Up to double concentration (1 ml/hr = 2 mcg/kg/min) can be given through peripheral line. Higher concentration should be given through central line only (max 1 ml/hr = 5 mcg/kg/min).

For epinephrine, norepinephrine, and milrinone
- 0.6 mg/kg in 100 ml of NS/5% dextrose gives 1ml/hr = 0.1 mcg/kg/min.

Selection of Agents for Different Hemodynamic Disturbances

<table>
<thead>
<tr>
<th>Hemodynamic pattern</th>
<th>BP normal</th>
<th>BP decreased</th>
<th>BP increased</th>
</tr>
</thead>
<tbody>
<tr>
<td>Septic shock warm</td>
<td>Norepinephrine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Septic shock cold</td>
<td>Dobutamine</td>
<td>Dopamine/Epinephrine</td>
<td>Dobutamine + Vasodilator or Milrinone</td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>Dobutamine/Milrinone</td>
<td>Dopamine/Epinephrine</td>
<td>Dobutamine + vasodilator or Milrinone</td>
</tr>
<tr>
<td>Myocardial dysfunction</td>
<td>Dobutamine/Milrinone</td>
<td>Dopamine/Epinephrine</td>
<td>Dobutamine + vasodilator or Milrinone</td>
</tr>
</tbody>
</table>
**Common Notes**

- Inotropes and vasopressors should be given only after intravenous volume repletion.
- Catecholamine should not be given with alkaline solution like soda bicarbonate.
- Dopamine infusion > 10 mcg/kg/min, epinephrine > 0.3 mcg/kg/min and norepinephrine infusion are preferably given through a central line.
- All inotropic agents increase myocardial oxygen consumption (decrease efficiency) except milrinone.
- All catecholamines have half-life of 1–2 minutes only. So peak will come after 10 min of starting of infusion and effect of infusion will be there up to 10 min when infusion stopped.
- Extravasation of vasopressors agents may cause ischemia and necrosis of skin. Phentolamine should be given 5–10 mg in 10 ml NS with fine hypodermic needle.
- Inotropes should not be given in patients with dynamic left outflow obstruction (hypertrophic aortic stenosis).
- Arbitrary value of blood pressure is not the end point of therapy in shock. The lowest infusion rate that improve perfusion as judged by skin color, temperature, CFT, mental status, urine output and plasma lactate, should be used.

**Further Reading**

Tachyarrhythmia *(Also see Chapter 66)*

**Two Types**
- Narrow QRS complex: AVNRT, AVRT, Atrial flutter
- Wide QRS complex: VT, SVT (with aberrant conduction, with BBB, with antidromic conduction/WPW)

Wide complex tachycardia should be treated as a VT until proven otherwise.

Investigations: K, glucose, Ca, Mg, ABG.

**Management**

**A. With Adequate Perfusion (Stable)**
Arrhythmias

* Because adenosine has very short half-life (<10 sec), administer as rapidly as possible by rapid flush technique (use 2 syringes connected to T connector, give adenosine rapidly with one syringe and immediately flush with >5 ml saline filled in the second syringe)

**Abbreviations**: RR, respiratory rate; HR, heart rate; IV, intravenous; SVT, supraventricular tachycardia; VT, ventricular tachycardia

**B. With Poor Perfusion (Unstable) (Also see Chapter 66)**

![Diagram of Arrhythmias](image_url)
**Search for Reversible Causes and Treat: 6H, 5T**

Hypovolemia, Hypoglycemia, Hypothermia, Hypoxia, Hypo/Hyperkalemia, Hydrogen (acidosis)

Toxins (drugs), Tamponade, Tension pneumothorax, Thrombosis, Trauma (hypovolemia, ↑ ICP—Any IV drug can be given by intraosseous route also.

**Bradyarrhythmias**

- **Sinus Bradycardia**
  - PR interval normal
  - HR < 100/min NB < 60/min child

- **AV Block**
  - 1° block: increase PR interval only and constant
  - 2° block: type 1 (Wenckebach's) block at AV node progressive prolongation of PR interval followed by dropped beat
  - Type 2 blocks below AV node. PR interval is normal and fixed. Intermittent fixed ratio block 2:1, 3:1
  - 3° block: complete AV dissociation

**Isolated 1° and 2° type 1 do not require treatment**

2° type 2 and 3° always pathological and require treatment

**Bradycardia with poor perfusion**

- ABC: Oxygen
- Volume resuscitation

Still bradycardia and if HR < 60/min with poor perfusion then start CPR

- Epinephrine 0.01 mg/kg IV or 0.1 mg/kg ET
- Repeat every 3–5 min
- Cont. CPR

If increased vagal tone then give atropine 0.02 mg/kg IV (may repeat) (min. dose 0.1 mg, max. dose 1 mg)

**Cardiac pacing**

Abbreviations: HR, heart rate; IV, intravenous; CPR, cardiopulmonary resuscitation; AV, atrioventricular
Further Reading


Rhythm Disturbances Algorithm

Contd...
Abbreviations: IV, intravenous; CPR, cardiopulmonary resuscitation; IO, intraosseous; AED, automated external defibrillator; SVT, supraventricular tachycardia; VT, ventricular tachycardia; VF, ventricular fibrillation; PEA, pulseless electrical activity; ECG, electrocardiography

Figs 24.1A to C
Figs 24.1D to I
Figs 24.1A to M: Common rhythm disturbances
A: Normal sinus rhythm rate 100/min
B: Sinus bradycardia rate 45/min
C: Sinus tachycardia rate 180/min
D: Sinus rhythm with first degree heart-block (PR interval is prolonged)
E: Second degree heart block Mobitz I Wenckebach
F: Second degree heart block Mobitz II
G: Third degree heart block with escape rhythm
H: Supraventricular tachycardia rate 230/min
I: Atrial flutter
J: Ventricular tachycardia rate 150
K: Polymorphic ventricular tachycardia (Torsade-de-pointes)
L: Ventricular fibrillation
M: Tall peaked T waves (Hyperkalemia)
Diabetic Ketoacidosis Protocol

Dheeraj More, KG Ravikumar, Bala Ramachandran

**Diabetic Ketoacidosis: Management**

**Clinical History**
- Polyuria
- Polydipsia
- Weight loss
- Abdominal pain
- Weakness
- Vomiting
- Confusion
- Rapid respiration

**Clinical Signs**
- Dehydration
- Deep sighing respiration (Kussmaul)
- Lethargy, drowsiness

**Biochemical Signs**
- Ketones in urine or blood
- Elevated blood glucose (> 200 mg%)  
- Acidemia (pH < 7.3)
- Collect blood for RFT, CBC, SGOT, SGPT, blood C/S and urine C/S (if evidence of infection)

Confirm diagnosis of diabetic ketoacidosis

- Mild: pH < 7.3, bicarbonate < 15 mmol/L
- Moderate: pH < 7.2, bicarbonate < 10 mmol/L
- Severe: pH < 7.1, bicarbonate < 5 mmol/L

- Shock
- Reduced peripheral pulse volume
- Reduced conscious

- Dehydration > 5%
- Not in shock
- Clinically acidic
- Vomiting

- Dehydration < 5%
- Clinically

**Resuscitation**
- Airway ± NG tube
- Breathing (100% O₂)
- Circulation (10–20 ml/kg of 0.9% NS over 30–60 min)
- Repeat if necessary—initial expansion should not exceed total 30 ml/kg

**Intravenous Therapy**
- Calculate fluid requirements (maintenance + deficit)
- Correct over 48 hrs with 0.9 NS for first 8–12 hrs followed by 0.45% NS (Add KCl 40 mEq/L)
- Start insulin infusion 0.1 U/kg/hr
- ECG for hypo/hyperkalemic changes

- Start with subcutaneous insulin
  - 0.25 U/kg 3–4 hourly

Conflict...
Abbreviations: RFT, renal function tests; CBC, complete blood count; SGOT, serum glutamic oxaloacetic transaminase; SGPT, serum glutamic pyruvic transaminase; NG, nasogastric tube; NS, normal saline; ECG, electrocardiography

Notes

- Subcutaneous insulin indicated when
  - Acidosis and dehydration are minimal
  - Intensive follow-up of patient is certain

- Fluid requirement
  - For fluid requirement (maintenance and dehydration) the Milwaukee protocol can be used for children of all ages and with all degrees of diabetic ketoacidosis (DKA). Use 0.9 NS for atleast first 4–6 hours and preferably upto 12 hours
  - Corrected sodium level should rise as blood glucose levels fall during treatment. If blood glucose level does not fall, then continue with 0.9 NS and do not change to 0.45 NS

\[
\text{True S. Na} = \text{S. Na} + \frac{(\text{glucose} - 100) \times 1.6}{100}
\]

- A standard water deficit of 85 mL/kg (8.5% dehydration) is assumed. The rehydration fluid used usually is 0.45 NS
Diabetic Ketoacidosis Protocol

For example when planned to correct dehydration over 48 hrs:

\[
IV\ rate\ per\ hour = \frac{85\ ml/kg + \text{maintenance for 48 hrs} - \text{bolus infusion}}{48}
\]

Example: For 30 kg child, 1st hour = 300 ml NS

\[
2\text{nd hour onwards} = \frac{(85\ ml \times 30) + 3400\ ml - 300\ ml}{48}
\]

= 118 ml/hr (0.9 NS for at least first 4 to 6 hours, preferably up to 12 hours, then shift to 0.45 NS and add inj. KCl 40 mEq/L)

- **Potassium supplementation**
  - If the patient is hypokalemic, start potassium replacement at the time of initial volume expansion and before starting insulin therapy
  - Otherwise, start replacing potassium after initial volume expansion and concurrent with starting insulin therapy
  - If the patient is hyperkalemic, defer potassium replacement therapy until urine output is documented

- **Initial infusion bolus of 0.1 U/kg is no longer recommended in children because it does not speed recovery and may increase risk of hypokalemia and hypoglycemia**

- **Use of bicarbonate to correct acidosis is not recommended. Bicarbonate may worsen acidosis within the central nervous system and cause potassium shifts that can provoke arrhythmias**

  The practical outcome of patients with DKA is not improved by bicarbonate administration. Bicarbonate is reserved for patients with pH < 6.9, persisting after the first hour of hydration or those having impending cardiovascular collapse despite adequate fluid and insulin therapy.

**Further Reading**

“Severely injured children have improved outcomes if managed in designated pediatric centers by well organized, skilled multidisciplinary teams” (Potoka et al. 2000, 2001).

Broselow Pediatric Resuscitation Measuring Tape is essential adjunct for the rapid determination of weight based on length for appropriate drug doses and equipment size. BE PREPARED!

**Anatomic and Physiologic Differences**

**General Considerations**

**Size and Shape**
- Greater distribution of force per unit body area because of the smaller body mass
- Energy is transferred to a body that has less fat, less elastic connective tissue and close proximity of multiple organs
- Result: multiple organ injuries
- Larger surface area relative to volume predisposes children to thermal evaporative loss
- Result: hypothermia.

**Skeleton**
- More pliable skeleton due to incomplete calcification
- Result: Serious organ injury without overlying skeletal fracture
- Caution: If rib fractures identified anticipate serious organ injury as the force must have been great
- Multiple active growth centers
- Result: Unique fractures with potential growth arrest/abnormality.
Surface Area

- Disproportionate ratio of body surface area (BSA): Body volume (highest at birth and then diminishes) Result: Maintenance requirements for free water, trace elements, and minerals are magnified
- Vital functions assessed as per age normals.

Primary Survey

Goals

- Identify immediate/potential threats to life and initiate treatment.

Structure of the Pediatric Airway

- Passive flexion due to large occiput → Airway obstruction
- Relatively larger tongue → Airway obstruction
- Mass of adenoidal tissues → NP airways difficult to pass
- U-shaped, floppy epiglottis → May necessitate use of a strait blade
- Anterior and cephalad larynx → Visualization more difficult
- Airway narrowest at the cricoid ring → Uncuffed tubes up to mm or ~ 8 years
- Narrow tracheal diameter and distance between the rings → Needle circothyroidotomy over surgical for a difficult airway
- Short tracheal length (4 cm NB; 8 cm toddler) → Right main stem (RMS) intubation and dislodgement
- More narrow large airways → Greater airway resistance ($R = 1/r^4$)

Airway with C-Spine Stabilization

Inability to establish and maintain a patent airway with subsequent hypoxemia and hypercapnea is the most common cause for pediatric cardiac arrest.

Airway Assessment

Patency

Assess for obstruction due to: position, injury, blood, teeth, vomitus, foreign object

- Level of consciousness
- Maxillofacial injury
- Stridor or cyanosis.

Management

Noninvasive Airway Adjuncts

- Neutral position: shoulder role in infancy
- Alleviate obstruction: chin lift or jaw thrust
- Bag mask +/- oral airway: Jackson-Reese.

Invasive Airway Adjuncts

- Oxygenate before attempting to establish a definitive airway
  - Orotracheal intubation +/- Rapid sequence intubation (RSI)
Needle cricothyroidotomy versus surgical cricothyroidotomy → Needle cricothyroidotomy preferred in children ≤ 8 years

Cautions for a definitive airway
- Lack of a patent/protected airway
- Impending loss of the airway
- Comatose patient glasgow coma scale (GCS) < 9
- Inability to oxygenate
- Inability to ventilate
- Full/impending cardiac arrest
- Head injury.

Indications for a definitive airway
- Lack of patent/protected airway
- Impending loss of airway
- Comatose pt GC < 9
- Inability to oxygenate
- Inability to ventilate
- Full/impending cardiac arrest

Method

Rapid Sequence Intubation
- Infant 40–60 bpm
- Child 20 bpm
- Adolescent < 20 bpm
- Diaphragm is easily fatigued and easily displaced → Nasogastric tube is essential to eliminate restrictive defect
- Marked compliance of their chest wall → Alveolar collapse
- Smaller functional residual capacity (FRC) → Hypoxia develops rapidly

Breathing

Assessment
- Rate
- Chest wall movement: Paradoxical breathing; flail segment
- Oxygen status
- Percussion note
- Tracheal deviation
- Crepitus
- Open wounds.

Management
- Warm humidified oxygen
- Gastric decompression
- Appropriate mechanical ventilation → avoid barotrauma and volutrauma
- Chest tube(s) if indicated
Circulation

Hemodynamics
- Blood volume = 70–90 ml/kg
- Systolic blood pressure (SBP) = 70 + 2 (age in years)
- Diastolic blood pressure (DBP) = 2/3 SBP

Pediatric vasculature readily constricts and increases systemic vascular resistance (SVR) to maintain perfusion. Tachycardia and poor skin perfusion may be the only keys to hypovolemia. Hypotension in a child leads to decompensated shock and estimated blood loss \( \geq 45\% \) and may be abruptly followed by bradycardia.

Circulation with Hemorrhage Control

Assessment
- Heart rate
- Systolic BP and pulse pressure
- Peripheral pulses
- Skin condition/perfusion/capillary refill
- Sensorium
- Deadly bleeding checks.

Management
- External Hemorrhage Control
  - Direct pressure
- Vascular Access
  - Aim for adequate access above and below the diaphragm
  - Trauma labs drawn at the time of vascular access
  - Peripheral percutaneous (2 attempts or 90 sec)
- Large-bore
  - Percutaneous femoral > subclavian > jugular access
  - Venous cutdown
  - umbilical stump in an infant < 2 weeks of age.
- Intraosseous Access
  - Maximal flow rate 25 ml/min
  - Contraindicated at site of fractures
  - Should not be performed distal to a fracture site or in a devitalized limb
- Fluid resuscitation
  - 20 ml/kg warmed NS = first line therapy
  - Recall the 3:1 rule of crystalloid resuscitation
  - Consider packed red blood cells (PRBCs) if: 10 ml/kg of type specific or O negative warmed PRBCs
    - Obvious exsanguination injury
    - Initiating third bolus
- "Best" fluid remains controversial
Saline or Ringers are likely most suitable
Both colloid and crystalloid maybe effective without propensity to adverse outcome.

Optimal Response to Resuscitation
- Slowing of HR
- Increased pulse pressure (PP) (> 20 mmHg)
- Normal skin perfusion
- Improvement in level of consciousness
- SBP > 80 mmHg (age based)
- U/O = 1–2 ml/kg/hr.

Disability Assessment
- AVPU/Modified GCS
- Pupil size and reactivity
- Extremity movement and tone
- Posturing
- Reflexes.

Management
Address findings in keeping with increased intracranial pressure (ICP) and/or spinal cord injury (SCI).

Exposure with Environmental Control
Assessment
- Logroll to examine back
- Look under hair, collar and splints.

Management
- Overhead heat lamps, thermal blankets, convective air warmers must be considered in the infant and toddler as hypothermia greatly increases metabolic needs
- Mortality approaches 100% in trauma patients that start euthermic and become hypothermic (< 32°C).

Psychosocial
- A child’s ability to cope with pain and interact in a frightening and threatening environment is limited
- Families cope differently
- Primary Survey.

Family/child needs: Determine if family wishes to be present at the resuscitation
- Rapidly inform the family of what has occurred
- Dedicated team member assigned to the family.
**Secondary Survey**

It commences once the injured child is stabilized.

**Goals**

- Identify any new/potential threats to life and limb
- Identify injuries that may impact the child later (e.g. minor fractures, dislocations and lacerations)
- Obtain a more complete history
- Continuous monitoring (HR, BP, T, SaO₂ +/- etCO₂)
- Achieved through a well-organized head to toe examination
- Placement of Foley catheter
- Catheter with inflatable retaining device only when child > 15 kg
- Secondary survey contd
- Review labs
- Radiology
  - Chest, pelvis, lateral c-spine if not done with primary survey
  - Additional images on the basis of threat to life/limb
- Tetanus toxoid
- Antibiotics
- Analgesia (systemic and local) continuous reassessment; tracking of vitals and urine output.

**Head Trauma**

- Leading cause of death
- Results in significant morbidity
- Hypotension and hypoxia from concurrent injury adversely affect the outcome from intracerebral injury.

**Neck**

- Cervical supporting ligaments and joint capsules are more flexible
- Vertebral bodies are wedged anteriorly and tend to slide forward with flexion
- Horizontal facet joints
- Fulcrum of flexion is higher.

**SCIWORA**: spinal cord injury without radiological abnormality

- If SCI is suspected on the basis of mechanism or clinical exam do not be dissuaded by normal X-rays
- A normal X-ray and initial exam is not enough.

Be aware of local pain, torticollis.

**Management**

- Avoid secondary and iatrogenic injury
- Corticosteroids are currently not recommended.
Chest

Pliable chest wall → Transmission of force within the thoracic skeleton to the pulmonary parenchyma.

Mobility of the mediastinal structures → Vulnerability to tension pneumothoraces and vascular disruptions. Rib fractures are a marker of significant injuring force and occur with < 50% of chest trauma.

Clinical Features

- Increased RR
- Abnormal thoracic examination
- Abnormal chest auscultation
- GCS < 15
- Low SBP
- Femur fracture.

Pattern of Injury

- Pulmonary contusion is common +/- direct intrapulmonary hemorrhage or pneumothorax, usually without rib fractures
- Rare are diaphragmatic rupture, aortic transection, major tracheobronchial tears, flail chest, sternal fractures and cardiac contusion
- Anatomic and physiologic differences—abdominopelvic
- Anterior placement of the liver and spleen; less protective musculature and subcutaneous tissue mass → Internal organs are more susceptible to injury
- Kidneys are relatively mobile without protective fat → Kidney is very susceptible to deceleration force.

Abdominal Trauma

- Clinical exam improved with gastric and bladder decompression
- Abdominal exam is insensitive in the presence of distracting injury.

Diagnostic Adjuncts

- Computed Tomography
  - Only suitable for the stable child
  - Less sensitive for bowel, spleen, early pancreas, liver.
- Ultrasound
  - Good for hepatic and splenic injury.

Visceral injuries specific to the pediatric population

- Pancreatic injury
- Small bowel perforation (ligament of Treitz)
- Mesenteric and small bowel avulsion
- Duodenal hematoma
- Spleen, liver and kidney disruption
- Bladder injury.

Missed abdominal trauma is the leading cause of preventable morbidity and mortality.
Clinical findings associated with intra-abdominal injury:
- Initial Hct < 30%
- Abdominal tenderness
- Femur fracture
- AST > 200 U/L and/or ALT > 125 U/L
- UA with > 5 RBCs/hpf.

Indications for Surgery
- Hemodynamic instability despite maximal resuscitative measures +/- a positive diagnostic study (US or CT)
- Falling hematocrit
- Transfusion of greater than 50% of total blood volume
- Radiographic evidence of pneumoperitoneum, intraperitoneal bladder rupture, grade V renovascular injury
- Peritonitis or the development to peritoneal signs
- Evisceration of intraperitoneal contents
- Evidence of fecal or bowel contamination on DPL.

Nonoperative approach mandates PICU accessibility.

Primary survey meant to discover life threatening conditions and stabilize patient

Airway
- Maintainable or definite airway
- Facial injuries
- Cx spine stabilization open with chin lift/jaw thrust
- Stable → continue survey
- Unstable → RSI

Breathing
- Rate, air entry
- Oxygen status
- Stable → continue unstable

Circulation
- HR, pulse vol BP
- Skin color
- Fluid boluses x 20 ml/kg x 2 PRBC ordered with 3rd bolus

Look for cause of blood loss: long bones, pelvic, abdominal, scalp only in < 2 years
FAST USG* (focused abdominal sonography for trauma) solid organ trauma
X-ray*: Cx spine, bones, abdomen, pelvis and chest

Reassess GCS and dropping conscious level could be head trauma or hypoxia from blood loss/lung injury.
Secondary survey meant after stabilization

When the child is stable as to head trauma and abdominal injury, fractures can be definitively tackled.

Pediatric Multiple Trauma Algorithm

- **Primary Survey**
  - Assess airway, breathing, circulation

- **Airway**
  - Stabilize cervical spine
  - Open airway using "jaw thrust" maneuver
  - Remove visible foreign material by finger sweep and wide bore suction device
  - Intubate if necessary using rapid sequence intubation if victim is not spontaneously breathing

- **Breathing**
  - If victim is spontaneously breathing, give oxygen by nasal cannula, face mask and self inflating bag or mouth to mouth
  - If victim is not spontaneously breathing, begin mechanical ventilation

- **Circulation**
  - Stop active bleeding by direct pressure, tourniquets, MAST (military antishock trousers)
  - Establish vascular access
  - Manage hypovolemic shock by colloids/crystalloids/blood

Contd...
Secondary Survey
Manage various organ system injuries

CNS Injury
- Manage as per head injury protocol
- Rule out cervical and spinal injury

Bony Injuries
- Stabilize fractures and control pain
- Do not try to reduce fracture

Chest Injuries
- Tension pneumothorax – Needle decompression
- Rib fracture– analgesia
- Hemothorax– chest tube insertion

Abdominal Injuries
- Look for presence of abdominal injuries
- Plan appropriate investigation

Plan Further Treatment
- Monitor child in PICU
- Take multidisciplinary consultation
Gastrointestinal (GI) bleed can be lower GI or upper GI. The outcome of an episode of active hemorrhage depends on:

- Control of active bleeding
- Avoidance of the major complications and its treatment
- Establishing the correct diagnosis.

**Approach to Upper Gastrointestinal Bleed**

Upper gastrointestinal (UGI) bleeding refers to bleeding occurring above the ligament of Treitz.

Melena refers to black, tarry stools. Melena can be produced by relatively small volumes of blood (50–100 ml) in the stomach and persist for 3–5 days and thus is not an indication of ongoing bleeding.

**Step 1: Confirm its Gastrointestinal Bleed**

Appropriate history and clinical examination is vital even in the emergency setting with emphasis on airway, breathing and circulation.

Red coloring agents and food like jellies, tomatoes and strawberries can give appearance of blood in plain vomitus. Even for melena occult blood testing is a useful option.

**Step 2: Confirm its Gastrointestinal**

Epistaxis, swallowed maternal blood in case of neonates (Apt test), blood-tinged sputum, and oropharyngeal bleeding are the probable conditions where swallowed blood may confabulate as its source as GI.

A quick head and neck and throat examination to rule out a local cause is needed.
Step 3: Immediate Assessment and Hemodynamic Resuscitation

- Secure large bore peripheral intravenous lines or a central line
- Use crystalloid as well as colloids
- Use blood packed cell volumes
- Measure hematocrit every 6–8 hours keeping hemoglobin (Hb) at 7–8 gm/dL
- Avoid overtransfusion as there is a risk of rebleed.

If the bleeding is severe, therapy must begin before the localization of the source. Significant GI bleeding will initially manifest as tachycardia, whereas hypotension occurs later. Correction of blood loss and improving the oxygen carrying capacity are equally important steps after securing airway, breathing and circulation.

With blood transfusion of approximately 85 ml/kg or greater, emergency exploratory surgery is indicated. Surgical consultation is mandatory in any case of severe UGI bleeding.

Blood should be kept cross matched and ready if required any time for the next 5 days. Try and use blood products conservatively and if UGI bleed is secondary to portal hypertension, keep Hb of 8 gm/dL.

Step 4: Pharmacologic Therapy

Drugs used depending on etiology:
- Acid suppression
- Vasoactive agents
- Antibiotics.

*Acid Suppression*

Proton pump inhibitor therapy: High-quality evidence of receiving prophylactic treatment to prevent UGI bleeding is still limited but proton pump inhibitor (PPI) therapy is definitely useful for peptic ulcer, erosive gastritis and in general intensive care unit (ICU) patients with small or large bleeds of “stress ulcers.”

*Vasoactive Medications*

Somatostatin or octreotide; terlipressin, *dosage:
They are initiated as soon as variceal hemorrhage is suspected and continued for 3–5 days after the diagnosis is confirmed.
- Octreotide is better than placebo or vasopressin and has fewer side effects than vasopressin.
- Terlipressin is preferred where available since it is the only pharmacologic treatment associated with a reduction in mortality compared with placebo.

*Octreotide:* 1 µg/kg bolus followed by 1 µg/kg/hour. Titrate to response. Reduce by 50% every 12 hours after bleeding stops for 24 hours. Stop when dose is 25% of original.
Vasopressin/terlipressin: 0.002–0.005 units/kg/minute, maximum 0.001 units/kg/minute. Taper 12 hours after bleeding stops and taper over 24–48 hours.

**Antibiotics**

Short-term (maximum 7 days) antibiotic prophylaxis should be instituted in any patient with liver disease and GI hemorrhage.

**Step 5: Other Supportive Measures and Determination of the Cause**

- No role of ice saline lavage. Waste of time and the suction of the syringe can cause more bleeding
- **Laboratory:**
  - Complete blood count (CBC), prothrombin time (PT), partial thromboplastin time (PTT)
  - Liver function if suspected
  - Type and cross match for up to 5 days
  - Electrolytes
  - Blood urea nitrogen (BUN), creatinine, ammonia
  - Sepsis work up if warranted

- **Angiography:** Bleeding must be at least 0.5 ml/minute to be detected by angiography. In cases of massive UGI bleeding, angiography offers diagnostic and therapeutic use, e.g. embolization but experience in children is limited.

**Step 6: Endoscopy**

Esophagogastroduodenoscopy (EGD) is particularly useful in the diagnosis of gastritis, esophagitis, peptic ulcers, and Mallory-Weiss tears. Bancroft and colleagues found that 20% had esophageal inflammation, 17% had gastric mucosal abnormalities, 6% had peptic ulcers, 6% had varices, 2% had Mallory-Weiss tears, and 8% had nonspecific mucosal abnormalities.

An EGD done within 24 hours of initial symptoms leads to better outcome as compared with a delayed EGD. Control of the bleeding can also be achieved and there is excellent pediatric experience.

Banding appears to be much better tolerated in children compared with sclerotherapy, with less retrosternal pain and no fever.

A transjugular intrahepatic portosystemic shunt (TIPS) should be considered in patients in whom hemorrhage from fundal varices cannot be controlled or in whom bleeding recurs despite combined pharmacological and endoscopic therapy. Expert GI/interventional radiological help needs to be sought and appropriate transfers may need to be done.
In patients who rebleed after initial endoscopic hemostasis, repeat endoscopic therapy is recommended before considering surgical or radiologic intervention.

**Step 7: Refractory Bleed**
Transjugular intrahepatic portosystemic shunt is indicated in patients in whom hemorrhage from esophageal varices cannot be controlled or in whom bleeding recurs despite combined pharmacological and endoscopic therapy.

Balloon tamponade (Sengstaken-Blakemore tube) should be used as a temporizing measure (maximum 24 hours) in patients with uncontrollable bleeding for whom a more definitive therapy is planned. The problem in children is to find the correct size and fix it in the correct part of the esophagus. Incorrect placement is dangerous and can cause pressure necrosis and sloughing.

**Step 8: Surgery**
Surgical intervention for gastroesophageal varices requires a portosystemic shunting. It can be life saving and a surgeon should be informed and kept ready when bleeding is severe.

For perforation peritonitis secondary to any cause, e.g. peptic ulcer early surgical reference is advocated.

**Step 9: Remember Uncommon Causes**
Dieulafoy’s lesions, gastric antral vascular ectasia, portal hypertensive gastropathy, hemophilia, hemosuccus pancreaticus, aortoenteral fistulas, UGI tumors, and Cameron lesions.

Specific therapy directed to underlying cause is the major line of treatment. Endoscopic metallic clips have been used in UGI bleeding for Mallory-Weiss tears, ulcers, Dieulafoy’s lesions, gastric angiectasias, and gastric tumors.

**Approach to Lower Gastrointestinal Bleed**
The clinical presentation of lower gastrointestinal (LGI) bleeding depends on the rate and amount of blood loss and range from stools positive for fecal occult blood test to profound shock.

**Step 1: Confirm its Gastrointestinal Bleed**
Syrup medications, and gelatin desserts or of tomato skins, peach skins, and beets can give appearance of GI bleed. Iron preparations, bismuth subsalicylate, spinach, dark chocolate, purple grapes may present as melena. Proper history taking and thorough physical examination are of paramount importance. Fecal occult blood testing has been shown as an efficient screening aid for detecting GI blood loss.
Step 2: Initial Hemodynamic Stabilization
Same as in UGI bleed

Step 3: First Episode of Lower Gastrointestinal Bleed
- Stool culture and examination
- Ultrasonography
- Proctosigmoidoscopy
- Further investigations depending on clinical profile may be considered by the pediatric gastroenterologist.

Step 4: Recurrent Lower Gastrointestinal Bleed
- Colonoscopy
- Meckel scan
- Bleeding scan
- Capsule endoscopy
- Angiography.

Common Causes
- Anal fissures
- Intussusceptions
- Amebic colitis (25%)
- Meckel’s diverticulum
- Colonic + Juvenile polyps (50% + 13%)
- Inflammatory bowel disease
- Solitary rectal ulcer (10%)

Rare Causes
- Hemorrhoids
- Angiodysplasia
- Dieulafoy’s lesion
- Telangiectasia
- Angioma
- Hamartoma
- Hemangioma
- Hemangioendothelioma
- Blue rubber bleb nevus syndromes

Role of Endoscopy in Lower Gastrointestinal Bleed
- Colonoscopy is effective in the diagnosis and the treatment of lower gastrointestinal bleeding (LGIB) and for early evaluation of severe acute LGIB.
- Nasogastric (NG) tube placement and/or upper endoscopy to look for a UGI source of bleeding should be considered if a source is not identified on colonoscopy, particularly if there is a history of UGI symptoms or anemia.
- Thermal contact modalities, including heat probe and bipolar/multipolar coagulation, and/or epinephrine injection can be used in the treatment of bleeding diverticula, vascular ectasia, or postpolypectomy bleeding sites.
- Angiography and/or tagged-red-blood-cell scanning can be used in the setting of active, persistent bleeding or in cases of obscure and occult GI bleed.
- Preoperative localization of bleeding should be attempted in all patients before surgical intervention.
Further Reading

Acute Gastrointestinal Bleeding Algorithm

Lower GI bleed
- Meckel's diverticulum
- Polyp
- Volvulus
- Intussusception
- Inflammatory bowel disease (ulcerative colitis, Crohn’s disease)

Upper GI bleed
- Signs of shock
  - No
  - Yes

Determine source of bleeding

Mucosal bleed
- Antacids +/- sucralfate, treat underlying cause

Undetermined
- Specific imaging and diagnostic test
  - Vascular malformations and tumors

Variceal bleed
  - Hemodynamically stable
    - Yes
    - No
  - No facility for sclerotherapy or band ligation

Maintain intravascular volume, RBC/FFP ratio if required
Acute Gastrointestinal Bleeding Algorithm

Abbreviations: GI, gastrointestinal; RBC/FFP, red blood cell/fresh frozen plasma; CI, continuous infusion; IV, intravenous; TIPS, transjugular intrahepatic portosystemic shunt
Pediatric Multiorgan Failure (Liver and Kidney)

Case Scenario
One-year-old infant admitted in pediatric intensive care unit (PICU) with fever, shock and lethargy. After receiving fluid boluses and starting on inotropes, he was intubated and shifted to PICU. Investigations showed leucocytosis and thrombocytopenia with severe metabolic acidosis. Within 6 hours of admission into PICU urine output started coming down and he started bleeding from the nasogastric tube (NG). He had persistent tachycardia with cold extremities. Further investigations showed persistent metabolic acidosis, elevated liver enzymes (> 10,000) and severe coagulopathy [both prothrombin time (PT) and partial thromboplastin time (PTT) elevated] with rising serum creatinine.

Definitions

Definition of renal dysfunction: Serum creatinine > two times upper limit of normal for age or two-fold increase in baseline creatinine.

Definition of hepatic dysfunction: Total bilirubin > 4 mg/dL or alanine aminotransferase (ALT) two times normal for age.

Acute Kidney Injury in Multiple Organ Dysfunction

Definition of acute kidney injury: One of the many definitions described is an “abrupt decrease in glomerular filtration rate (GFR) of at least 50% from baseline with a corresponding 50% or greater increase in serum creatinine.” Because of lack of consensus as to which is the best definition and serum creatinine and urine output being late markers of renal injury, now intensivists prefer the pediatric modified risk, injury, failure, loss and end stage kidney disease (RIFLE) criteria to define and classify acute kidney injury.
**Pediatric Modified RIFLE Criteria (Table 29.1)**

The acronym RIFLE stands for the increasing severity classes—risk, injury, failure and the two outcome classes—loss and end stage kidney disease.

**Table 29.1: Pediatric modified RIFLE criteria**

<table>
<thead>
<tr>
<th>Serum creatinine criteria</th>
<th>Urine output criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Risk</td>
<td>eCCL decrease by 25%</td>
</tr>
<tr>
<td></td>
<td>UO &lt; 0.5 mL/kg/hour × 8 hours</td>
</tr>
<tr>
<td>Injury</td>
<td>eCCL decrease by 50%</td>
</tr>
<tr>
<td></td>
<td>UO &lt; 0.5 mL/kg/hour × 16 hours</td>
</tr>
<tr>
<td>Failure</td>
<td>eCCL decrease by 75% or eCCL &lt; 35 ml/min/1.73 m²</td>
</tr>
<tr>
<td></td>
<td>UO &lt; 0.3 mL/kg/hour × 24 hours or Anuria × 12 hours</td>
</tr>
<tr>
<td>Loss</td>
<td>Persistent failure &gt; 4 weeks</td>
</tr>
<tr>
<td>ESKD</td>
<td>End stage kidney disease (&gt; 3 months)</td>
</tr>
</tbody>
</table>

Abbreviations: eCCL, estimated creatinine clearance; UO, urine output; ESKD, end stage kidney disease

We can classify renal failure as prerenal and renal causes by using renal failure indices (Table 29.2).

**Table 29.2: Classification of renal failure**

<table>
<thead>
<tr>
<th></th>
<th>Prerenal</th>
<th>Renal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine sediment</td>
<td>Bland</td>
<td>Broad, brownish granular casts</td>
</tr>
<tr>
<td>Urine sodium (mEq/L)</td>
<td>&lt; 20</td>
<td>&gt; 30</td>
</tr>
<tr>
<td>Urine osmolality (mOsm/l)</td>
<td>&gt; 400</td>
<td>&lt; 350</td>
</tr>
<tr>
<td>Fractional excretion of Na (FeNa)</td>
<td>&lt; 1</td>
<td>&gt; 1</td>
</tr>
</tbody>
</table>

Fractional excretion of Na = \[
\frac{\text{Urine sodium} \times \text{Plasma creatinine} \times 100}{\text{Plasma sodium} \times \text{Urine creatinine}}
\]

**Principles of Management in Renal Dysfunction**

- Avoid nephrotoxic drugs.
- **Maintain kidney perfusion with fluids and inotropes**: Judicious use of fluids to prevent fluid overload and prevent further ischemic damage to kidney.
- Adjust drug dosages according to estimated creatinine clearance (eCCL).

Schwartz formula for calculating GFR:

\[
\text{GFR (ml/min/1.73 m²)} = \frac{k \times \text{height in cm}}{\text{Serum creatinine}}
\]

where \(k = 0.33\) for preterm infants, \(0.45\) in infants \(0.55\) in older children.

Schwartz formula is not accurate in a sick child with rapidly changing physiological status. Measuring creatinine clearance directly by using the formula,
U (urine creatinine) x V (volume of urine in ml/min)/P (plasma creatinine) x 1.73/body surface area (BSA) in m² is better estimate of GFR. In a sick child with oliguria and kidney injury, it is better to assume GFR < 10 while dosing.

- Early nutrition support: High calorie enteral diet with adequate protein is started early.
- Try to convert oliguric to nonoliguric renal failure if possible by using diuretics, if hemodynamically stable and there is a response to diuretics.
- Start renal replacement therapy early.

**Investigation and Monitoring**

Urine routine examination, renal function test with electrolytes, urine sodium, urine osmolality, urine specific gravity, arterial blood gas (ABG), electrocardiogram (ECG).

- Hourly intake output chart, daily weight if possible
- Hemodynamic monitoring
- Sixth hourly electrolytes, daily renal function test.

**Algorithm**
Renal Replacement Therapy

*Indications in Acute Kidney Injury*
- Oliguria/anuria with fluid overload, refractory to diuretic therapy.
- Persistent hyperkalemia not responding to other measures.
- Severe metabolic acidosis unresponsive to medical management.
- Severe electrolyte abnormality.

*Types of Dialysis (Table 29.3)*
- Peritoneal dialysis
- Intermittent hemodialysis
- Continuous renal replacement therapy (CRRT): Continuous venovenous hemofiltration (CVVH) or continuous venovenous hemodiafiltration (CVVHDF).

<table>
<thead>
<tr>
<th>Table 29.3: Types of dialysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type</td>
</tr>
<tr>
<td>Peritoneal dialysis</td>
</tr>
<tr>
<td>Intermittent hemodialysis</td>
</tr>
<tr>
<td>CVVH</td>
</tr>
<tr>
<td>CVVHDF</td>
</tr>
</tbody>
</table>

*Abbreviations: CVVH, continuous venovenous hemofiltration; CVVHDF, continuous venovenous hemodiafiltration*

The choice of renal replacement therapy depends on the clinical circumstances, availability of expertise and good vascular access, size of the child and hemodynamic stability.

*Peritoneal Dialysis*
Peritoneal dialysis is the easiest and most widely used modality. Solute clearance is by diffusion and solvent drag. Fluid removal happens by osmosis. It can be done through a catheter placed at the bed side or surgically placed Tenckhoff catheter. Dialysate volume of 10–20 ml/kg with dwell time of 30 minutes to 1 hour is a good starting prescription. Increasing the dextrose concentration of the dialysate, increasing dwell volume, shortening the dwell time and doing more cycles helps in more ultrafiltrate. Hypertonic dialysate fluid may cause hyperglycemia and rapid ultrafiltrate. Heparin 500 units/ml may be added to the dialysate fluid to prevent catheter block. Potassium can be added to the peritoneal dialysis (PD) fluid to a maximum of 4 mEq/L of PD fluid if there is hypokalemia. Dialysate can be changed to bicarbonate based instead of lactate based if there is severe lactic acidosis.
Intermittent Hemodialysis

Intermittent hemodialysis is done in hemodynamically stable children. Children with multiorgan dysfunction and shock may not be good candidates for it. The advantage of hemodialysis is the rapid removal of toxins and ultrafiltration of fluid.

Continuous Renal Replacement Therapy

Continuous venovenous hemofiltration (CVVH) is preferred in hemodynamically unstable children. Care should be taken to minimize the amount of blood in the extracorporeal circuit and blood priming of the hemofiltration circuit may be necessary at the outset. Fluid removal is adjusted according to the patient’s clinical state during the treatment. The extracorporeal circuit requires good central venous access, usually via a dual lumen catheter, to allow the high blood flows necessary to prevent clotting in the hemofilter. Suggested catheter sizes in French gauge (FG) are:

<table>
<thead>
<tr>
<th>Patient size (kg)</th>
<th>Vascular access</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.5–10</td>
<td>6.5 FG dual lumen (10 cm)</td>
</tr>
<tr>
<td>10–20</td>
<td>8 FG dual lumen (15 cm)</td>
</tr>
<tr>
<td>&gt; 20</td>
<td>10.8 FG or larger dual lumen (20 cm)</td>
</tr>
</tbody>
</table>

Blood volume in the extracorporeal circuit should be less than 10% of the patient’s circulatory volume. Blood flows of 6–9 ml/kg/minute or 8% of circulating blood volume prevents excessive hemoconcentration in the filter. Automated machines with appropriate accuracy for children are recommended for delivering the continuous renal replacement therapy (CRRT) prescription safely and have replaced pump-assisted hemofiltration using volumetric pumps.

If only fluid removal is required then relatively low rates of filtration are needed, often referred to as slow continuous ultrafiltration (SCUF). There will be negligible solute removal under these circumstances.

When more solute clearance is needed in addition to fluid removal, dialysis component is added to the CVVH to make it CVVHDF.

Hepatic Dysfunction in Pediatric Multiorgan Dysfunction

Definition of acute liver failure: The Pediatric Acute Liver Failure Study Group defined acute liver failure as:

- Biochemical evidence of liver injury.
- No history of known chronic liver disease.
- Coagulopathy not corrected by vitamin K administration.
- International normalized ratio (INR) greater than 1.5 if the patient had encephalopathy or greater than 2.0 if the patient does not have encephalopathy.
Laboratory Evaluation
Liver function test [bilirubin is not usually high, alanine transaminase (ALT)/aspartate transaminase (AST) can be in 10,000, AST is usually more than ALT], PT and INR, electrolytes, hourly glucose, serum ammonia.

Principles of Treatment
- Prevent further hepatic injury by not using hepatotoxic drugs.
- Prevent and treat hypoglycemia, and electrolyte abnormality.
- Early nutrition with low protein and high calorie (120–150% requirement) enteral feeds.
- Correct coagulopathy with blood products, only when there is bleeding or for invasive procedures.
- Look for and treat raised intracranial hypertension as part of hepatic encephalopathy—head end elevation to 30°, adequate sedation and analgesia is needed for children with hepatic encephalopathy and grade 3 or 4 encephalopathy. Intracranial pressure (ICP) monitoring is considered for patients who are listed for liver transplantation. Mannitol can be given for acute rise in ICP.
- Three percent saline is a better option in a child with shock and coexistent renal failure.
- N-acetyl cysteine: There is evidence favoring the use of N-acetyl cysteine infusion in children with non-paracetamol liver failure, but the use in septic shock and ischemic hepatic dysfunction has not been studied. Ischemic hepatic dysfunction usually responds well to correction of the shock.
- Lactulose, branched chain amino acids, enteral rifaximine and bowel wash have insufficient evidence for routine use.
- Liver-support devices may be used as a bridge to transplantation or to help recovery of the ailing liver. They have limited role outside of clinical trials. Two main categories of support devices exist, bioartificial and artificial MARS.
- Consider liver transplant if no improvement and where prognostic factors indicate a high likelihood of death. Liver dysfunction as part of septic shock and multiple organ dysfunction syndrome (MODS) improves on correction of shock and rarely requires transplant.

Further Reading
Acute Renal Failure Algorithm

Acute Renal Failure

Determine volume status

Euvolemic or hypovolemic

NS bolus 20 ml/kg over 30 minutes; more if required

Passed urine within 24 hours

Yes

Acute prerenal failure; FeNa < 1

Treat the cause

Restrict fluids (400 ml/m² 5% dextrose + losses)

Manage Complications
- Hyperkalemia, fluid overload, hypocalcemia, hypernatremia, metabolic acidosis, hypertension, anemia

Anticipate Problems
- Adjust drugs for renal failure
- Avoid nephrotoxic drugs

No response

Intrinsic renal failure; FeNa > 1

Diuretics
- Mannitol 0.5 gm/kg
- Furosemide 2-4 mg/kg or 2-3 μg/kg/minute drip

Postrenal failure

Treat the cause

RRT/dialysis

Volume overload or CHF

Injection furosemide 2-4 mg/kg IV

No

Abbreviations: NS, normal saline; CHF, congestive heart failure; IV, intravenous; FeNa, fractional excretion of sodium; RRT, renal replacement therapy; BUN, blood urea nitrogen
Fulminant Hepatic Failure Algorithm

Praveen Khilnani, Pankaj Vohra

**Abbreviations**: CVP, central venous pressure; ABP, ambulatory blood pressure; CBC, complete blood count; PT, prothrombin time; FHF, fulminant hepatic failure; GCS, Glasgow coma scale; GI, gastrointestinal
Clinical Approach to a Newborn Infant with Symptomatic Hyperammonemia

Hypercalcemia

Pseudohypercalcemia
- Hemococoncentration
- Elevated prolactin level

Real
- Lethargy +/- confusion or coma
- ECG changes

No

Yes
- Forced saline diuresis (Normal saline +/- Lasix)
- Consider verapamil if arrhythmias

Yes

No response
- Dialysis

- Bisphosphonates preferably pamidronate 30–90 mg IV over 24 hours
- Calcitron 3–6 mg/kg
- Gallium nitrate 200 mg/m² infusion x 5 days
- Plicamycin 25 mcg/kg/d IV x 3–5 days

Abbreviations: CPS, carbamyl phosphate synthetase; HHH syndrome; hyperammonemia-hyperornithinemia-homocitrullinemia; NAG, N-acetylglutamate; OTC, ornithine transcarbamylase
### Treatment of Acute Hyperammonemia in an Infant

1. Provide adequate calories, fluid, and electrolytes intravenously (10% glucose and intravenous lipids 1 gm/kg/24 hr). Add minimal amounts of protein preferably as a mixture of essential amino acids (0.25 gm/kg/24 hr) during the 1st 24 hour of therapy.

2. Give priming doses of the following compounds:
   - To be added to 20 ml/kg of 10% glucose and infused within 1–2 hour
   - Sodium benzoate 250 mg/kg (5.5 gm/nm)
   - Sodium phenylacetate 250 mg/kg (5.5 gm/nm)
   - Arginine hydrochloride 200–600 mg/kg (4.0–12.0 gm/nm) as a 10% solution

3. Continue infusion of sodium benzoate (250–500 mg/kg/24 hr), sodium phenylacetate (250–500 mg/kg/24 hr), and arginine (200–600 mg/kg/24 hr) following the above priming doses. These compounds should be added to the daily intravenous fluid.

4. Initiate peritoneal dialysis or hemodialysis if above treatment fails to produce an appreciable decrease in plasma ammonia.
Importance of Arterial Blood Gas in PICU

Arterial blood gas (ABG) is an important weapon in the armamentarium of the pediatric intensivist as it provides a lot of information in a very short time and helps a lot in fast decision making in the setting of an intensive care unit (ICU). ABG provides information on number of parameters like acid base status, oxygenation, ventilation, electrolytes, hemoglobin, alveolar-arterial oxygenation difference, etc. But in this chapter we restrict ourselves to the most important aspect of ABG, i.e. acid base status, oxygenation and ventilation. Because of this the ABG helps in the day to day monitoring and decision making in ICU. ABG should be done in any sick child admitted to PICU as it provides vital information in most of the diseases requiring ICU admission.

Basics of Arterial Blood Gas

Before going on to the interpretation of ABG, one needs to know some basic principles and pathophysiology of acid base balance and some basic definitions.

- **Principles of Acid-Base Balance**
  - pH is defined as negative logarithm of H+ conc. Normal pH in our body is maintained within a narrow limit of 7.35–7.45
  - Buffer is a substance which on combining with strong acid or base makes it weak acid or base. It is a combination of a strong acid or base and its salt with a weak base or acid. Various buffers are present in our body of which intracellular buffers are Hb, phosphates and extracellular buffers are bicarbonates and serum proteins
  - Henderson-Hasselbach equation
    \[ H_2O + CO_2 = H_2CO_3 = H^+ + HCO_3^- \]
    \[ H^+ = \frac{H_2CO_3}{HCO_3^-} \]
Interpretation of Arterial Blood Gas

\[
\log H^+ = \log \frac{H_2CO_3}{HCO_3^-}
\]

\[- \log H^+ = pH = PK + \frac{\log HCO_3^-}{H_2CO_3}
\]

\[H_2CO_3 \text{ is in equilibrium with dissolved CO}_2 \text{ which is dependent on PCO}_2\]

\[\text{pH} = PK + \frac{\log HCO_3^-}{0.03 \times \text{PCO}_2}\]

Change in ratio of PCO2 and HCO3- conc. and not the absolute value of either is determinant of H+ conc. or pH.

- Compensation is defined as a process of altering the unaffected component in the ratio (HCO3 or PCO2) in an attempt to normalize the overall ratio and attain a normal pH.
- Base excess describes the presence (in mEq/L) in the blood of an excess of base or deficit of fixed acid. Base excess is a positive value and base deficit is a negative value. Quantity of base or acid that must be added to the blood to restore normal pH. BE = -1.2 (24-measured HCO3).

Interpretation of Arterial Blood Gas

- Acidemia is pH < 7.35 and Alkalemia is pH > 7.45. For calculations: normal pH is taken as 7.40
  - Normal PCO2 35 – 45 mmHg. For calculations: normal PCO2 is taken as 40 mm Hg
  - Normal HCO3 21 – 24 mEq/L. For calculations: normal HCO3- is taken as 24 mEq/L.
- pH and CO2 move in opposite directions – respiratory acidosis – pH low, CO2 high, respiratory alkalosis – pH high, CO2 low
- pH and HCO3- move in same directions – metabolic acidosis – pH low, HCO3- low, metabolic alkalosis – pH high, HCO3- high.
- For compensation CO2 and HCO3- move in same directions
  - Primary disorder
  - Respiratory acidosis (CO2 high) Metabolic alkalosis (HCO3- high)
  - Respiratory alkalosis (CO2 low) Metabolic acidosis (HCO3- low)
  - Metabolic alkalosis (HCO3- high) Respiratory acidosis (CO2 high)
  - Metabolic acidosis (HCO3- low) Respiratory alkalosis (CO2 low)
- Compensation – Time frame
  - Respiratory compensation starts immediately and is complete within 4–6 hrs
  - Metabolic compensation starts after 10–12 hrs and may take 3–7 days
  - Body never overcompensates
- Compensation
  - Resp. acute
    - ↑ CO2 by 10 ↑ HCO3 by 1
    - ↓ CO2 by 10 ↓ HCO3 by 2
- Resp. chronic
  - ↑ CO₂ by 10  ↑ HCO₃ by 3–4
  - ↓ CO₂ by 10  ↑ HCO₃ by 3–5

- Metabolic
  - ↓ HCO₃ by 1  ↓ CO₂ by 1–1.5
  - ↑ HCO₃ by 1  ↑ CO₂ by 0.2–1

For every acute rise of PCO₂ by 20 mmHg, pH decreases by 0.1 unit
For every acute fall of PCO₂ by 10 mmHg, pH increases by 0.1 unit

- Oxygenation
  - PaO₂ is dependent on the FiO₂
  - At room air (FiO₂ 21%) PaO₂ is 80–100 mmHg (PaO₂: FiO₂ = 4–5:1)
  - So interpretation of PaO₂ should always be in relation to the FiO₂

- Recognize simple disorders
  - Assess the acid base status by pH to define acidosis or alkalosis
  - Then look for the cause of change in pH, i.e. respiratory or metabolic
  - Then look for compensation
    - Partial: pH remains altered
    - Complete: pH comes to near normal
  - Then look for oxygenation (based on FiO₂)

- Recognize complex disorders
  - This requires reference to the below mentioned nomogram. This nomogram has pH on the x-axis, HCO₃⁻ on the y-axis and CO₂ levels marked on the graph as multiple lines
  - Matching all three at one point will clarify whether the disorder is a simple disorder or a mixed disorder
When to ask for ABG?
- Any seriously ill child
- Renal failure—Acute and chronic
- Respiratory distress—Moderate/Severe
- Chronic persistent respiratory disorder
- Neurological disorders—GBS
- Cardiac disorders—Acute worsening
- Suspected inborn errors of metabolism

Factors in interpretation
- When interpreting an ABG report in a serious patient consider the following:
  - Original disease
  - Complication—e.g. nosocomial pneumonia
  - Associated medical conditions
  - Electrolyte disturbances—(hypophosphatemia shifts $O_2$ dissociation curve to left)
- Therapeutic Interventions
  - Type of IV fluids
  - Ventilator settings
  - Blood transfusions
  - NG aspiration/diarrhea
  - Drugs
  - TPN
  - $NaHCO_3$ administration
  - Compensatory mechanisms
    - Time factor
    - Ability of kidney/lungs

The 10 Commandments
- Confirm that the sample sent is arterial and not venous
- Use only minimal amount of heparin to rinse the syringe
- Ensure there are no air bubbles in the blood samples
- Send the sample in ice and analyze it quickly, and keep the TLC in mind, esp. when there is a delay
- Always take the history of sodium bicarbonate administration even in respiratory cases
- Always consider the effects of furosemide, steroids, salbutamol, blood transfusion and hypokalemia
- Always take $FiO_2$ into consideration when interpreting $PO_2$ values. Also look at the $PCO_2$ values carefully
- Take the history into consideration before instituting therapy for Chronic respiratory failure
In a ventilated child with abnormal PaO$_2$ or PCO$_2$, always remember the acronym “DOPe” in such situations—
- D - Displacement
- O - Obstruction
- P - Pneumothorax
- E - Equipment failure

Practice gentle mechanical ventilation and not try to bring ABG to perfect normal.

**Five Steps for ABG Analysis**

**Step 1**
Look at the pH is the patient acidemic pH < 7.35 or alkalemic pH > 7.45

**Step 2**
Who is responsible for this change in pH (culprit)?
- CO$_2$ will change pH in opposite direction
- HCO$_3$ will change pH in same direction.

**Acidemia**
- With HCO$_3$ < 20 mmol/L = metabolic
- With PCO$_2$ > 45 mmHg = respiratory.

**Alkalemia**
- With HCO$_3$ > 28 mmol/L = metabolic
- With PCO$_2$ < 35 mmHg = respiratory.

**Step 3**
If there is a primary respiratory disturbance, is it acute?
- For every change of PaCO$_2$ of 10 mmHg
  - pH changes by 0.08 (Acute)
  - pH changes by 0.03 (Chronic).

**Step 4**
If the disturbance is metabolic or respiratory? Is the compensation appropriate?

*Metabolic Acidosis*
- Expected PaCO$_2$ = (1.5 × [HCO$_3$]) + 8 ) ± 2
- Or simply expected PaCO$_2$ = last two digits of pH

*Metabolic Alkalosis*
Expected PaCO$_2$ = 6 mmHg for 10 mEq rise in Bicarb.
Interpretation of Arterial Blood Gas

Suspect if
- Actual PaCO₂ is more than expected: additional respiratory acidosis
- Actual PaCO₂ is less than expected: additional respiratory alkalosis.

If there is metabolic acidosis, is there a wide anion gap?

\[ \text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-) = \text{Anion gap} \]

usually <12, if >12, Anion Gap Acidosis:
- Methanol, uremia, diabetic ketoacidosis, paraldehyde, infection (lactic acid), ethylene glycol, salicylate

- Common pediatric causes
  - Lactic acidosis
  - Metabolic disorders
  - Renal failure.

Step 5 Clinical Correlation

Some Important Equations and Principles

Equation 1
\[ \text{CaO}_2 (\text{O}_2 \text{ content}) = \text{O}_2 \text{ bound to Hb} + \text{O}_2 \text{ dissolved in plasma} \]
\[ (\text{ml O}_2 /\text{dl}) = (\text{Hb} \times 1.34 \times \text{SaO}_2 + (0.003 \times \text{PaO}_2) \]

Equation 2
\[ \text{Alveolar PO}_2 (\text{PAO}_2) = \text{FiO}_2 (P_a - 47) - 1.2 (\text{PaCO}_2) \]
\[ \text{Alveolar-arterial PO}_2 \text{ difference (A-a Gradient)} = \text{PAO}_2 - \text{PaO}_2 = P (A-a) O_2 \]

Note: Simple relationship between FiO₂ and PaO₂ is FiO₂ × 5 = PaO₂, this is clinical working formula with acceptable limitation.

Equation 3
\[ \text{PaCO}_2 = \text{VCO}_2 \times 0.863 \]
\[ \text{VCO}_2 = \text{CO}_2 \text{ prod/min} ; \quad \text{VA} = \text{VE} - \text{VD} = f (V_t - V_d) \]
\[ \text{VA} = \text{alveolar ventilation} \quad f = \text{resp. rate.} \quad V_t \text{ and } V_d \text{ = tidal and dead space volume} \]
\[ \text{PaCO}_2 \text{ can rise with} \]
- Inadequate VE (f & or VT)
- Increased VD
- CNS drugs
- Parenchymal lung disease (V-Q imba)
- Respiratory muscle weakness
- Rapid, shallow breathing
- Central hypoventilation.

Note: There is no correlation between paCO₂ and respiratory rate, V, or clinical appearance, as none of them indicate either CO₂ production or effective alveolar ventilation.

Further Reading

Inborn error of metabolism (IEM) should be suspected in any infant with presentation of lethargy, coma, seizures, “sepsis” and associated history of neonatal death in past or mental retardation, or unexplained acidosis hyperammonemia or hypoglycemia.

**Principles of Treatment**

- Correct acidosis:
- Prevent hyponatremia
- Prevent cerebral edema
- Correct ventilatory abnormalities
- Prevent catabolism
- Prevent infection (common cause of mortality)
- Supplement substrates that may help
- Management while awaiting results
- Quickly: Stop all milk feeds
- Tell mother to express and store in case diagnosis is wrong
- Collect while feed effect still present:
  - EDTA blood
  - Lithium heparin blood
  - Frozen urine
  - Filter paper blood and urine
  - Skin, liver and muscle biopsy (feeding not an issue)
- If symptoms are mild
- Stop feeds and give IV D10 with electrolytes as per biochemistry results

- Determine if there is metabolic acidosis
- Is anion gap >16?
- Is there hypoglycemia?
- Is there hyperammonemia (HA)?

Go to algorithm for HA and urea cycle defect (Chapter 32)
- B₆ if seizures
- Thiamine, Biotin, B₁₂, Riboflavin
- 100 × RDA

Metabolic Acidosis with Increased Anion Gap in Young Infant
Acidosis

Abbreviations: AG, anion gap; GIT, gastrointestinal tract; HA, hyperammonemia; RDA, recommended daily allowance; IVA, isovaleric acidemia; PA, propionic acidemia; MMA, methyl malonic acidemia

Metabolic Acidosis

- Increased AG acidosis
- Normal AG acidosis

Bicarbonate Loss:
- GIT: Diarrhea
- Renal: Renal tubular acidosis
- Levels of chloride: Salt poisoning or large volume of I/V 0.9% NS

Toxic Ingestion:
- Salicylates, methanol, ethylene glycol

Over production of endogenous acids

Decreased excretion of fixed acids:
- Renal failure

Lactic acid:
- Shock and mitochondrial dysfunction

Abnormal acids:
- Inborn errors in metabolism e.g., organic acidemias

Ketoacids:
- Diabetic ketoacidosis

Abbreviations: AG, anion gap; GIT, gastrointestinal tract; HA, hyperammonemia; RDA, recommended daily allowance; IVA, isovaleric acidemia; PA, propionic acidemia; MMA, methyl malonic acidemia
Metabolic Acidosis

Causes of Metabolic Acidosis

<table>
<thead>
<tr>
<th>Increased Anion Gap</th>
<th>Causes of Hyperchloremic Metabolic Acidosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Lactic acidosis</td>
<td>• Gastrointestinal bicarbonate loss</td>
</tr>
<tr>
<td>– Tissue hypoxia (shock, hypoxemia, severe anemia)</td>
<td>– Diarrhea</td>
</tr>
<tr>
<td>– Liver failure</td>
<td>– External pancreatic or small bowel drainage</td>
</tr>
<tr>
<td>– Malignancy</td>
<td>– Ureterosigmoidostomy, jejunal loop</td>
</tr>
<tr>
<td>– Intestinal bacterial overgrowth</td>
<td>– Drugs</td>
</tr>
<tr>
<td>– Inborn errors of metabolism</td>
<td>• Calcium chloride (acidifying agent)</td>
</tr>
<tr>
<td>– Medications (nucleoside analogues, metformin)</td>
<td>• Magnesium sulfate (diabetes)</td>
</tr>
<tr>
<td>• Ketoacidosis</td>
<td>• Cholestyramine (bile acid diarrhea)</td>
</tr>
<tr>
<td>– Diabetic ketoacidosis</td>
<td>– Renal tubular acidification defects</td>
</tr>
<tr>
<td>– Starvation ketoacidosis</td>
<td>• Hypokalemia</td>
</tr>
<tr>
<td>– Alcoholic ketoacidosis</td>
<td>– Proximal renal tubular acidosis (RTA) (type 2 RTA)</td>
</tr>
<tr>
<td>• Kidney failure</td>
<td>– Classical distal RTA (type 1 RTA)</td>
</tr>
<tr>
<td>• Poisoning</td>
<td>• Hyperkalemia</td>
</tr>
<tr>
<td>– Ethylene glycol</td>
<td>– Generalized distal nephron dysfunction (type 4 RTA)</td>
</tr>
<tr>
<td>– Methanol</td>
<td>– Mineralocorticoid deficiency</td>
</tr>
<tr>
<td>– Salicylate</td>
<td>– Mineralocorticoid resistance</td>
</tr>
<tr>
<td>– Toluene</td>
<td>– Decreased delivery of Na+ to the distal nephron</td>
</tr>
<tr>
<td>– Paraldehyde</td>
<td>* Liver disease</td>
</tr>
<tr>
<td>• Inborn errors of metabolism</td>
<td>– Normokalemia or hyperkalemia</td>
</tr>
<tr>
<td></td>
<td>* Early renal failure</td>
</tr>
<tr>
<td></td>
<td>• Other</td>
</tr>
<tr>
<td></td>
<td>– Acid loads (ammonium chloride, hyperalimentation with insufficient alkali infusion)</td>
</tr>
<tr>
<td></td>
<td>– Loss of potential bicarbonate: ketosis with ketone excretion</td>
</tr>
<tr>
<td></td>
<td>– Expansion acidosis (rapid saline administration)</td>
</tr>
<tr>
<td></td>
<td>– Posthypocapnic state</td>
</tr>
<tr>
<td></td>
<td>– Glue sniffing</td>
</tr>
</tbody>
</table>

Distinguishing Features of Hyperchloremic Acidosis

<table>
<thead>
<tr>
<th></th>
<th>Proximal RTA (Type 2 RTA)</th>
<th>Classical distal RTA (Type 1 RTA)</th>
<th>Generalized distal defect (Type 4 RTA)</th>
<th>Extra-renal HCO₃⁻ Loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anion gap</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Plasma (K⁺)</td>
<td>Low (with Rx)</td>
<td>Low</td>
<td>High</td>
<td>Low</td>
</tr>
<tr>
<td>Urine anion gap or urine osmolal gap</td>
<td>Low</td>
<td>Low</td>
<td>Very low</td>
<td>High</td>
</tr>
</tbody>
</table>

Contd...
Proximal RTA (Type 2 RTA) | Classical distal RTA (Type 1 RTA) | Generalized distal defect (Type 4 RTA) | Extra-renal HCO₃⁻ Loss
--- | --- | --- | ---
Urine pH | Low | High | Low or high | Low or High
Urine PCO₂ | > 70 | < 40 | < 40 | > 70
FEHCO₃ | > 15% (with Rx) | 5–10% | 10–15% | < 5%
Urine citrate | High | Low | Low | Normal
TTKG | High | High | Low (not high) | Low

**Abbreviations:** RTA, renal tubular acidosis; TTKG, transtubular potassium gradient

Urine net charge or urinary osmolal gap (UNC or UAG) = \[ \text{Na}^+ + \text{K}^+ \] u – \( \text{CL}^- \) u.

TTKG = \( \frac{\text{UK}^+/\text{PK}^+}{\text{U Osm/P Osm}} \)

**Treatment**

1. The most effective therapeutic approach for patients with a metabolic acidosis is repair of the underlying disorder, if possible.
2. The administration of insulin in diabetic ketoacidosis and restoration of adequate perfusion in lactic acidosis eventually result in normalization of the acid-base balance.
3. In other diseases, the use of bicarbonate therapy is indicated because the underlying disorder is irreparable.
4. Children with metabolic acidosis due to RTA or chronic renal failure require long-term base therapy.

**Forms of Alkali Replacement**

<table>
<thead>
<tr>
<th>Shohl Solution</th>
<th>Each 1 ml contains 1 mEq sodium and is equivalent to 1 mEq of bicarbonate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na⁺ citrate 500 mg</td>
<td>Cicitric acid 334 mg/5 ml</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>NaHCO₃ Tablets</th>
<th>3.9 mEq/tablet (325 mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.8 mEq/tablet (650 mg)</td>
<td></td>
</tr>
</tbody>
</table>

| Baking Soda | 60 mEq/tsp |
| K-Lyte | 25 or 50 mEq/tablet |

<table>
<thead>
<tr>
<th>Polycitra (K-shohl solution)</th>
<th>Each ml contains 1 mEq potassium and 1 mEq sodium and is equivalent to 2 mEq bicarbonate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Na⁺ citrate 500 mg</td>
<td>K⁺ citrate 550 mg</td>
</tr>
<tr>
<td>Cicitric acid 334 mg/5 ml</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Polycitra Crystals</th>
<th>Each packet contains 30 mEq potassium and is equivalent to 30 mEq bicarbonate</th>
</tr>
</thead>
<tbody>
<tr>
<td>K⁺ citrate 3300 mg</td>
<td>Citric acid 1002 mg per packet</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Urocit-K Tablets</th>
<th>5 or 10 mEq per tablet</th>
</tr>
</thead>
</table>
Acidosis

Approach to Child with Acidosis

Respiratory acidosis
- CNS depression: Encephalitis, head trauma, brain tumor, stroke, hypoxic brain damage, drugs
- Disorders of spinal cord, peripheral nerves or neuromuscular junction: Diaphragmatic paralysis, GB syndrome, Polio, Botulism, Myasthenia, etc.
- Respiratory muscle weakness: Muscular dystrophy, hypokalemia, hypophosphatemia, medications like Sch.
- Pulmonary diseases: Pneumonia, asthma, Bronchiolitis, etc.
- Upper airway diseases: Aspiration, Slayngospasm, Angioedema

Treatment (Resp Acidosis):
- Treat underlying etiology
- Supplement oxygen
- Patients hypercapnias: mechanical ventilation

Metabolic acidosis
- Normal anion gap or hyperchloremic metabolic acidosis
  - Diarrhea (loss of HCO₃ in stool)
  - Renal tubular acidosis
  - Fistula between ureter and gut
  - Infusion of isotonic saline
  - Early renal insufficiency
  - Suspect aminoacidopathies
- ↑Anion gap
  - Lactate increase

Yes
- Lactic acidosis
- Tissue hypoxia
- Liver failure, malignancy

No
- BS ↑ and urinary ketones

Yes
- DKA
- ↑BUN/Creatinine

Yes Uremia

Treatment (Metabolic acidosis)
- Most effective - Repair of underlying etiology:
  - For DKA - Insulin
  - For lactic acidosis - Restoration of adequate perfusion
  - For IEM - Disease specific therapies
- Base therapy - use with caution
  - NaHCO₃ at dose of 1 mEq/kg can be used in emergency
- Hemodialysis
  - For patients with renal failure with significant uremia or hyperkalemia
  - For methanol or ethylene glycol ingestion
  - If odor of alcohol - Suspect ethylene glycol
  - Methanol
  - If auditory s/s salicylate
  - Organic acidermias (See inborn errors of metabolism flow)

Abbreviations: CNS, central nervous system; BUN, blood urea nitrogen; DKA, diabetic ketoacidosis; IEM, inborn error of metabolism; GB syndrome, Guillain-Barre syndrome
Alkalosis

Praveen Khilnani

Respiratory alkalosis
- Causes
  1. Hyperventilation (in presence of lung disease): Pneumonia, pulmonary edema, CHF, etc.
  2. Central stimulation (hyperventilation in absence of lung disease)
     - Lesions such as infarcts or tumors near the central respiratory center in midbrain
     - SAH, meningitis, stroke, brain tumor
  3. Hyperventilation may be secondary to underlying disease that cause pain and stress on anxiety

Treatment
- Treat the underlying cause
- For psychogenic hyperventilation, reassurance and benzodiazepines may help

Metabolic alkalosis
- Chloride responsive
  - Urinary chloride < 15 mEq/L
  - Gastric losses (emesis, NG suction)
  - Diuretics (loop and thiazide)
  - Volume depletion
  - Posthypercapnia

Treatment—
- Chloride responsive
  - For mild metabolic alkalosis (HCO₃ < 32), intervention unnecessary
  - Treatment needed for moderate and severe metabolic alkalosis
  - Most effective is to look for and correct the underlying etiology
  - Decrease NG suction/adequately replace gastric losses of sodium and potassium
  - Diuretics:
    - Stop or ↓ dose
    - K⁺ supplementation
    - or add K⁺ sparing
  - Diuretic
    - Acetazolamide
    - Administration of NaCl or KCl

- Chloride resistant
  - High BP
    - Adrenal adenoma or hyperplasia
    - Renovascular disease
    - Renin secreting tumor
    - Liddle syndrome
    - Normal BP
    - Gitelman syndrome
    - Bartter syndrome
    - Base administration

Treatment—
- Chloride resistant
  - Focuses on eliminating excess aldosterone effect:
    - Adrenal adenoma can be resected
    - Treat renovascular disease
    - ↓ Licorice intake
  - Liddle syndrome: Use triamterene or amiloride
  - Bartter syndrome/Gitelman syndrome:
    - oral K⁺ supplementation
    - K⁺ sparing diuretic

Abbreviations: CHS, congestive heart failure; SAH, subarachnoid hemorrhage; NG, nasogastric suction
Hyponatremia

Praveen Khilnani, Nitesh Singhal

Abbreviations: ECF, extracellular fluid; SIADH, syndrome of inappropriate secretion of antidiuretic hormone; CNS, central nervous system; GIT, gastrointestinal tract; RTA, renal tubular acidosis; ATN, acute tubular necrosis
Hypernatremia

Causes of Hypernatremia

<table>
<thead>
<tr>
<th>Low total body sodium</th>
<th>Renal</th>
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</thead>
<tbody>
<tr>
<td>Extrarenal loses</td>
<td>Diabetes insipidus</td>
</tr>
<tr>
<td>Vomiting/diarrhea</td>
<td>• Central</td>
</tr>
<tr>
<td>Sweating</td>
<td>• Nephrogenic</td>
</tr>
<tr>
<td>70% sorbitol</td>
<td>• Hypodipsia (reset osmostat)</td>
</tr>
<tr>
<td>Renal loses</td>
<td></td>
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<tr>
<td>Osmotic diuresis</td>
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<tr>
<td>Mannitol, glucose, urea</td>
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<tr>
<td>Inadequate intake</td>
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<tr>
<td>Insufficient lactation</td>
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<table>
<thead>
<tr>
<th>Normal total body sodium</th>
<th>Inadequate intake</th>
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<tr>
<td>Extrarenal loses</td>
<td>Burns</td>
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<td>Respiratory insensible losses</td>
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<td>Dermal insensible losses</td>
<td>Phototherapy</td>
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<tr>
<td>Fever</td>
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<tr>
<td>Radiant warmers</td>
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</tr>
</tbody>
</table>

Diagnostic Approach to Hypernatremia

- **Known underlying disease?**
  - Yes
    - Diabetes insipidus
      - Central
      - Nephrogenic
  - No
    - History of excess sodium intake (improperly prepared formula)?
      - Yes
        - Salt poisoning
      - No
        - History of fluid loss?
          - Yes
            - Dry mucous membranes?
              - Yes
                - Decreased skin turgor?
                  - Yes
                    - Hypertonic dehydration
                  - No
                    - Hypaldosteronism
                      - Osmotic diuretics
                      - Obstructive uropathy
                      - Essential Hypernatremia
Hypernatremia

Treatment

- Hypernatremia should not be corrected rapidly.
- Goal to decrease serum sodium by $< 12 \text{ mEq/L}$ every 24 hours, a rate of 0.5 mEq/L/hr
- As the child is dehydrated, restore the intravascular volume with isotonic saline (0.9% NaCl).
- $\text{Na}^+$ concentration of the fluid, rate of fluid administration and the presence of continued water loses determine the rate of decrease of the sodium concentration.
- Formula for calculating water deficit

$$\text{Water deficit} = \text{Body weight (kg)} \times 0.6 \left(1 - \frac{145}{\text{Current Na}^+}\right)$$

Equivalent to between 3 ml and 4 ml of water per kilogram for each 1 mEq that the current Na$^+$ is greater than 145.
- Fluid sodium concentration between one-fourth normal saline (NS) and half NS.
- Fluid rate that is only 20–50% greater than maintenance.
- Excessive water and sodium loses may also need to be replaced.
- In general, solutions containing glucose should be avoided.
- Plasma electrolytes should be monitored every 2 hours until the patient is neurologically stable.

Hypernatremia Algorithm

```
Serum sodium > 150 mmol/L
Assess extracellular fluid volume

Normal/overhydration
- Excessive sodium containing IV fluids
- Sodium bicarbonate infusion
- Improper fluids for dialysis
- Renal failure
- Inappropriate dilution of formula
  Treat the cause

Decreased
- Gastroenteritis
- Feeding of concentrated formula
- Diabetes mellitus
- Diabetes insipidus
- Child abuse (lack of feeding)
  Serum sodium > 200 mmol/L
  Calculate water deficit (see text)

Yes
No
```

Contd...
Further Reading

Hyperkalemia

- Pseudohyperkalemia
- Hemolysis during sampling
- Polycythemia
- Capillary sample

True elevated serum $K^+ > 5$ mmol/L

Stop all source of additional $K^+$

$K^+ > 6$ mEq/L or ECG changes

Emergent $K^+$ reduction

ECG abnormal

No

Shift $K^+$ intracellularly

- Insulin (0.1–0.2 U/kg) and glucose (0.5–1 g/kg)
- Nebulize with salbutamol 5–10 mg (avoid in cardiac disease)
- 7.5% NaHCO$_3$ 1–2 mEq/kg IV (reserved for severe acidosis)

Yes

IV calcium gluconate 0.5–1 ml/kg (1ml = 9mg)

Caution: Digitalis toxicity; give slowly IV over 20–30 min or use IV MgSO$_4$

Is $K^+$ < 6 mEq/L

No

Repeat as above; consider hemodialysis

Yes

Sodium polystyrene sulfonate (1 g/kg oral or rectal route) +/- IV furosemide (1–2 mg/kg)

Further evaluation and long-term therapy if needed
Hypokalemia

**Abbreviations:** RTA, renal tubular acidosis; NS, normal saline; ABG, arterial blood gas
Hypocalcemia is frequently associated with critical illness in children. It is seen in nearly 15–50% pediatric intensive care unit (PICU) admissions. Hypocalcemia is the result when the influx of calcium in the extracellular fluid does not keep pace with calcium efflux into bone, excretion into the urine or less commonly as deposits into the soft tissue. The level of intravascular calcium is regulated and protected by the parathyroid and vitamin D systems and their effects on bone resorption, renal excretion, and gastrointestinal absorption of calcium. Acute hypocalcemia can cause life-threatening events especially through disordered neuromuscular and cardiovascular physiology.

**Definition**

The total serum calcium in children ranges from 8.8 mg/dl in infants to 9.4 mg/dl in pre-adolescents and dropping back to 9 mg/dl in adolescents.

The most important fraction ionized calcium does not exactly follow total calcium with lower limits of normal of 4.9 mg/dl until 6 years of age and then it drops slightly to older 4.5 mg/dl children and 5.6 mg/dl in young adults. Patients known to have changes in albumin, pH, or one of the complexing anions, ionized calcium should be measured directly.

Hypocalcemia = Less than 8.5 mg/dl

Adjusted calcium = Measured total calcium (mg/dl) + 0.8 [4.0 – serum albumin (g/dl)]

Prolonged QT interval = QT Interval / √RR interval

**Clinical Manifestations of Hypocalcemia**

**Cardiovascular**

- Prolonged QT interval on ECG
- Heart failure.
Neuromuscular

- Paresthesias, perioral tingling
- Muscle cramps, tetany
- Laryngospasm
- Trousseau’s sign
- Chvostek’s sign
- Seizures (all types)
- Irritability, abnormal mental function.

Other

- Cataracts, papilledema, coarse skin, brittle nail.

Causes of Hypocalcemia

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<th>Causes</th>
<th>Manifestations</th>
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<td>Pancreatitis</td>
<td>Renal failure</td>
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<td>Hyperphosphatemia</td>
<td>Exogenous</td>
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<td>Hyperparathyroidism</td>
<td>Phosphorus containing enemas</td>
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<td>Congenital hypoparathyroidian</td>
<td>High phosphorus formulas</td>
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<tr>
<td>DiGeorge and related syndromes</td>
<td>Hypophosphatemia</td>
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<tr>
<td>Maternal hyperparathyroidian</td>
<td>Vitamin D deficiency</td>
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<tr>
<td>Calcium receptor activating mutations</td>
<td>Lack of sun and dietary</td>
</tr>
<tr>
<td>Acquired hypoparathyroidian</td>
<td>Malabsorption</td>
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<tr>
<td>Autoimmune</td>
<td>Renal failure</td>
</tr>
<tr>
<td>Surgical removal or damage</td>
<td>Increased metabolism</td>
</tr>
<tr>
<td>Hypomagnesemia</td>
<td>Vitamin D dependent rickets (VDDR) type I</td>
</tr>
<tr>
<td>PTH resistance</td>
<td>Resistance to VDDR type II</td>
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<tr>
<td>Pseudohypoparathyroidian</td>
<td>Deposition of calcium and phosphorus into tissues</td>
</tr>
<tr>
<td>Hypomagnesemia</td>
<td>Hungry bone syndrome</td>
</tr>
<tr>
<td>Phosphorus loads</td>
<td></td>
</tr>
</tbody>
</table>

Drugs

- Aminoglycosides, cisplatin, beta adrenergic drugs, glucagon, calcitonin, ethylenediamine tetra acetate (EDTA)

Approach to Hypocalcemia

- Suspect hypocalcemia
- Symptoms include cramping, tetany, tingling

Order

- Calcium, serum or plasma
- Phosphorus, inorganic plasma or serum
- Albumin, serum by spectrophotometry
- Magnesium, plasma or serum
- Creatinine, serum or plasma

Contd...
Hypocalcemia

Low corrected calcium
Corrected calcium = Measured total calcium + 0.8 (4 – serum albumin)

Order
Parathyroid hormone, intact

Low

- Creatinine normal
- Phosphorus normal or high

- Magnesium low
  Hypoparathyroidism
  Magnesium deficiency

Normal

- Creatinine normal
- Phosphorus low or normal
- Magnesium normal

- Albumin low
  Hypoalbuminemia (Pseudohypocalcemia)

High

- Creatinine high
- Phosphorus normal or high
- Magnesium normal

- Vitamin D, 25-hydroxy
  Order
  Pseudohypoparathyroidism

Low

- Renal disease

Treatment

Asymptomatic hypocalcemia

- 10% calcium gluconate 1–2 ml/kg/dose q 4–6 hourly bolus infusion diluted to 2% (mix 10 ml of 10% calcium gluconate in 40 ml NS)

Symptomatic hypocalcemia

- Continuous IV infusion 5–8 ml/kg/day of 10% calcium gluconate diluted to 2%

Emergency

- 10% calcium gluconate over 10–15 min at a dose of 0.5–1 ml/kg at a rate not more than 1 ml/min using constant cardiac monitoring

- Or

- 10% calcium gluconate 1–2 ml/kg/dose q 4–6 hourly bolus infusion diluted to 2% mix 10 ml of 10% calcium gluconate in 40 ml NS

Monitor calcium levels 4–6 hourly

Hypomagnesemic hypocalcemia refractory to this treatment and should be treated with 0.1–0.2 ml/kg of 50% magnesium sulfate (50–100 mg/kg) IV over a 10-minute period, a dose that can be repeated every 12–24 hours.
In severe, symptomatic hypocalcemia, emergency treatment with intravenous calcium is indicated. Ten percent of calcium gluconate over 10–15 min at a dose of 0.5–1 ml/kg at a rate not more than 1 mL/min using constant cardiac monitoring is often sufficient to stop the acute manifestations including seizure.

Then 10% calcium gluconate 1–2 ml/kg/dose q 4–6 hourly bolus infusion diluted to 2% (mix 10 ml of 10% calcium gluconate in 40 ml NS)

Or

Continuous IV infusion 5–8 ml/kg/day of 10% calcium gluconate diluted to 2% monitor calcium levels 6 hourly and infusion can be stopped once normalized.

Hypomagnesemic hypocalcemia will be refractory to this treatment and should be treated with 0.1–0.2 ml/kg of 50% magnesium sulfate (50–100 mg/kg) IV over a 10-minute period, a dose that can be repeated every 12–24 hours.

Calcium infusions can be given for persistent hypocalcemia in children by mixing 10 mL calcium gluconate ampules in 1 L D5W and given at a rate of 50–75 mg/kg/day. It is imperative that it is only given in a well-functioning IV to avoid chemical.

Burns with calcium extravasation into the tissues and with continuous cardiac monitoring to avoid bradycardia.

Calcium can be given on the chronic basis orally at a dose 50 mg/kg/day of elemental calcium daily. The form of oral calcium generally is not clinically relevant except for states achlorhydria in the stomach where calcium carbonate does not dissolve and calcium citrate in preferable.

Further Reading

Hypercalcemia

Pseudohypercalcemia
- Hemococoncentration
- Elevated prolactin level

Real
- Lethargy +/- confusion or coma
- ECG changes

No

Yes
- Forced saline diuresis (Normal saline +/- Lasix)

Yes

No response

Dialysis

Bisphosphonates preferably pamidronate 30–90 mg IV over 24 hours
- Calcitonin 3–6 mg/kg
- Gallium nitrate 200 mg/m² infusion x 5 days
- Plicamycin 25 mcg/kg/d IV x 3–5 days

Consider verapamil if arrhythmias
Critically-ill children are reported to frequently have low ionized magnesium despite normal total magnesium levels. Nearly 44% of pediatric intensive care unit (PICU) admissions are reported to have hypomagnesemia. Ionized hypomagnesemia may be associated with more complicated postoperative conditions that occur after surgical correction of congenital heart disease. Hypomagnesemia within 5 days of a cardiac operation in more than 80% of pediatric patients have been reported.

Magnesium depletion results in hypocalcemia. The mechanisms include the suppression of parathyroid hormone (PTH) secretion and bone resistance to PTH. Hypokalemia has also been reported to occur. Magnesium deficiency impairs the Na⁺/K⁻ pump allowing potassium loss from the intracellular fluid (ICF), which in turn, excreted in the urine. Several studies have shown an inability to correct potassium until magnesium stores have been repleted.

### Causes of Hypomagnesemia

- **Decreased intake**: Low Mg**, TPN, IV fluids
- **Increased losses**
  - **Gastrointestinal**
    - Malabsorption
    - Familial primary hypomagnesemia
    - Small bowel disease
    - Regional arteritis, ulcerative colitis, massive bowel resection
    - Pancreatic insufficiency, pancreatitis
    - Cystic fibrosis
  - **Renal**
    - Congenital renal magnesium wasting
    - Diffuse tubular disorders
    - Hypophosphatemia
    - Post-renal transplantation
- **Drugs**: Aminoglycosides, cisplatin, amphotericin B, diuretics, cyclosporine, tacrolimus, pentamidine, foscarnet, GM-CSF
- **Hypercalciuria**
- **Diabetic ketoacidosis**
- **Hyperaldosteronism**
- **Inappropriate anti-diuretic hormone (ADH) secretion**
Signs and Symptoms

In addition to biochemical derangements associated with hypomagnesemia, a wide spectrum of other clinical disorders has been attributed to its depletion.

Common Symptoms

- Increased sensitivity to digoxin
- Coronary spasm
- Hypertension
- Neuromuscular derangements.

Uncommon Symptoms

- Ventricular premature beats
- Ventricular tachycardia
- Torsade de pointes
- Ventricular fibrillation.

Personality changes, including apathetic behavior and depression, have also been associated in older children. Neuromuscular changes may include tremors, fasciculations, spontaneous carpopedal spasm, muscle cramps, paresthesias, seizures and coma.

Treatment

Important principles in the treatment of hypomagnesemia are as follows:

- Severe hypomagnesemia may be life-threatening with hypocalcemia, seizures, and tetany and the serum must be brought up to greater than 1 mg/dl with intravenous replacement over a 5–10 minutes period.
- Secondly, the total body magnesium deficit may not be reflected by the serum magnesium level, especially after treatment and may need prolonged therapy for complete replacement.
- As serum magnesium rises with intravenous therapy, there is significant ongoing renal loss that requires additional therapy. In contrast, if there is renal insufficiency present, magnesium replacement must proceed with caution. Finally, simultaneous deficits of potassium may be present and should be replaced as well. During intravenous therapy, monitoring of the deep tendon reflexes may allow detection of hypermagnesemia with levels greater than 2.5 mg/dl.
- Patients undergoing cardiac surgery are at immediate risk of hypomagnesemic malignant ventricular arrhythmias or seizures can be given magnesium sulfate intravenously with careful monitoring.
- 0.12 ml/kg per dose of 50% magnesium sulfate (0.5 mEq/kg or 60 mg/kg) IV over 1–4 hours, can repeat every 12 hours. Doses may be repeated until values come to normal.
Complications of Parenteral Magnesium Therapy

- Neuromuscular depression
- Respiratory depression
- Malignant arrhythmias
- Flushing and hypotension.

Hypotension and malignant arrhythmias have been reported more frequently in young children who require close monitoring if intravenous (IV) replacement is used. Other routes of therapy include intramuscular magnesium sulfate, injections of which are painful, and oral therapy with magnesium oxide or citrate. In situations known to be associated with the development of hypomagnesemia, it seems particularly important to attempt to avoid deficiency by adequate magnesium intake before development of life-threatening symptoms.

Further Reading

Bleeding Child in the PICU

S Senthil Kumar, KG Ravikumar, B Ramachandran

Medical History
Detailed history regarding the sites of bleeding, the severity and duration of bleeding, and the age at onset should be obtained.
- Was the bleed spontaneous or did it occur after trauma?
- Does the symptom correlate with the degree of injury or trauma?
- Was there a previous personal or family history of similar problems?
- When a child has had previous surgery or dental extractions without bleeding complications, it is unlikely there is an underlying congenital hemorrhagic disorder.

Family History
Questioning must include inquiries about previous surgical procedures, dental extractions, and transfusions of all family members, as well as the menstrual and obstetric histories of female relatives.

Types of Bleeding Manifestations
Mucosal membrane bleeding (gingival hemorrhage, epistaxis, menorrhagia), petechiae, and bruising are more typical of quantitative or qualitative platelet disorders and von Willebrand disease (vWD).
Conversely, spontaneous, deep muscle, and joint bleeding are seen more commonly with bleeding due to coagulation-factor deficiencies such as in hemophilia.

*Healthy children*: Congenital bleeding disorders, idiopathic thrombocytopenic purpura (ITP), vitamin K deficiency.

*Sick children*: Disseminated intravascular coagulation (DIC), liver disease, infection, hemolytic uremic syndrome (HUS).

*Malabsorption and other gastrointestinal disease*: Impaired vitamin K absorption.

*Renal disease*: Abnormalities in platelet function.

**Physical Examination**

Bruises in any area that appear excessively large for the degree of trauma or those with underlying palpable hematomas may be seen in patients with significant bleeding disorders.

- Petechiae, small superficial ecchymosis or mucosal bleeds: platelet related
- Large bruises: deficiency of clotting factors, DIC, liver disease or vitamin K deficiency.

**Investigations**

**Complete Blood Count and Peripheral Blood Smear**

Platelet: Number, size and type of platelet count.

Anemia: Microcytic anemia—a history of prolonged blood loss

Normocytic anemia—recent hemorrhage with significant amounts of blood loss

Anemia, thrombocytopenia, and leukocyte abnormalities—bone marrow failure syndromes

Fragmented RBC: DIC, HUS

**Coagulation Studies**

- Prothrombin time (PT)—test of the extrinsic clotting system
- Activated partial thromboplastin time (aPTT)—a test of the intrinsic clotting system
- Specific clotting factor assays
- Fibrinogen measurement
- FDP/d-dimer
- Thrombin time.
### Bleeding Child in the PICU

<table>
<thead>
<tr>
<th>Test results</th>
<th>Differential diagnosis</th>
<th>Possible follow-up lab studies</th>
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<td>PT normal</td>
<td>von Willebrand disease</td>
<td>PFA-100</td>
</tr>
<tr>
<td>aPTT normal</td>
<td>Platelet function disorder</td>
<td>von Willebrand studies</td>
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<tr>
<td>Platelet count normal</td>
<td>Factor XIII deficiency</td>
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<td>Fibrinolytic defect</td>
<td>Urea clot lysis test</td>
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<td>Euglobulin clot lysis</td>
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<td>Alpha-2-antiplasmin, PAI-1, TPA</td>
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<tr>
<td>PT normal</td>
<td>PTT inhibitor</td>
<td>PTT mixing study</td>
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<tr>
<td>aPTT prolonged</td>
<td>von Willebrand disease</td>
<td>von Willebrand disease</td>
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<tr>
<td>Platelet count normal</td>
<td>Hemophilia A or B</td>
<td>Factor assays (VIII, IX, XI)</td>
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<td>Factor XI deficiency</td>
<td>Thrombin time/reptilase time</td>
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<td>PT prolonged</td>
<td>PT Inhibitor</td>
<td>PT mixing study</td>
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<tr>
<td>aPTT normal</td>
<td>Vitamin K deficiency</td>
<td>Factor assays (II, VII, IX, X)</td>
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<td>Factor VII deficiency</td>
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<td>Factor deficiency</td>
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<td>(II, V, X, or fibrinogen)</td>
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<td>Dysfibrinogenemia</td>
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<td>Tests for <em>Helicobacter pylori</em></td>
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<td>von Willebrand factor multimeric analysis</td>
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<td>Bone marrow aspirate</td>
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<td>Marrow chromosomal analysis</td>
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</tbody>
</table>

**Abbreviations:** ITP, idiopathic thrombocytopenic purpura; DIC, disseminated intravascular coagulation; HUS, hemolytic uremic syndrome; PT, prothrombin time; aPTT, activated partial thromboplastin time; TPA, tissue plasminogen activator
### Platelet Disorders in PICU

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<th>Qualitative</th>
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<td>• SLE</td>
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<td>• Acquired immunodeficiency syndrome</td>
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<td>• Heparin</td>
<td>• Usual dose is 10–15 ml/kg.</td>
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<td>• Local anesthetics</td>
<td>• Repeat doses as needed.</td>
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<td>• Phenothiazines</td>
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<td>• Nitrates</td>
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</table>

**Abbreviations:** ITP, idiopathic thrombocytopenic purpura; DIC, disseminated intravascular coagulation; HUS, hemolytic uremic syndrome; NSAID, nonsteroidal anti-inflammatory drug; SLE, systemic lupus erythematus; TTP, thrombotic thrombocytopenic purpura; CMV, cytomegalovirus; EBV, Epstein Barr virus; HSV, herpes simplex virus
Guidelines for Pediatric Fresh Frozen Plasma Transfusions

- Severe clotting factor deficiency and bleeding
- Severe clotting factor deficiency and planned invasive procedure
- Emergency reversal of warfarin effects
- Dilutional coagulopathy and bleeding
- Anticoagulant protein (Antithrombin III, protein C and protein S) replacement
- Plasma exchange replacement fluid for thrombocytopenic purpura.

Platelets

Usual dose is 10 ml/kg, which increases platelet counts by approximately 50,000/mm³.

Guidelines for Pediatric Platelet Transfusions

Children and adolescents

- PLTs < 50000/dl and bleeding
- PLTs < 50000/dl and an invasive procedure
- PLTs < 20000/dl and marrow failure with hemorrhagic risk factors
- PLTs < 10000/dl and marrow failure without risk factors
- PLTs any count, but with PLT dysfunction plus bleeding or an invasive procedure.

Infants within the first four months of life

- PLTs < 1 lakh/dl and bleeding
- PLTs < 50000/dl and an invasive procedure
- PLTs < 20000/dl and clinically stable
- PLTs < 1 lakh/dl and clinically unstable
- PLTs any count, but with PLT dysfunction plus bleeding or an invasive procedure.

Clotting Factor Concentrates

Factor VIII and factor IX deficiency.

Recombinant Factor VIIa Therapy

Recommendations for the Use of Recombinant Factor VIIa Therapy

Food and drug administration (FDA) indications

- Treatment of bleeding episodes in patients with hemophilia A or B and inhibitors to factor VIII or factor IX
- Prevention of bleeding in surgical interventions or invasive procedures in patients with hemophilia A or B and inhibitors to factor VIII or factor IX
- Treatment of bleeding episodes in patients with congenital factor VII deficiency
- Prevention of bleeding in surgical interventions or invasive procedures in patients with congenital factor VII deficiency.
Though not an FDA-approved indication, recombinant factor VIIa (rFVIIa) has shown potential as therapy for life-threatening hemorrhage secondary to acquired coagulopathy after major surgery or trauma. rFVIIa is effective in improving clotting ability in patients with impaired liver function, thrombocytopenia, and functional platelet defects.

**Off-Label Use**

*Closed-Space Bleeding*
- Nontraumatic intracranial bleeding
- Isolated traumatic head injury
- Retroperitoneal bleeding.

*Rescue Therapy for Surgical Patients*
- Cardiac surgery
- Hepatic resection or liver transplantation
- Nontraumatic high blood loss in orthopedic surgery.

*Postpartum Period and After Hysterectomy*
- Severe multiple trauma
- Hepatic failure with gastrointestinal (GI) bleeding or pending invasive procedure
- Thrombocytopenia with severe bleeding refractory to conventional treatment.

For nonemergency anticoagulant reversal, a dose of 20–40 μg/kg is recommended. For all other indications, 41–90 μg/kg is recommended.

**Treatment of Specific Disorders**
- DIC
- Liver disorders.

**Further Reading**

Abbreviations: CBC, complete blood count; PT, prothrombin time; APTT, activated partial thromboplastin time; ITP, idiopathic thrombocytopenic purpura; TTP, thrombotic thrombocytopenic purpura; HELLP, hemolysis, elevated liver enzymes, low platelet count; IVIG, intravenous immunoglobulin; FFP, fresh frozen plasma; GP, glycoprotein
Approach to a Child with Poisoning

Praveen Khilnani

History
- Take medication history of child and family members
- Find out route of poisoning and estimated time lapsed from exposure

Physical examination
- Detailed general and systemic examination
- Examination of skin and clothes
- Breath odor: garlicky in organophosphates, fruity in nail polish remover, bitter almonds in cyanide poisoning

Monitoring
- Admit in PICU
- Repeated physical examination, vitals monitoring
- Repeated neurological monitoring
- Keep mechanical ventilator stand by

Radiography and laboratory assessment
- X-ray skull, spine and CT chest if trauma suspected
- X-ray chest in cases of respiratory distress, cyanosis or suspected aspiration
- Gastric aspirate and urine for toxicological screen

Management

Stabilization
- Monitor airway, breathing and circulation
- Close cardiological monitoring
- Intropes for hypotension
- Antiepileptics for seizure control

Decontamination
- Emesis: Syrup of ipecac used. Most effective when used soon after ingestion. Contraindicated in hydrocarbon, caustic ingestion, coagulopathy, GI bleeding or sharp foreign body ingestion
- Gastric lavage: Use warm normal saline or tap water. Dose is 15 ml/kg. Contraindicated in caustic ingestion, coagulopathy or sharp foreign body ingestion
- Activated charcoal: Use 1 gm/kg diluted 1:4 in well mixed slurry. Not effective for caustics, hydrocarbons, iron, methanol, ethanol, ethylene glycol. Contraindicated in gastric ileus/obstruction
- Catharsis: Used as an adjunct to activated charcoal
- Whole bowel irrigation: Polyethylene glycol is used. Dose is 25 ml/kg/ hour. Can be used in iron or heavy metal poisoning. Contraindicated in gastric ileus/obstruction

Specific antidote therapy
- Use specific antidote when poison is identified

Abbreviations: PICU, pediatric intensive care unit; GI, gastrointestinal
Paracetamol is the most widely used analgesic and antipyretic. The safety and efficacy of paracetamol in children is well established. In general, the risk of developing toxic reactions to paracetamol appears to be lower in children than in adults.

**Dose and Pattern of Paracetamol Ingestion**

The acute toxic dose of paracetamol is 200 mg/kg in children younger than 12 years. A single ingestion of more than 7.5 gram is considered a minimum toxic dose in adolescents and adults.

An acute ingestion is defined as any number of ingestions that occur within a period of up to 8 hours.

In recent years, the phenomenon of “chronic” paracetamol toxicity has been described. Repeated supratherapeutic ingestion (RSTI) involves any pattern of multiple ingestions over a period of greater than 24 hours that results in a total dosage of more than 4 g per day.

**Pathophysiology of Paracetamol Toxicity**

The toxicity of paracetamol is related to the production of the reactive intermediate N-acetyl-p-benzoquinoneimine (NAPQI) by the hepatic cytochrome P450 system. When the production of NAPQI exceeds the capacity to detoxify it, as can occur in overdose, the excess NAPQI binds to cellular components to cause the death of hepatocytes.

When therapeutic doses are taken, only 4% of the dose is metabolized to NAPQI, which is immediately conjugated with glutathione to form a harmless mercapturic acid conjugate. When hepatic stores of glutathione are depleted to less than 70% of normal, NAPQI metabolite can combine with hepatic macromolecules to produce hepatic injury.

N-acetylcysteine (NAC) serves as a precursor for glutathione synthesis, thus replenishing glutathione stores and preventing the reaction of NAPQI with hepatocytes.
It is well established that the time between ingestion of paracetamol and administration of acetylcysteine affects the outcome of paracetamol poisoning. While the precise threshold is unknown, a delay of more than 8–10 hours results in higher serum aminotransferase levels.

**Clinical and Laboratory Manifestations**

Paracetamol toxicity should be considered in any child who has received paracetamol and has signs of acute hepatic dysfunction even if paracetamol levels are not in the toxic range. If the levels are in the toxic range after long-term treatment with paracetamol, it is an ominous finding associated with a high-risk of mortality.

Paracetamol intoxication typically includes four phases.

**Phase I (First 24 Hours)**
- Anorexia
- Nausea
- Vomiting
- Malaise
- Diaphoresis.

**Phase II (24–48 Hours)**
- First-phase signs resolve
- Right upper quadrant pain or tenderness
- Liver enlargement
- Oliguria in some patients
- Elevation of bilirubin and hepatic enzymes
- Prothrombin time becomes prolonged.

**Phase III (72–96 Hours)**
- Anorexia, nausea, vomiting and malaise reappear.
- Signs of hepatic failure—jaundice, hypoglycemia, coagulopathy and encephalopathy
- Renal failure and cardiomyopathy may also develop. In severe toxicity, markedly elevated alanine transferase (ALT) and aspartate transferase (AST) (> 10,000 IU/L), elevated total bilirubin level of more than 4 mg/dl (primarily indirect) and hyperammonemia and acidosis are reported.

**Phase IV (4 Days to 2 Weeks)**
Either recovery or progression to death from complete liver failure. Rarely, it may present as central nervous system depression, shock, hypothermia and metabolic acidosis. Because delays in treatment with NAC are associated with worse outcomes, early treatment is indicated when paracetamol hepatotoxicity is considered likely.
Management

Detoxification of Paracetamol Metabolite—NAPQI

N-acetylcysteine should be initiated as soon as possible after ingestion, but may have value even if started 24–36 hours after the ingestion in severe cases. Hence, even if the enzymes are abnormal after 16 hours of NAC infusion, it may be continued for a total of 72 hours.

*Management of Children (< 12 Years) Who Present within 8 Hours of Ingestion

Paracetamol poisoning with children’s liquid preparations is rarely serious. Confusion often occurs with drops being given at syrup dosing.

Gastrointestinal Decontamination

There is little evidence that undertaking gastric lavage will be of benefit in a child in whom paracetamol is known to have been the only substance ingested. Although the benefit has not been demonstrated in paracetamol poisoning, administration of activated charcoal may be considered if:

- More paracetamol than 150 mg/kg body weight is thought to have been ingested
- It can be given without difficulty and within 1 hour of the overdose.

Activated charcoal (1 g/kg) mixed with carbonated (soda) water is administered as soon as possible. The efficacy of activated charcoal is decreased when it was administered more than 1 hour after ingestion.

Gastrointestinal decontamination could be particularly important if NAC cannot be administered within 8 hours of ingestion.

Children at enhanced risk of developing severe liver damage include:

- Underweight children with “failure to thrive” whatever the cause
- Those with anorexia nervosa
- Recent fasting
- Receiving enzyme-inducing drugs (e.g. carbamazepine, phenytoin, phenobarbitone, primidone and rifampicin).

The toxic dose for this category is 75 mg/kg

- Plasma paracetamol concentration at 4 hours or more since the time of ingestion: Note earlier paracetamol concentration measurements are clinically uninterpretable. If there is absolute certainty that a lower single dose of paracetamol of less than 150 mg/kg body weight has been ingested, or less than 75 mg/kg in children at enhanced risk of liver damage, this can reasonably be considered unnecessary and the child may be discharged.
- Start NAC (applies to children with toxic range ingestion when levels are not available) if the plasma paracetamol concentration is above line A of the paracetamol overdose treatment graph or above line B for “at enhanced risk” patients (Fig. 58.1).
Paracetamol Poisoning

"Need not to be admitted if the plasma paracetamol concentration is below the relevant line on the treatment graph and the history is consistent with less than 150 mg/kg body weight paracetamol having been ingested.

When NAC is started within 8 hours of the overdose, complications are unlikely and the child will most likely be fit for discharge immediately on completion of infusion. International normalized ratio (INR) and enzymes should be normal at discharge.

Advice should be given for the child to return to hospital if vomiting or abdominal pain develop or recur.

**Management of all Patients Who Present 8–15 Hours after Ingestion or When Paracetamol Levels are not Available**

- Urgent action if it is thought that more than 150 mg/kg body weight or in adults, a total of more than 12 g (whichever is the smaller) has been ingested. Start NAC immediately without waiting for the result of the plasma paracetamol concentration.
- Blood for an urgent
  - Plasma paracetamol concentration
  - PT/INR

Figure 58.1: The curve for the treatment showing both normal risk and high-risk lines for treatment with NAC from NPIS guidelines
– Plasma creatinine
– Liver enzymes.

- Discontinue NAC if (continue if any doubt as to the timing of the overdose)
  - The plasma paracetamol concentration is below the relevant treatment line on the graph
  - Normalized INR, plasma creatinine and ALT and → not normal
  - Asymptomatic

- Discharge if the patient has normal INR, plasma creatinine and ALT, and is asymptomatic

- Continue infusion and supportive treatment till normal (72 hours)

- Before discharge, take blood sample for INR, creatinine and liver enzymes

- Discharge asymptomatic patients with normal INR, plasma creatinine and liver enzymes; they are advised to return to hospital if vomiting or abdominal pain develop or recur.

**Management of all Patients Who Present 15–24 Hours after Ingestion**

The treatment is as above. The prognostic accuracy of these lines on the graph after 15 hours is uncertain but a plasma paracetamol concentration above the relevant treatment line should be regarded as carrying serious risk of severe liver damage (Fig. 58.2).

**Dosage for NAC Intravenous Infusion**

<table>
<thead>
<tr>
<th>Adult and child over 12 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Load 150 mg/kg in 200 ml 5% Dx over 15 minutes</td>
</tr>
<tr>
<td>Followed by 50 mg/kg in 500 ml 5% Dx over 4 hours</td>
</tr>
<tr>
<td>100 mg/kg in 1000 ml glucose 5% given over 16 hours</td>
</tr>
<tr>
<td>(half the amount of fluid volume but same dose of NAC for children over 20–40 kg)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Children 1 month to 5 years (under 20 kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Load 150 mg/kg in 3 ml/kg 5% Dx over 15 minutes</td>
</tr>
<tr>
<td>50 mg/kg in Dx 5% given at 7 ml/kg over 4 hours</td>
</tr>
<tr>
<td>100 mg/kg in Dx 5% at 14 ml/kg given over 16 hours</td>
</tr>
</tbody>
</table>

Dextrose and normal saline/normal saline (DNS/NS) solution may be substituted.

As most of us do not have levels available, the protocol would be as in * and ** where NAC would be administered giving the benefit of doubt to all patients who fall within the toxic ingestion limits (Fig. 58.2).
Figure 58.2: Rumack-Matthew nomogram for single acute acetaminophen poisoning

Further Reading

Paracetamol Poisoning Algorithm

Clinical scenario
- Accidental in children below 6 years but suicidal in adolescents
- Maximum daily dose is 90 mg/kg. A single dose of more than 150 mg/kg is potentially toxic.
- Hepatotoxicity is mainly due to NAPQI

Clinical features
- **Stage 1**: Lasts up to 24 hours. Symptoms include anorexia, nausea and vomiting.
- **Stage 2**: Presents between 24 hours and 48 hours. Symptoms include right upper quadrant pain and tenderness that coincides with transaminase elevation.
- **Stage 3**: Develops 3–4 days post-ingestion. Symptoms of hepatic failure with jaundice, bleeding or encephalopathy develop. Death may occur due to cerebral edema or sepsis.
- **Stage 4**: Develops 4–14 days post-ingestion. This is stage of recovery and hepatic enzymes levels come down.

Investigations
- Liver function tests
- Renal function tests

Management

Supportive management
- GI decontamination activated charcoal
- Gastric lavage only in recent (within 60 min) and potentially life-threatening toxicity

Specific treatment
- NAC is antidote for paracetamol poisoning.
- NAC acts by directly detoxifying NAPQI to nontoxic metabolites. It also replenishes glutathione stores.
- Oral dose is 140 mg/kg followed by 70 mg/kg every 4 hours for three 3 days up to total of 17 doses.
- IV therapy with NAC is recommended in GI bleeding or obstruction, potential fetal toxicity from maternal toxicity, oral intolerance or when patient is unconscious.

Abbreviations: NAPQI, N-acetyl-p-benzoquinoneimine; GI, gastrointestinal; NAC, N-acetyl cysteine.
Iron toxicity in pediatric age is commonly due to accidental consumption of mother's sugar-coated iron tablets by the child. Toxic dose is 10–20 mg/kg elemental iron and more than 60 mg/kg can be lethal. Toxic effects are on local gastrointestinal (GI) mucosa leading to corrosion and occasionally perforation. Systemic effects are through mitochondrial toxicity leading to deranged energy metabolism. Systemic effect is predominantly through metabolic acidosis and is mainly on liver, lungs and heart. Diagnosis is made by history, and serum iron level helps in monitoring the management. Within 6 hours of ingestion, local GI symptoms manifest, and later, there may be short-term clinical improvement due to conservative management; but subclinically, the child is deteriorating with development of metabolic acidosis. If not intervened, the child develops hepatic failure, cardiogenic shock and severe metabolic acidosis after 24 hours of consumption. After 2–6 weeks, sequel of GI corrosion may develop with scars and strictures.

Serum iron levels taken at 2–6 hours can indicate the severity of toxicity as mild toxicity: less than 300 µg/dL, moderate: 300–500 µg/dL and severe: greater than 500 µg/dL. X-ray abdomen soon after consumption of iron tablets can detect nonabsorbed iron in the gut.

**Treatment**

Treatment is at four levels:

1. **Primary stabilization** focuses at airway, breathing and circulation. In iron toxicity, fluid and cardiovascular stabilization is a challenging task.
2. **Prevention of further absorption of iron from the GI tract**: Ipecac-induced vomiting and gastric lavage are not recommended since they are not effective, and at times, can be harmful. Whole bowel irrigation with polyethylene glycol electrolyte solution (PEGES) is used either orally or by nasogastric tube to be given in infusion at 30 ml/kg/hr to be continued till effluent coming per rectum is clear. Whole bowel irrigation is contraindicated in suspected or impending perforation and obstruction. Activated charcoal is not effective in preventing absorption of iron from the gut.
3. Deferoxamine is the iron-chelating agent of choice. Deferoxamine binds absorbed iron, and the iron-deferoxamine complex is excreted in the urine. Deferoxamine does not bind iron in hemoglobin, myoglobin or other iron-carrying proteins. Indications for using deferoxamine are based on both clinical and laboratory parameters.

Indications for the treatment include shock, altered mental status, persistent GI symptoms, metabolic acidosis, pills visible on radiographs, serum iron level greater than 500 µg/ml or estimated dose greater than 60 mg/kg of elemental iron. Always initiate chelation if serum iron levels are not available and symptoms are present. Deferoxamine is given by IV infusion 15 mg/kg/hr for 6 hours in mild case, for 6–12 hours in moderate case and for 24 hours in severe case. Clinical end point of treatment can be resolution of shock and acidosis. Side effects can be allergic reactions, hypotension and acute respiratory distress syndrome (ARDS) if infusion is given for longer duration. Reddish color urine confirms effective iron chelation with deferoxamine.

4. Surgical intervention may be needed if iron is radiologically detected in the gut after bowel irrigation. It can be removed surgically or scopically, and further absorption or perforation can be avoided.

Iron Poisoning

**Primary stabilization**
Airway, breathing and circulation

**Routine ICU investigations with serum iron levels**
(not mandatory for starting treatment)

**Prevention of further absorption**
- Gastric lavage if patients report within 2 hours of consumption.
- Whole bowel irrigation with polyethylene glycol solution

**Deferoxamine**
**Indications:** Shock, altered mental status, persistent gastrointestinal symptoms, metabolic acidosis, pills visible on radiographs, serum iron level > 500 µg/ml or estimated dose > 60 mg/kg of elemental iron

**IV infusion:** 15 mg/kg/hr for 6 hours in mild case, for 6–12 hours in moderate case and for 24 hours in severe case

**Clinical end point** of treatment can be resolution of shock and acidosis.

**Intense hemodynamic, acid-base and electrolyte monitoring**

**Surgical/scopical removal**
If there are radiologically detected pills after whole bowel irrigation.
Further Reading

Iron Poisoning Algorithm

Clinical scenario
- More common in males below 5 years
- Most cases are due to accidental ingestion of iron tablets prescribed to mother for anemia prophylaxis
- Clinical features arise due to corrosive damage to gastrointestinal (GI) mucosa and due to reactive oxygen species

Clinical features
- Stage 1: Last up to 3 hours. Symptoms include abdominal pain, hematemesis, nausea and vomiting.
- Stage 2: Presents up to 12 hours. Period of apparent stability. Symptoms subside.
- Stage 3: Develops 12–48 hours postingestion. This is the stage of mitochondrial toxicity. Shock, acidosis, coma, seizures, coagulopathy and hypo/hyperglycemia are seen.
- Stage 4: Develops after 48 hours postingestion. This is the stage of hepatic necrosis. Jaundice and hepatic encephalopathy may develop.
- Stage 5: Develops after 2–4 weeks postingestion. Gastric scarring, gastric and pyloric strictures.

Investigations
- Complete blood count (CBC) and electrolytes
- Coagulation profile
- Liver function tests
- Renal function tests
- Arterial blood gas (ABG)
- X-ray chest and abdomen
- Serum iron level

Contd...
Contd...

**Management**

- Plan management as per alleged iron ingestion
- < 20 mg/kg (Little risk): Decontaminate and observe for at least 6 hours
- 20–60 mg/kg (Moderate risk): Decontaminate and observe for 6 hours. Consider desferrioxamine chelation.
- > 60 mg/kg (High risk): Decontaminate and start desferrioxamine chelation.

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**Supportive management**

- Gastric lavage with normal saline/tap water
- Whole bowel irrigation with polyethylene glycol is done. Dose is 30–40 ml/kg/hour for 4–8 hours. Contraindicated in gastric ileus obstruction
- Monitor airway, breathing and circulation
- Close cardiorespiratory monitoring
- Inotropes for hypotension

**Specific treatment**

- Desferrioxamine chelation is specific antidotal treatment
- Desferrioxamine is given as IV infusion in normal saline at the rate of 15 mg/kg/hour (maximum daily dose 360 mg/kg and total 6 g)
- Stable clinical state of patient along with vin rosé color of urine indicates response to desferrioxamine chelation
- Serum iron level < 100 μg/dl is appropriate end point
Organophosphorus Poisoning

In suspected cases of poisoning if cholinergic toxidromes are noticed then commonest toxic agents suspected in Indian population are organophosphates (OPs) and carbamates. Common OP agents observed in poisoning are malathion, fenitrothion, diclorvos, etc. This poisoning records almost 3–25% mortality rate.

**Pathophysiology**

Organophosphate agents are cholinesterase inhibitors, thus increase the synaptic levels of acetylcholine (Ach). Effects of increased Ach levels are mainly at three levels:

1. **Nicotinic effect:** Neuromuscular junctions at skeletal muscles—increased stimulation at this level cause fasciculations, cramps, and later paralysis, weakness.
2. **Muscarinic effects:** Postganglionic parasympathetic stimulation—increased stimulation leads to increased exocrine secretions in body, e.g. sweat gland, salivary gland, bronchial secretions and excessive smooth muscle contraction leading to hyperperistalsis, micturition, etc. The end effect is clinical presentation of SLUDGE, i.e. Salivation, Lacrimation, Urination, Diarrhea, gastrointestinal (GI) upset, Emesis. Postganglionic sympathetic stimulation due to larger doses of intoxication can lead to tachycardia, hypertension and arrhythmia.
3. **Central effects:** Direct effect on brain leads to ataxia, disorientation, drowsiness, seizures, coma, etc.

In pediatric patients, most common presenting complaints are seizures and coma which may be due to proportionately higher dose of toxin and patients brought late after consumption.
Diagnosis
Essentially it is clinical diagnosis but sometimes laboratory support may be needed for diagnosis as well as for monitoring the patient. Red blood cell (RBC) cholinesterase levels correlate with central nervous system (CNS) cholinesterase levels and levels less than 25% of baseline, denote poisoning. Plasma cholinesterase is liver generated acute phase reactant and is not reliable parameter for diagnosis.

Serial monitoring of levels are more reliable since baseline levels of individual patient are not known. This parameter helps more in monitoring recovery of the patient and levels above 75% denote good recovery.

Rest investigations are done to monitor various system functions while treatment.

Treatment
The most common cause of death in OP poisoning is hypoxia due to airway compromise secondary to bronchospasm, bronchorrhea, laryngospasm and seizures. Equally important cause is hemodynamic failure due to sympathetic overstimulation in severe intoxication. Aggressive airway, breathing, circulation (ABC) support and intense respiratory and hemodynamic monitoring is essential for successful outcome.

Endotracheal (ET) intubation and mechanical ventilation may be needed to protect the airway and maintain adequate breathing. Invasive hemodynamic monitoring also may be needed.

Specific treatments are:

- **Surface decontamination**: Organophosphate poisoning can occur through oral consumption, skin contamination, inhalation and conjunctival absorption. Patient’s clothing should be removed first and skin should be cleaned with soap and water. Gastric lavage should be done after protecting the airway. Conjunctiva can be cleaned with normal saline (NS) or Ringer’s lactate solution.

- **Medication**:
  - Aggressive treatment with atropine is done. Dose is 0.05 mg/kg IV to be repeated every 5 minutes. Subsequent doses can be doubled. It is to be continued till signs of atropinization are achieved, i.e. bronchial and salivary secretions are dried. Pupillary dilatation is not the sign of atropinization. Tachycardia is not limiting factor for atropine injections. Once atropinization is achieved, periodic low dose is needed for next 48 hours while observing for recurrence of signs.
  - 2-Pyridine aldoxime methyl chloride (2-PAM, pralidoxime) should be used aggressively. OP phosphorylates cholinesterase to deactivate it temporarily. Subsequently this deactivated enzyme is hydrolyzed in body to get permanently deactivated. 2-PAM reactivates the phosphorylated
cholinesterase. 2-PAM is not needed in carbamate poisoning because cholinesterase is temporarily deactivated in carbamate poisoning. Dose of 2-PAM is 50 mg/kg in NS infusion over 30 minutes followed by 20 mg/kg/hour for next 24 hours.

- **Other supplementary medications**: Inotropes, cardiac stabilizers like magnesium sulphate may be needed in acute phase of poisoning.

Suspected case of OP poisoning should be observed for at least 12 hours. Confirmed case should be observed for at least 48 hours after clinical recovery. Three types of paralytic syndromes are observed in OP poisoning:

1. **Acute paralysis during active poisoning in initial 1–3 days**. This will recover fast with treatment.
2. **Intermediate syndrome**: Four to eighteen days presentation probably due to incomplete initial treatment. Mainly proximal and truncal weakness noticed.
3. **OP-induced delayed polyneuropathy (OPIDP)** due to inhibition of neuropathy target esterase. It is mainly distal weakness. Starts at 2–3 weeks and may recover after 12 months.

**Algorithm**

- **Recognize OP poisoning**
  - **History of consumption**
  - **Presentation with cholinergic toxidrome**

  **Assess ABC**: Airway cleaning and stabilization; if not maintainable, ET intubation and sedation optimization strategy (SOS) ventilation, hemodynamic support with fluids and inotropes as per need

  **Specific treatment**

  **Surface decontamination**: Remove all clothes; in case of inhalation poisoning, take victim out of accident site; eye and skin cleaning.

  **Gastric lavage**: Normal saline lavage within 2 hours of consumption.

  **Activated charcoal**: 1 g/kg stat and repeat SOS after 4–6 hours.

  Intense respiratory and hemodynamic monitoring is the key to success. Monitoring RBC cholinesterase can guide treatment.

  **Medications**

  **Atropine**: Dose 0.05 mg/kg to be repeated every 5 minutes till atropinization is achieved (atropinization is denoted by drying of secretions and not pupillary dilatation).

  **2-PAM (pralidoxime)**: Given in all OP poisoning cases except in carbamate confirmed cases where it is not needed. Dose: 50 mg/kg over 1 hour in NS followed by infusion of 20 mg/kg/hour in severe intoxication or 20 mg/kg 6 hourly boluses.
Further Reading

Salicylates were commonly used in the past for their analgesic, antipyretic and anti-inflammatory action. In recent times, paracetamol has replaced it in many conditions of day-to-day use.

**Pathophysiology**

Salicylate intoxication can occur by oral route or topical route in chronic large dose application. In smaller doses, it stimulates respiratory center leading to respiratory alkalosis, and in higher doses it inhibits the Krebs cycle of metabolism thus limiting the production of adenosine triphosphate (ATP) which leads to multisystem failure. In the process it also inhibits amino acid metabolism and stimulates lipid metabolism. Its renal effects induces excretion of sodium, potassium and bicarbonate molecules. End effect of all is severe metabolic acidosis. Initially urine is alkaline but with severe metabolic acidosis urine becomes acidic.

The following four categories are helpful for assessing the potential severity and morbidity of an acute, single event, nonenteric-coated, salicylate ingestion:

1. **Less than 150 mg/kg**: Spectrum ranges from no toxicity to mild toxicity
2. **From 150–300 mg/kg**: Mild-to-moderate toxicity
3. **From 301–500 mg/kg**: Serious toxicity
4. **Greater than 500 mg/kg**: Potentially lethal toxicity.

**Clinical Presentation**

It depends upon time elapsed since intoxication and dose of intoxication.

- **Respiratory**: Tachypnea, bradypnea, apneas, pulmonary edema, etc.
- **Auditory**: Ototoxicity, tinnitus, deafness.
- **Cardiovascular**: Tachycardia, arrhythmias, shock.
- **Central nervous system**: Cerebral edema, encephalopathy, tremors, coma.
Salicylate Poisoning

- **Gastrointestinal**: Hemorrhage, gastritis, perforation, pancreatitis, hepatitis.
- **Dyselectrolytemia**: Hypokalemia, hypocalcemia, acidemia, syndrome of inappropriate antidiuretic hormone secretion (SIADH)
- **Hematologic**: Prolonged bleeding profile.

**Laboratory Studies**

Basic hematological profile, bleeding, renal, hepatic profiles, electrolytes and arterial blood gases need to be performed.

Salicylate levels to be checked at 6 hours and periodically thereafter to note the response to treatment.

Done nomograms can be followed up for toxicity levels.

- **Less than 50 mg/dl**: Asymptomatic
- **51–110 mg/dl**: Mild-to-moderate toxicity
- **110–120 mg/dl**: Severe toxicity.

**Urine analysis**: It is done for toxin diagnosis and excretion. After treatment starts with urine alkalization, urine pH should be maintained between 7.5 to 8 and blood pH should be maintained in the range of 7.3–7.5.

X-ray chest may show respiratory signs as pulmonary edema or aspiration. X-ray abdomen may show impaction of tablets in stomach or intestines which may have delayed absorption and prolonged toxicity.

**Treatment**

**Criteria for Admission or Observation**

- Infants and children
- Poisoning with sustained release formulation
- Those with acute consumption > 150 mg/kg.

Therapeutic objectives include:

- Cardiopulmonary stabilization
- Prevention of absorption
- Correction of fluid deficits
- Correction of acid-base abnormalities
- Enhancement of excretion and elimination.

Initial airway, breathing circulation stabilization should be followed by efforts to remove the toxin from gastrointestinal (GI) tract, to prevent its absorption and to facilitate its excretion. Early endotracheal intubation helps to protect the airway and optimize ventilation especially in severe acidosis.

**Methods to Prevent Absorption**

- **Emesis with ipecac**: Not safe without airway protection and yield is minimal so not done in present clinical practice.
- **Gastric lavage**: Yield of 30–70% if performed within 1 hour of toxin consumption and airway protection is must.
- **Activated charcoal**: Very effective way of preventing absorption. Dose 1 g/kg in powder form and can be repeated after 4–6 hours. Catheretic dose can be given with first dose.

- **Whole bowel irrigation**: Whole bowel irrigation with polyethylene glycol (Pegleg) is indicated if pills are impacted in bowels or sustained release formulation is used in intoxication.

- **Correct fluid balance**: Correct deficits by Ringer’s lactate or normal saline and give maintenance to achieve urine output of > 1 ml/kg/hour. Optimize the fluid to have alkalinization of urine and to prevent pulmonary edema.

- **Urinary alkalinization**: Urinary alkalinization to be done if plasma salicylate levels are > 35 mg/dl and increasing or acid-base imbalance is increasing. It is achieved by single intravenous (IV) bolus of NaHCO₃ at 1–2 mEq/kg. Follow this with a constant infusion of 5% dextrose in water (D5W) with NaHCO₃ 100–150 mEq/L and KCl 20–40 mEq/L at 1.5–2.5 ml/kg/hour to produce a urine flow of 0.5–1 ml/kg/hour. Closely monitor the serum electrolytes and urine pH, and maintain the urinary pH between 7.5 and 8. Adequate potassium levels should be maintained to achieve adequate urinary alkalinization.

- **Hemodialysis**: Hemodialysis is the best method of removing salicylates from blood. It is indicated in severe clinical intoxication, severe acidosis, plasma levels of > 100 mg/dL.

- During the treatment blood sugar has to be monitored intensely to avoid hypoglycemia which can lead to long-term central nervous system (CNS) damage.

---

**Algorithm**

<table>
<thead>
<tr>
<th>Criteria for admission or observation</th>
<th>Therapeutic objectives include</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Infants and children</td>
<td>- Cardiopulmonary stabilization</td>
</tr>
<tr>
<td>- Poisoning with sustained release formulation</td>
<td>- Prevention of absorption</td>
</tr>
<tr>
<td>- Those with acute consumption &gt; 150 mg/kg</td>
<td>- Correction of fluid deficits</td>
</tr>
<tr>
<td>- Those with acute consumption &gt; 150 mg/kg</td>
<td>- Correction of acid-base abnormalities</td>
</tr>
<tr>
<td>- Those with acute consumption &gt; 150 mg/kg</td>
<td>- Aggravate excretion and elimination</td>
</tr>
</tbody>
</table>

**Management**

- **Airway, breathing, circulation stabilization**
  - Airway support and SOS early intubation to optimize ventilation while correcting acidosis

- **Prevention of absorption**
  - *Gastric lavage*: if patient presents within 2 hours of consumption
  - *Activated charcoal*: 1 g/kg to be repeated after 4–6 hours
  - *Whole bowel irrigation*: if sustained release product is consumed.

- **Correct fluid deficit and optimize intravascular volume with normal saline or Ringer’s lactate**

- **Urinary alkalinization**: Do if salicylate levels > 35 mg/dL or increasing acidosis. Single IV bolus of NaHCO₃ at 1–2 mEq/kg followed by constant infusion of D5W with NaHCO₃ 100–150 mEq/L and KCl 20–40 mEq/L at 1.5–2.5 ml/kg/hour to produce a urine flow of 0.5–1 ml/kg/hour.

- **Monitor**: serum electrolytes urine pH between 7.5–8 blood pH between 7.3–7.5 maintain euglycemia

- **Hemodialysis**: If severe acidosis or plasma salicylates > 100 mg/dL
Further Reading

Hydrocarbons are a diverse group of compounds: (a) hydrocarbons which are easily aspirated following ingestion and (b) hydrocarbons that may produce systemic toxicity in addition to their aspiration potential. The first group includes aliphatic hydrocarbons such as kerosene, mineral spirits, gasoline, naphtha and mineral seal oil. They are poorly absorbed from the gastrointestinal (GI) tract, and therefore are not expected to produce systemic effects.

Pulmonary effects are the result of aspiration. A severe necrotizing pneumonitis, with direct tissue destruction, can occur. Aspiration can occur at the time of ingestion, or during vomiting or gastric lavage. Aspiration can occur from minute amounts of hydrocarbon. Pulmonary toxicity represents the most common complication of hydrocarbon ingestion and reason for mortality.

- When aspiration occurs, the patient may initially experience coughing, choking, gagging or grunting respirations. Dyspnea and cyanosis may be noted. Rales, rhonchi and decreased breath sounds may be present by auscultation. Fever and leucocytosis may also be present, but are not thought to correlate with an infectious process.
- X-ray findings are usually significant at 2–8 hours after ingestion. Pulmonary infiltrates or perihilar densities have commonly been seen. Following aspiration, deterioration of the patient may occur over the first 24–72 hours, with resolution of symptoms in 3–6 days. Reported complications of hydrocarbon aspiration include pneumatoceles, pleural effusion or pneumothorax. Bacterial superinfection is also possible. Hemorrhagic edema can rapidly lead to the patient’s demise.
- Aspiration of aliphatic hydrocarbons may result in lethargy, tremors, and rarely, convulsions or coma. These effects are more likely due to severe pulmonary injury or hypoxia. Death from central nervous system (CNS) depression following aliphatic hydrocarbon ingestion has not been reported. Treatment is supportive.
Hydrocarbon Ingestion

Treatment

Recommendations can be made based on properties of the ingested hydrocarbon.

- Regardless of the amount involved, gastric emptying is not indicated for accidental ingestion of a hydrocarbon lacking systemic toxicity (Kerosene). The risk of aspiration during vomiting or lavage far outweighs any benefit from removal of the substance.

- In the case of ingestion of a hydrocarbon capable of causing systemic toxicity, or, when coingestion is suspected, GI decontamination would be warranted. The mnemonic CHAMP has been proposed for delineation of toxic hydrocarbons—Camphorated, Halogenated, Aromatic, heavy Metals and Pesticides. The primary toxicity of these agents relates to their systemic effects, and treatment should be based on expected effects.

- An endotracheal tube cuff is not protective against aspiration. For these reasons, syrup of ipecac is the preferred method of gastric emptying, provided the patient has a gag reflex, is alert, and is likely to remain so, and provided the substance is not expected to cause seizures.

- Activated charcoal does not effectively adsorb hydrocarbons, and in the absence of coingestants, has no role in therapy.

- Oxygen and aggressive respiratory support are indicated. For symptomatic children with severe pulmonary complications, continuous positive airway pressure (CPAP) or positive end-expiratory pressure (PEEP) may be required. Steroids have not been shown to be useful, and antibiotics should be reserved for documented infection. A beta-2 selective agonist can be given for bronchospasm, while epinephrine should be avoided as it may precipitate dysrhythmias.

- All symptomatic patients should have a chest X-ray (CXR) taken no sooner than 2 hours postingestion, and should be observed for a minimum period of 6 hours. The patient may be discharged with observation at home if asymptomatic throughout and X-ray is negative. In the presence of a positive 2-hour X-ray, the patient should be admitted for monitoring of blood gases, repeat chest X-rays, and respiratory support if required (Table 53A.1).

- Ventilation criteria are no different from any other. CPAP, PEEP and low tidal volume ventilation with vigilance for the occurrence of pneumothorax is the key. Hemodynamic support is often needed.

- Diarrhea is often seen with ingestion of significant amounts but this warrants no special treatment.

Table 53A.1: Triage of patients—admission criteria

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Chest X-ray*</th>
<th>Disposition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>Abnormal</td>
<td>Admit</td>
</tr>
<tr>
<td>No</td>
<td>Normal</td>
<td>Discharge</td>
</tr>
<tr>
<td>Yes</td>
<td>Normal</td>
<td>Repeat chest X-ray (CXR) in 6–8 hours</td>
</tr>
<tr>
<td>No</td>
<td>Abnormal</td>
<td>Admit</td>
</tr>
</tbody>
</table>
Drowsiness and coma with seizures may also be seen with the nonaliphatic hydrocarbons but mortality is usually from respiratory failure.

In the case of certain inhaled hydrocarbons especially in vulnerable populations like street children, huffing causes neurological symptoms and overdoses can be overlooked.

Abbreviations: GI, gastrointestinal; CNS, central nervous system; ABG, arterial blood gas; PEEP, positive end-expiratory pressure
Scorpion Sting Poisoning

Poisonous scorpion sting is common rural emergency in western India, Konkan, Karnataka, and other sporadic geographical pockets in India. There are two poisonous species; the *Mesobuthus tamulus* or Indian red scorpion and *Palamneus gravimanus* or Ingli. The red scorpion can be fatal if emergency treatment is not given.

**Pathophysiology and Clinical Features**

Poisonous scorpion in India mainly has its effect on cardiovascular system. It also has some effect on glucose metabolism and occasionally on nervous system. Poisonous sting usually do not cause pain.

Scorpion venom after sting causes autonomic storm. Initially parasympathetic system is stimulated leading to excess sweating, cold extremity, salivation, vomiting and defecation due to hyperperistalsis and priapism in male child. There is bradycardia at this stage and child may be irritable and anxious. Parasympathetic phase usually last for 1–2 hours and gradually gives way to sympathetic overdrive. Both alpha (α) and beta (β) activity is stimulated. Alpha stimulation is more predominant giving rise to peripheral vasoconstriction and increase after load while β stimulation causing tachycardia and increased contractility. End effect is increased cardiac workload leading to cardiac failure, pulmonary edema, arrhythmias and death. Sympathetic overtone progress may occur in initial 6–12 hours.

Metabolic derangements mainly lead to insulin suppression and diabetic ketoacidosis like state.

Neurological manifestations are very rarely seen in Indian scorpion envenomations.

**Investigations**

Diagnosis is purely clinical and investigations are needed to monitor the clinical status while treatment. X-ray chest to know the pulmonary condition and
electrocardiography (ECG) to monitor heart are routine investigations. Cardiac enzyme levels are not very sensitive to record cardiac injury.

**Treatment**

Understanding the pathophysiological state of envenomation will guide the treatment. Usually painful stings are not poisonous. At times poisonous stings start becoming painful after starting treatment. This is due to vasodilatation relieving nerve ischemia.

Supportive treatment with intravenous (IV) fluids to correct dehydration and oxygen if patient is hypoxic or tachypneic should be started. Patient developing pulmonary edema or cardiac failure may need ventilatory support. There is no role of any local treatment in case of poisonous sting.

After confirming clinical signs of envenomation, pharmacological antidote, i.e. prazosin should be given, irrespective of stage of poisoning. This is selective α-receptor blocker relieving vasoconstriction thus reducing the afterload and improves the cardiac function. If patient presents in early hypertensive phase, systemic (oral/sublingual) nifedipine can be tried to reduce blood pressure immediately, but one should carefully rule out impending or established cardiac failure before giving nifedipine since it is a negative inotropic drug (calcium channel blocker) and can aggravate congestive cardiac failure (CCF). If patient presents in severe vasoconstriction phase with CCF, potent vasodilators like sodium nitroprusside (SNP) or nitroglycerine (NTG) infusion can be given in infusion. Impending cardiac failure can be treated with dobutamine or phosphodiesterase inhibitor like milrinone infusion. Evidence of significant cardiac injury with ischemia and arrhythmias may be seen in few severe cases. They can be treated with NTG infusion. Glucose insulin infusion has been tried in such conditions successfully in few studies.

Prazosin still remains pharmacological gold standard of treatment. Dose is 40 µg/kg first dose followed by 20 µg/kg 3–4 hourly for next 12–24 hours while monitoring the clinical progress of patient. Rarely catastrophic fall in blood pressure is recorded following prazosin or SNP which should be controlled with fluid boluses and appropriate inotropes.

Scorpion antivenom was used since almost a decade but recently has been tried in larger studies and has shown response much faster than and as effective as prazosin. It is given as 30 ml (three vials) first dose and to be repeated after 2–4 hours later depending upon response. Being horse serum derived antivenin, precautions of allergic reactions should be taken. Its major drawback is poor availability and it is effective only in the areas of manufacturing.

**Pharmacotherapy**

- *Prazosin*: α1-adrenergic receptor blocker; dose, 30 µg/kg in children every 6 hourly and can be repeated 3 hourly till there are signs of clinical response.
Scorpion Sting Poisoning

- **Pulmonary edema:**
  - Intravenous furosemide
  - Intravenous aminophylline
  - Intravenous SNP (0.3–8 µg/kg/min)  
  - Intravenous NTG (5–8 µg/kg/min)  
  Stop prazosin and restart  
  - Intravenous NTG (5–8 µg/kg/min)  
  30 minutes before stopping SNP/NTG.

- In case of shock or hypotension:
  - Dobutamine: 5–15 mcg/kg/min
  - Dopamine: 3–5 mcg/kg/min

- Other therapies like oxygen inhalation, intubation and ventilation, as need

- Cardiac arrhythmias
  - Ventricular premature contractions (VPCs) at times ventricular bigeminy
    are self-limiting (correct electrolytes and hydration).
  - Intravenous lidocaine for ventricular tachycardia (VT)
  - Mexiletine: Used in VPC as it does not alter QTc interval.
  - Intravenous amiodarone.
  - Intravenous milrinone.

**Doses of Medications**

- **Prazosin:** 40 mg/kg stat followed by 20 mg/kg
- **Nifedipine:** 0.5–1 mg/kg
- **Sodium nitroprusside:** 0.1–0.3 µg/kg/min
- **Nitroglycerine:** 0.2–0.3 µg/kg/min
- **Dobutamine:** 10–20 µg/kg/min
- **Milrinone:** 0.25–0.75 µg/kg/min

**Algorithm**

<table>
<thead>
<tr>
<th>Recognize the sting</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of sting</td>
</tr>
<tr>
<td>Local pain, no</td>
</tr>
<tr>
<td>systemic signs</td>
</tr>
<tr>
<td>Nonpoisonous</td>
</tr>
<tr>
<td>No pain, systemic</td>
</tr>
<tr>
<td>toxicity</td>
</tr>
<tr>
<td>Incidental findings</td>
</tr>
<tr>
<td>of sweating,</td>
</tr>
<tr>
<td>cold limbs,</td>
</tr>
<tr>
<td>hypertension,</td>
</tr>
<tr>
<td>priapism, bradycardia and</td>
</tr>
<tr>
<td>later tachycardia.</td>
</tr>
<tr>
<td>Poisonous sting</td>
</tr>
<tr>
<td>No local treatment</td>
</tr>
</tbody>
</table>

Contd...
Further Reading

Mammalian Bites

Dog bites (80–90%), cats (5–10%), rodents (2–3%) and other wild or domestic animals

Human bites share with cat wounds a notoriously high infections and complication rate.

Management

- Meticulous and prompt local care; clean with 1% povidone iodine and irrigate with 200 ml of normal saline.
- Open lacerations can be sutured if local care is effected in several hour (facial wounds often mandate primary closure for cosmetic reasons).
- Extremities with extensive wounds should be immobilized in a position of function and kept elevated. Recheck wound in next 24–48 hours.

Suggested indications for antibiotics:
- Human and cat bites through dermis
- Bites closed prematurely
- Bites more than 8 hours old with significant crush injury or edema
- Potential damage to bones, joints or tendons
- Bites to hands and feet
- Patients with increased risk of infection
- Signs of infection within 24 hours

- Tetanus prophylaxis
- Rabies prophylaxis
  - Indicated for bites—dogs, cats, monkey, skunks, foxes, bats, raccoons, mongoose, jackals, hyena, cattle
  - Rabies immunoglobulin (RIG), 20 IU/kg once half locally infiltrate and half intramuscularly.
  - Rabies human diploid cell vaccine (HDCV) immunization: 0, 3, 7, 14 and 28 for 5 doses of 1.0 ml each IM.

Further Reading

Snake Bite Management Algorithm

Prehospital management
- Reassurance
- Tourniquet:
  - Width: 4 inch
  - Apply proximal to bite
  - Loose enough to allow one finger
  - and distal pulses should be well felt
  - Remove only after anti-snake venom has been administered
- Immobilize bitten part
  - Use a sling or splint
- Avoid:
  - Incision over bite marks
  - Sucking out venom
  - Local ice packs

Rapid assessment and resuscitation in the emergency ward
- Assess airway, breathing, and circulation (ABGs)
- Mechanical ventilation is indicated for respiratory paralysis in case of neurotoxicity by Cobra and Krait

Dosage and administration of anti-snake venom (ASV)
- Sensitivity testing: 0.1 unit of ASV diluted in 1 ml diluent and injected subcut
- Dose of ASV same for adult and child: 10 vials dissolved in 200 ml of normal saline and infused over 1 hour, then five vials given over next 24 hours
- Maximum dose: 30 vials for neurotoxic, 40 vials for coagulopathic effects
- Best when given within 4 hours of bite but useful in symptomatic patients even up to 1 week after the bite

Indications for ASV for local envenomation
- Local swelling involving more than half of bitten limb
- Swelling after bite on digits
- Rapid expansion of swelling
- Enlarged tender region

Indications for ASV for systemic envenomation
- Hemostatic abnormalities
- Neurotoxic signs
- Shock dysrhythmias
- Acute renal failure, hemoglobinuria, myoglobinuria or evidence of intravascular hemolysis
- Vomiting, headache, inappropriate drowsiness

Other supportive measures
- Neostigmine:
  - Useful for neuromuscular junction blockade caused by neurotoxin snake bites
  - Dose: 25–50 μg/kg 4 hourly till complete neurological recovery
  - To be given with atropine
- Local wound management
  - Clear thoroughly and keep open
  - Hourly progression of edema is to be recorded
  - Wound debridement often required on day 3 to 5
- Antibiotics if injection at bite site is suspected
- Booster dose of tetanus toxoid
- Manage renal failure conservatively; occasionally peritoneal dialysis may be required
“Dead” snakes and decapitated snakes can bite reflexively for up to 1 hour.1
Antivenom is indicated if evidence is observed of systemic envenomation or progressive limb swelling or necrosis.
“Dead” snakes and decapitated snakes can bite reflexively for up to 1 hour.
Antivenom is indicated if evidence is observed of systemic envenomation or progressive limb swelling or necrosis.
Investigations: Complete blood count (CBC), coagulation studies, platelet count, urinalysis blood cross match, red blood cells (RBCs), myoglobin, serum electrolytes, blood urea nitrogen (BUN), creatinine, fibrinogen, arterial blood gas (ABG), creatine kinase (CK), prothrombin time (PT) or international normalized ratio (INR).
### Classification of Envenomation Severity

<table>
<thead>
<tr>
<th>Grade</th>
<th>Clinical feature</th>
<th>Antivenom dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No envenomation</td>
<td>0</td>
</tr>
<tr>
<td>1</td>
<td>Minimal envenomation (local swelling and pain without progression)</td>
<td>5</td>
</tr>
<tr>
<td>2</td>
<td>Moderate envenomation (swelling, pain or ecchymosis progressing beyond the site of injury, mild systemic or laboratory manifestations)</td>
<td>10</td>
</tr>
<tr>
<td>3</td>
<td>Severe envenomation (marked local response, severe systemic findings and significant alteration in laboratory findings)</td>
<td>15</td>
</tr>
</tbody>
</table>

#### Antivenin
- Each vial is to be reconstituted with 10 ml of diluent (distilled water or isotonic saline); do not shake; roll between palms until it dissolves.
- Antivenin is to be given in normal saline (NS) or Ringer’s lactate (RL) (volume 20 ml/kg) as a slow infusion at a rate of 1 ml/min, increasing rate as per tolerance and infuse over 1 hour.
- Antivenin should be given in first 4 hours but may be efficacious even till 48 hours and reports suggest its effectiveness even after 6–7 days.
- Skin test with 0.2 ml intradermal of 1:10 diluted antivenin in forearm and control with NS on other hand
- A wheal or erythema greater than 10 mm in 30 minutes indicates hypersensitivity.
- Negative results do not rule out possibility of anaphylaxis, and positive result is not absolute contraindication to antivenin.

#### Further Reading
Approach to Pediatric Hypertension

Praveen Khilnani, Prashant Pruthi
Hypertensive Crisis in the PICU

S Senthil Kumar, KG Ravikumar, Bala Ramachandran

Hypertensive Crisis: Management Protocol

Definitions

Hypertension
Systolic/diastolic blood pressure greater than 95th percentile for the age/gender and height of the patient measured at least on three separate occasions 1–3 weeks apart.

Stage I Hypertension
Systolic/diastolic blood pressure exceeding 95th percentile and up to 5 mm above 99th percentile for age/gender and height of the patient.

Stage II Hypertension
Systolic/diastolic blood pressure exceeding 5 mmHg above 99th percentile for age/gender and height of the patient.

All patients falling into the stage II hypertension criteria are to be considered in hypertensive crisis. All patients with hypertensive crisis should be managed in PICU, irrespective of the etiology.

All patients falling into hypertensive crisis should be labeled into either of the two:

1. Hypertensive urgency: Stage II hypertension without end-organ damage.
2. Hypertensive emergency: Stage II hypertension with end-organ damage in the form of either of the following:
   - Hypertensive encephalopathy
   - Intracranial bleed
   - Acute left ventricular failure
   - Renal failure.
In patients with hypertensive emergencies, blood pressure should be controlled within hours while the same can be controlled in 2–3 days in case of urgencies. In hypertensive emergencies, target to achieve one-third reduction over 0–6 hours (except aortic dissection where it should be over 0–60 minutes), further one-third over 6–24 hours, and then the last third over next 48 hours.

**Protocol of Management**

**Clinical Evaluation: Goals**

- To differentiate emergency from urgency
- To know the extent of involvement (one organ dysfunction does not preclude evaluation of others)
- To know the duration and severity of pre-existing hypertension and any previous end-organ damage
- History of severe chest/abdominal pain with abrupt onset (to rule out acute aortic dissection)
- Examination to include 4-limb blood pressure, fundus examination (by an ophthalmologist to rule out chronic hypertension), features of cardiac failure and detailed neurological assessment
- Investigations to include complete blood count (CBC), renal function test (RFT), peripheral smear for evidence of hemolysis, urinalysis, ECG and chest X-ray
- CT scan can be a part of initial work-up only if clinical suspicion of intracranial bleed is present
- After initial work-up and stabilization, investigations for the secondary causes can be performed
- Brain MRI to check for the presence of posterior reversible leukoencephalopathy can be done after stabilization.

An important consideration prior to initiating IV therapy is to assess the patient’s volume status. Due to pressure natriuresis, patients with hypertensive emergencies may be volume depleted, and restoration of intravascular volume with IV saline solution will serve to restore organ perfusion and prevent a precipitous fall in blood pressure when antihypertensive regimens are initiated.

**Treatment**

- To assess airway, breathing and circulation initiate appropriate interventions.
- At least two IV lines to be inserted
- Arterial line and invasive arterial blood pressure monitoring (essential in case of hypertension emergency).
- Start oral drugs till the IV drugs are started.
- Goal is to prevent end-organ damage and bring down the blood pressure gradually except in the case of acute aortic dissection.
Neurological assessment every hourly.
Stat oral Tab. nifedipine 0.25–0.5 mg/kg at presentation in case of hypertensive emergency (the use of sublingual nifedipine is not recommended).

Sodium nitroprusside (Table 57.1)
- Dose: 0.5–5 µg/kg/min
- Dilute 3 mg/kg in 50 ml normal saline/5% dextrose (NS/D5) and start at 0.5 ml/hr (0.5 µg/kg/min). Cover the infusion set with foil paper
- Monitor continuous arterial blood pressure. Use syringe infusion pump
- Monitor 6–12 hourly lactate level (indicates rising thiocyanate level
- Use with caution in renal failure. Do not use for more than 5 days. Monitor thiocyanate levels if prolonged infusions are required.
- Adverse effects are rare if used in the prescribed dose for less than 5 days.

Labetalol (Table 57.1)
- Dose: 0.25 mg/kg stat followed by 0.25–3 mg/kg/hr
- Contraindications are asthma, cardiac failure (bradycardia, heart block) and used with caution in suspected pheochromocytoma (interferes with urinary metanephrine/vanillylmandelic acid levels)
- Procedure: Add 100 mg (1 ml = 5 mg) of labetalol to 80 ml of 0.45/0.9 glucose normal saline (GNS) (1 mg/ml), infuse according to weight calculation.

Principles of addition of new drugs
- Start a drug at lower dose; increase up to the maximum dose; add next drug

Table 57.1: Antihypertensive drugs for the management of severe hypertension in children 1–17 years old

<table>
<thead>
<tr>
<th>Drug</th>
<th>Class</th>
<th>Dose</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium nitroprusside</td>
<td>Vasodilator</td>
<td>0.53–10 mg/kg/min IV</td>
<td>Monitor cyanide levels with prolonged (&gt; 72 hours) use or in renal failure; or coadminister with sodium thiosulfate</td>
</tr>
<tr>
<td>Labetalol</td>
<td>α- and β-blocker</td>
<td>Bolus: 0.2–1.0 mg/kg/dose up to maximum 40 mg/dose IV Infusion: 0.25–3.0 mg/kg/h IV</td>
<td>Asthma and overt heart failure are relative contraindications</td>
</tr>
<tr>
<td>Nicardipine</td>
<td>Calcium-channel blocker</td>
<td>1–3 mg/kg/min IV</td>
<td>May cause reflex tachycardia</td>
</tr>
<tr>
<td>Esmolol</td>
<td>β-blocker</td>
<td>100–500 mg/kg/min IV</td>
<td>Very short-acting; constant infusion is preferred; may cause profound bradycardia</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>Vasodilator</td>
<td>0.2–0.6 mg/kg/dose IV/IM</td>
<td>Should be given every 4 hour when given IV bolus May cause reflex tachycardia, prolonged hypotension</td>
</tr>
</tbody>
</table>
Table 57.2: Blood pressure levels in boys by age and height percentile

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>BP (%)</th>
<th>Systolic BP (mmHg)</th>
<th>Diastolic BP (mmHg)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Height percentile</td>
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</table>
Table 57.3: Blood pressure (BP) levels for girls by age and height percentile

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>BP (%)</th>
<th>Systolic BP (mmHg)</th>
<th>Diastolic BP (mmHg)</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>Height percentile</td>
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</tbody>
</table>
Hypertensive Crisis in the PICU

Further Reading

Hypertensive Crisis Algorithm

Praveen Khilnani

Blood pressure greater than 99th percentile for age and sex

Therapeutic limb

Admit to PICU and maintain airway, breathing and circulation

Establish vascular access and insert arterial line

Start nitroprusside drip @ 0.3–0.5 μg/kg/min

Monitor blood pressure and aim 20–25% reduction in 15 minutes to 2 hours

Too rapid fall
- Decrease dose of SNP
- Administer fluid

Too slow fall
- Increase dose of SNP to a maximum of 5 μg/kg/min
- Consider adding another antihypertensive agent

Other antihypertensive agents
- Labetalol: 0.25 mg/kg bolus, then 0.25 mg/kg/hr
- Esmolol: 300–500 μg/kg bolus, then 25–200 μg/kg/min
- Nicardipine: 5–10 μg/kg bolus, then 1–3 μg/kg/min

Diagnostic limb

Take history of ingestion of drugs
- Cocaine
- Amphetamines
- Phenycyclidine
- Tacrolimus

Measure four extremity blood pressure

Blood pressure in legs

Echocardiography to rule out coarctation of aorta

Order serum chemistry, electrolytes, urinalysis for VMA, T3, T4, TSH, catecholamines, full blood count, plasma renin and aldosterone activity, fundus examination, pregnancy test

High BUN, creatinine and potassium

Consider renal parenchymal disorders
- Acute glomerulonephritis
- HUS, SLE, vasculitides

Increased plasma renin activity

Consider renovascular disorders
- Renal artery stenosis
- Fibromuscular dysplasia, PAN
- Renin secreting tumors

VMA

Consider phaeochromocytoma

All investigations normal

Essential hypertension

Increased plasma aldosterone activity and low potassium

Consider
- Conn’s disease
- Cushing’s syndrome

T3 and T4 high

Consider thyroid crisis

Pregnancy test positive

Pre-eclampsia

Abbreviations: PICU, pediatric intensive care unit; SNP, sodium nitroprusside; VMA, vanillylmandelic acid; BUN, blood urea nitrogen; HUS, hemolytic uremic syndrome; SLE, systemic lupus erythematosus; PAN, polyarteritis nodosa
Management of the Postoperative Pediatric Cardiac Surgical Patient

VSV Prasad, Anjul Dayal

The management of the child undergoing surgical correction of congenital cardiac defects has undergone vast refinement over the last two decades. Previously considered “very high risk” and “inoperable” congenital malformations are now routinely corrected with increasing successful results in major centers all over the world. Hitherto considered the domain of advanced countries, the surgical management as well as the postoperative care of these children in India has undergone paradigm changes. Many centers in the major metros across India now operate on children, infants and neonates, regularly with the excellent outcomes. Our understanding of surgical techniques, anesthesia and cardiac perfusion technology coupled with refinements in postoperative mechanical ventilation, cardiovascular and hemodynamic manipulation and monitoring has greatly improved. The general principles of management of the sick child just as in the regular pediatric intensive care unit (PICU) apply, though with different management protocols and goals. Application of these to the child who has returned from the cardiac operating room. The goals of postoperative cardiac management of the child are to optimize cardiopulmonary support through external monitoring and internal monitoring, and to prevent secondary injury to the myocardium and other organs during the period of recovery and healing.

Optimization of cardiac output, oxygenation and ventilation and perfusion are the key areas of focus and concern, as the repaired internal structures of the heart and myocardium adapt to new mechanical and functional stresses after surgery. Special emphasis is placed on close invasive hemodynamic monitoring, fluid management (tightly regulated as compared to care in the general PICU), and renal perfusion and protection. In the care of simpler defects of the heart, the risk of postoperative arrhythmias is of lesser concern. In general, early extubation and rapid weaning of inotropic and vasodilator support is executed as the myocardium recovers from the ischemia-reperfusion injury during cardiopulmonary bypass.
Every center that performs pediatric cardiac surgery would have its own unit-specific guidelines and protocols for postoperative care, but the following are general guidelines:

**On arrival in the PICU from the operating room:** Obtain a detailed version of the intraoperative course from the anesthesiologist, cardiac perfusionist and the team of surgeons, including the actual anatomy of the heart, the nature of lesion, nature of surgery, problems during surgery or post-cardiopulmonary bypass, the duration of bypass, cross clamp time, circulatory arrest (if any) and any induced hypothermia and rewarming; coagulation and bleeding status and hemodynamic status including fluid volumes infused, blood and blood products infused before transfer from the operating room. Examine and assess stability of the child on arrival and then every few minutes for the first hour.

The algorithm for managing the postoperative child is as follows:

**Airway**
- Note the size of endotracheal (ET) tube, its length at the point of entry (nose or mouth).

**Ventilation**
- Ensure that FiO₂, peak inspiratory pressure (PIP) and tidal volume are optimal.
- The initial settings for ventilation will depend on the ventilation that has been required in the operating room and the settings should be modified appropriately.

**Duration**
- After prolonged bypass (>120 min) ventilate patient overnight. Myocardial function is usually worst 9–12 hours post-cardiopulmonary bypass.

Weaning from ventilation should commence when:
- Patient is hemodynamically stable and will predictably remain so.
- No excessive drain loss or no other active process is likely to interfere with weaning.
- Stable arterial blood gases on FiO₂ <0.45, positive-end expiratory pressure (PEEP) = 5, pressure support = 5.
- Adequate analgesia/sedation.

**Table 60.1** provides a guide to the usual times from the start of weaning to a rate of 5 (able to routinely extubate from a rate of 5 unless specifically worried) or to continuous positive airway pressure (CPAP) + pressure support (PS).

**Table 60.1:** Time of weaning ventilation

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>ASD</td>
<td>2–4 hours</td>
</tr>
<tr>
<td>Fontan</td>
<td>4–6 hours</td>
</tr>
<tr>
<td>BT shunt</td>
<td>12–24 hours</td>
</tr>
<tr>
<td>Arterial switch</td>
<td>12–24 hours</td>
</tr>
<tr>
<td>PA banding</td>
<td>12–24 hours</td>
</tr>
</tbody>
</table>

**Abbreviations:** ASD, Atrial septal defects; BT, Blalock-Taussig; PA, Pulmonary artery
Circulation
Assess: Heart rate/rhythm/mode of pacing/blood pressure/filling pressures and PA pressure.

Target arterial and filling pressures will need to be individualized based on patient, lesion and repair. The anesthesiologist may handover the filling pressures required to establish an independent circulation coming off cardiopulmonary bypass. These are good starting points for the first postoperative minutes to hours.

Assessment of Cardiac Output
- Cardiac output is difficult to measure in children. Blood pressure is not a reliable indicator of cardiac output. Thus, patients who are vasoconstricted can be normotensive or hypertensive and yet have a low cardiac output.
- For clinical purposes indirect assessment of cardiac output must suffice. Table 60.2 lists the main elements which are integrated to reach a clinical conclusion.

Clinical Assessment
Table 60.2 is a guide to clinical assessment of cardiac output.

Causes of Impaired Cardiac Performance
The major causes of impaired cardiac performance after open heart surgery are:
- Hypovolemia
- Myocardial dysfunction, usually secondary to postoperative ischemia (X clamping)
- Residual lesions, e.g. ventricular septal defect (VSD), obstructive lesions
- Increased afterload, including pulmonary hypertension
- Abnormalities of heart rate and rhythm
- Subendocardial perfusion

Table 60.2: Signs to detect low cardiac output

<table>
<thead>
<tr>
<th></th>
<th>Low cardiac output</th>
<th>Adequate cardiac output</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peripheral Perfusion</td>
<td>Poor capillary refill</td>
<td>Good capillary refill (&lt;3 secs)</td>
</tr>
<tr>
<td>Core-Peripheral Temp. Gradient</td>
<td>&gt;3° C</td>
<td>&lt;3° C</td>
</tr>
<tr>
<td>Pulses</td>
<td>Impalpable or weak peripheral pulses</td>
<td>Full peripheral pulses</td>
</tr>
<tr>
<td>Urine Output</td>
<td>&lt;1 ml/kg/hour</td>
<td>&gt;1 mL/kg/hour</td>
</tr>
<tr>
<td>Mental Status</td>
<td>Combative, disorientated</td>
<td>Cooperative</td>
</tr>
<tr>
<td>Arterial Waveform</td>
<td>Small area under curve</td>
<td>Large area under curve</td>
</tr>
<tr>
<td></td>
<td>Dicrotic notch soon after peak</td>
<td>Dicrotic notch occurs later</td>
</tr>
<tr>
<td>Metabolic Acidosis</td>
<td>Base excess ≥5 mmol/l</td>
<td>Base excess ≤5 mmol/l</td>
</tr>
<tr>
<td>Lactate</td>
<td>&gt;4 mmol/l</td>
<td>&lt;2 mmol/l</td>
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</tbody>
</table>
Postoperative Intravenous Fluid Orders

Salt and water overload and retention are invariable in association with cardiopulmonary bypass and cardiac surgery (open and closed). Fluid restriction therefore is required postoperatively.

Standard Fluids Post Bypass

Total fluids: 2 mL/kg/hour (3–6 kg), 1 ml/kg/hour (7–40 kg), 40 ml/hour (>40 kg) for 36–48 hours, then based on clinical state of the child.
**Standard Maintenance Fluid**

Five percent glucose with 0.45%: If they have a higher glucose requirement, 10% glucose should be substituted initially.
- During the initial rewarming phase, additional volume expanders may be necessary to support the circulation.
- Postoperative fluid intake can be gradually liberalized on a daily basis, say 1 ml/kg/hour up to full maintenance provided clinical circumstances permit. Children who remain ventilated may require ongoing fluid restriction.

**Drains**

Observe chest drainage frequently and record the amount regularly half-hourly for 6 hours and hourly thereafter provided drainage minimal and decreasing. If losses are more than 10 ml/kg/hour at any time, the patient would require immediate surgical review.

**Sedation/Analgesia**

Usually opioid analgesics are used (Morphine or Fentanyl). If early extubation is not planned, an infusion of midazolam is also appropriate in addition to the opioid.

**Muscle Relaxants**

They are usually unnecessary in the presence of adequate sedation (morphine plus midazolam) except for shunts and PA bands. They may be necessary to facilitate hyperventilation and also when cooling is being used, to prevent shivering.

**Routine Tests**

Chest X-ray (CXR), blood gases (including lactate and mixed venous O$_2$ saturation (SvO$_2$) from RA or PA line), Hb, platelets, PT, aPTT, fibrinogen, electrolytes including magnesium, PO$_4$ and ionized calcium, creatinine are measured immediately on return from theater, 12-lead ECG and atrial electrogram.

*Note:* The repetition of these tests will depend on the condition of the child and the institution policy.

**Postoperative Problems**

The common postoperative problems are as mentioned below:

**Low Cardiac Output**

The flow diagram on the following page provides some guidance for an approach; however this needs to be individualized for each patient, depending on anatomy and physiology.
Abbreviations: ECHO, echocardiography; ECG, electrocardiogram; CVP, central venous pressure; LAP, left atrial pressure; LCOS, low cardiac output syndrome; peritoneal dialysis; PHT, pulmonary hypertension; PEEP, positive end expiratory pressure.
Management of the Postoperative Pediatric Cardiac Surgical Patient

**Pulmonary Hypertension**

**Treatment**
- Heavy sedation and analgesia
- Ventilation: modest hyperventilation and hyperoxygenation
- Cardiovascular support: inotropes as required. Consider dobutamine
- Suctioning: preoxygenate ($F_{O_2}$ 1.0) on ventilator for 5 minutes, consider lidocaine instillation in trachea to anesthetize the trachea to prevent pulmonary hypertensive crisis.

<table>
<thead>
<tr>
<th>Action</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increase $F_{O_2}$</td>
<td>$P_{O_2} &gt; 150$ mmHg</td>
</tr>
<tr>
<td>Sedation and paralysis</td>
<td>Decreases oxygen consumption</td>
</tr>
<tr>
<td>Hyperventilate on ventilator</td>
<td>$pH &gt; 7.50$, $PaCO_2 = 25–35$ mmHg</td>
</tr>
<tr>
<td>Inhaled nitric oxide</td>
<td>1–40 ppm (start at 10 ppm)</td>
</tr>
<tr>
<td>Inotropes/vasopressors</td>
<td>Maintain systemic arterial pressure in desired range</td>
</tr>
<tr>
<td></td>
<td>Consider dobutamine $5–15$ mcg/kg/min</td>
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<tr>
<td>Add/increase nitrovasodilator</td>
<td>Nitroglycerin (GTN) 1–10 mcg/kg/min</td>
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<td></td>
<td>Consider prostacyclin 5–25 nanogram/kg/min</td>
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</tbody>
</table>

**Renal and Electrolyte Problems**

**Renal Dysfunction**
- Aim for minimum 0.5–1.0 ml/kg/hour urine output.

Renal dysfunction is relatively common following cardiac surgery due to the following causes and corrective steps should include:
- Catheter blockage/leak: flush/change catheter
- Hypovolemia: give volume
- Low cardiac output: commence/increase support (usually inotropes).

If oliguria persists despite above measures, then follow:
- Give furosemide: 1 mg/kg
- If no response give high dose furosemide: 5 mg/kg
- Furosemide infusions may be useful once urine flow is re-established.

**Renal Replacement Therapy**
- Peritoneal dialysis
- CVVH: continuous veno-venous hemofiltration.

**Capillary Leak Syndrome**

**Features**
- Unstable circulation with falling systemic pressure/low diastolic BP
- Usually high-inotrope requirements/low-filling pressures
- Increased colloid requirement to keep filling pressures high.
Management

There is no specific treatment for capillary leak. Management is aimed at supporting compromised organ systems.

- Keep the filling pressures as low as is compatible with good cardiac output.
- Ventilation: Pulmonary compliance deteriorates as interstitial edema and pleural effusions accumulate. Thus higher ventilatory pressures are needed and increased levels of PEEP may be needed to improve oxygenation.
- Optimize hemodynamics.
- Some evidence that maintaining a high-normal hematocrit may help.
- Consider peritoneal drainage of ascitic fluid ± peritoneal dialysis if renal replacement therapy is indicated.

Common Arrhythmias seen in the PICU

Narrow Complex Tachyarrhythmia

May reflect global disturbances: hypoxia, pyrexia, electrolyte imbalances

- Sinus tachycardia
- Supraventricular tachycardia (SVT)
- Junctional ectopic tachycardia (JET).

Relatively common following open-heart surgery ECG appearance:

- Rate is usually 160–260 beats/minute
- QRS complex usually narrow but may be wide if there is associated incomplete or complete bundle branch block but must be same QRS as in sinus rhythm
- Atrial ECG is usually confirmatory.

Treatment Strategies for JET

- Cooling: Induction of moderate hypothermia (33–35°C) with cooling blanket
- Magnesium: Intravenous magnesium sulfate (30–50 mg/kg)
- Anti-arrhythmic drugs: Procainamide (Type Ia)/Amiodarone (Type III)/Propafenone (Type 1c)/Esmolol/Sotalol
- External cardiac pacing
- Mechanical support
- Mechanical support such as left ventricular assist device (LVAD) or ECMO may be effective as rescue therapy in severe, intractable JET.

Bradyarrhythmia

AV Block

- Surgical injury
- Peri-bundle edema
- Optimize oxygenation
  - Pacing – AV sequential
If no pacing wires, consider:
- Isoprenaline
- Transthoracic pacing until wire available
- Transvenous pacing (discuss with cardiologist).

Ventricular Ectopic Beats
- Exclude and correct remediable factors: K, Ca, Mg disturbance, acid base disturbance and hypotension.
- Consider overdrive pacing/lignocaine bolus 1 mg/kg IV slowly and followed by infusion (15–40 mcg/kg/min).

Common Postoperative Cardiac Scenarios with Common Lesions

**ASD repair**  
Postoperative problems are uncommon after ASD repair

**VSD Repair**  
Postoperative problems
- Pulmonary hypertension for treatment vide supra
- Arrhythmias: heart block or SVT including JET (consider medications/pacing)
- Pulmonary edema

**TOF (Total repair)**  
Postoperative problems
- Reduced cardiac output: occurs by 12 hours post operatively usually due to:
  - Arrhythmias especially JET
  - Poor RV performance: keep high CVP, avoid high ventilator pressures, early extubation
  - Effusions: use pericardial, peritoneal, pleural drains

Usual postoperative plan: no inotropic support, analgesia ± sedation, early weaning from ventilator (2–3 hours).

Postoperative plan:
- Small VSD: no inotropes, wean early from intermittent positive pressure ventilation (IPPV)
- High pulmonary blood flow known PHT: needs inotropes and vasodilators; wean carefully from ventilator
- Multiple VSD: may require palliative PA band to reduce pulmonary flow

Postoperative plan:
- Low dose inotropes
- Often require SNP for BP
- Keep sedated and paralyzed till circulation stabilizes
- Ascites: drain via PD catheter

Successful pediatric critical care for the child, undergoing surgical correction of congenital heart defects, depends on:
- Accurate anatomic preoperative diagnosis by the pediatric cardiologist.
- Liaison and discussion with the team of cardiac surgeons to decide on optimal timing for correction, approach and type of corrective repair.
- Meticulous intraoperative care with particular reference to myocardial preservation, maintenance of adequate perfusion of the vital organs.
- A good team effort on the part of the pediatric intensivists, nurses and ancillary support staff in the PICU.
- Excellent back-up and multispecialty support.
Further Reading

Lesion Specific Management of Pediatric Cardiac Patients

Vikas Taneja

**Approach to Acyanotic Heart Defects**

**Acyanotic heart defects**

- **Increased PBF**
  - LVH or CVH
    - Ventricular septal defect
    - Patent ductus arteriosus
    - Endocardial cushion defects
  - RVH
    - Atrial septal defect
    - Partial anomalous pulmonary venous return
    - Pulmonary vascular obstructive disease

- **Normal PBF**
  - LVH
    - Aortic stenosis
    - Coarctation of aorta
    - Mitral regurgitation
  - RVH
    - Pulmonary stenosis
    - Coarctation of aorta in infants
    - Mitral stenosis

*Abbreviations: PBF, pulmonary blood flow; CVH, combined ventricular hypertrophy; RVH, right ventricular hypertrophy; LVH, left ventricular hypertrophy*

**Approach to Cyanotic Heart Defects**

**Cyanotic heart defects**

- **Increased PBF**
  - LVH or CVH
    - Truncus arteriosus
    - Single ventricle
    - Transposition of great arteries with VSD
  - RVH
    - TGA
    - Total anomalous pulmonary venous return
    - Hypoplastic left heart syndrome

- **Normal PBF**
  - CVH
    - TGA with PS
    - Single ventricle with PS
    - Truncus arteriosus with hypoplastic PA
  - LVH
    - Tricuspid atresia
    - Pulmonary atresia with hypoplastic RV
  - RVH
    - Fallot's tetralogy
    - Ebstein's anomaly
    - Pulmonary vascular obstructive disease

*Abbreviations: PBF, pulmonary blood flow; LVH, left ventricular hypertrophy; CVH, combined ventricular hypertrophy; RVH, right ventricular hypertrophy; TGA, transposition of the great arteries; PS, pulmonary stenosis; VSD, ventricular septal defect; PA, pulmonary artery; RV, right ventricle*
**Postoperative Low Cardiac Output**

**Abbreviations**: LV, left ventricle; RV, right ventricle

**Left Ventricular Dysfunction**

**Diagnosis**
- Tachycardia, hypotension, low temperatures, poor perfusion, low SpO₂
- End-organ dysfunction: Low urine output or absent bowel sounds, metabolic acidosis
- CXR: Cardiomegaly with passive congestion of the lungs
- ECHO: Ventricular distension, decreased fractional shortening, increased end-systolic volume, increased LAP.

**Treatment**

1. Increased cardiac output
   - Optimize heart rate
     - Cardiac pacing
   - Optimize preload
     - LAP 8–12 mmHg
   - Evaluate for anatomic problems, repair in OR catheterization laboratory
   - Reduce afterload
     - Mechanical ventilation, milrinone, nitroprusside
   - Augment contractility
     - Calcium, dopamine, dobutamine, epinephrine
   - ECMO, LVAD, IABP

**Abbreviations**: ECMO, extracorporeal membrane oxygenation; LVAD, left ventricular assist devices; IABP, intra-aortic balloon pump; CXR, chest X-ray; LAP, left atrial pressure; OR, operation room
**RV Dysfunction**

### Diagnosis
- Tachycardia, hypotension, low temperatures, poor perfusion, low SpO₂
- Hepatomegaly, raised JVP
- CXR: Cardiomegaly
- ECHO: Decreased RV compliance, decreased fractional shortening
- Increased RA pressures, CVP

### Treatment
1. **Increased cardiac output**
   - Optimize heart rate
     - Cardiac pacing
   - Optimize preload
     - RAP < 15 mmHg
   - Augment contractility
     - Calcium, dopamine, dobutamine, epinephrine
   - Reduce RV afterload
     - Mechanical ventilation, milrinone, NO
   - Evaluate for anatomic problems, repair in OR catheterization laboratory
   - ECMO RVAD

**Abbreviations:** JVP, jugular venous pressure; CXR, chest X-ray; ECHO, echocardiography; CVP, central venous pressure; OR, operation room; ECMO, extracorporeal membrane oxygenation; RVAD, right ventricular arrhythmogenic dysplasia; ECHO, echocardiography
Pulmonary Hypertensive Crises

**Diagnosis**
Severe hypoxemia, decreased cardiac output leading to hypotension and bradycardia

**Physiology**
In infants, less than 3 months, pulmonary vasculature is hyper-reactive. Any precipitating factor leads to severe pulmonary hypertension, increased RV afterload and marked RV dysfunction. Pulmonary hypertensive crisis leads to sudden decrease in the pulmonary blood flow, marked hypoxemia, metabolic acidosis and further reduction in the RV cardiac output.

**Precipitating factors**
Hypoxia, hypercarbia, acidosis, hypothermia, hyperthermia, hypoglycemia, pain and tracheal stimulation due to suctioning

**Treatment**
Decrease RV afterload (treat the precipitating factors)

- **Correct hypoxia**
  - Increased FIO₂
- **Correct hypercarbia**
  - Increased ventilatory rate
  - Increased tidal volume
  - Decreased I-time
- **Decrease RV afterload**
  - Decreased PEEP
  - Decreased mean airway pressure

No improvement

- **Pharmacological management**
  - Milrinone nitric oxide
  - Increased preload (fluids)

No improvement

- **High frequency ventilation**

No improvement

- **ECMO**

**Abbreviations:** RV, right ventricle; ECMO, extracorporeal membrane oxygenation
Arrhythmias

Approach to arrhythmias in the postoperative period is same as treating any arrhythmia and is discussed elsewhere, but certain specific arrhythmias need special mention.

**Bradyarrhythmia**
- Atrial pacing if AV conduction is intact
- Ventricular pacing
- AV sequential pacing when LV function is impaired

**Ectopics**
- Look for and treat electrolyte abnormalities - hypo and hyperkalemia, hypocalcemia, hypomagnesemia, hypoxia, hyperthermia, hypoglycemia and acidosis

**Tachyarrhythmia**
- Junctional ectopic tachycardia
- Narrow QRS complex tachycardia with AV dissociation
- Ventricular rate is higher than the atrial rate
- Onset (warm-up phenomenon) and termination are gradual

**Management**
- Hypothermia (up to 33–35°C)
- Reduction of adrenergic drugs (dopamine, adrenaline)
- Overdrive pacing (atrial pacing at rates higher than the JET rate to restore AV synchrony and cardiac output)
- Mechanical ventilation with adequate sedation, analgesia and paralysis
- Amiodarone: drug of choice
- Digoxin is contraindicated as it decreases AV conduction

*Abbreviations: AV, atrioventricular; LV, left ventricle; JET, junctional ectopic tachycardia*
## Interpretation of Various Invasive Pressures in a Postoperative Cardiac Patient with Low Cardiac Output

Clinical signs: Tachycardia, poor perfusion, hypotension, cold extremities, low urine output

<table>
<thead>
<tr>
<th>ABP</th>
<th>CVP</th>
<th>LAP</th>
<th>PAP</th>
<th>Interpretation</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>Low</td>
<td>Low</td>
<td>Normal</td>
<td>Low intravascular volume</td>
<td>Give fluids</td>
</tr>
<tr>
<td>Low</td>
<td>High</td>
<td>Low</td>
<td>Normal</td>
<td>RV dysfunction</td>
<td>Decrease preload, increase contractility—inotropes and decrease afterload—milrinone, inhaled nitric oxide, optimize HR, ventilatory manipulations—to decrease pulmonary vascular resistance</td>
</tr>
<tr>
<td>Low</td>
<td>High</td>
<td>High</td>
<td>Normal</td>
<td>LV dysfunction</td>
<td>Increase contractility and decrease afterload—inodilator, keep HR towards upper side of normal, electively ventilate, decrease preload—diuretic if blood pressures permit</td>
</tr>
<tr>
<td>Low</td>
<td>High</td>
<td>Low</td>
<td>High</td>
<td>Pulmonary hypertension</td>
<td>Sedate, paralyze, hyperventilate, correct acidosis, hypoxia and hypercarbia, avoid painful stimuli, suctioning</td>
</tr>
</tbody>
</table>
**Algorithm for the Management of Low Cardiac Output after Fontan Procedure**

Clinical signs: Tachycardia, poor perfusion, hypotension, cold extremities, low urine output.

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Measurement</th>
<th>Interpretation</th>
<th>Treatment</th>
</tr>
</thead>
</table>
| 1      | CVP < 15 mmHg, PAP < 15 mmHg, LAP < 5 mmHg | Hypovolemia | Give fluids until:  
• LAP ≥ 8 or  
• CVP-LAP < 7 |
| 2      | CVP-PAP ≥ 2 mmHg | Obstruction of SVC-PA anastomosis, PA clot | Thrombolytic therapy, Interventional catheterization, Reoperation |
| 3      | CVP > 18 mmHg, PAP > 18 mmHg, LAP < 8 mmHg | Pulmonary vasoconstriction, Pulmonary vascular disease, Pulmonary venous obstruction | Maintain PaCO$_2$ at 30–35 mmHg, SpO$_2$ at 100%, pH > 7.5, PEEP 0, Normal glucose, temperature and calcium |
| 4      | CVP > 18 mmHg, PAP > 18 mmHg, LAP > 12 mmHg | Ventricular dysfunction, AV valve regurgitation, Ventricular outflow obstruction | Dopamine and nitroprusside, Milrinone, AV replacement for AV regurgitation, Enlargement of restrictive VSD for subaortic stenosis |

*Abbreviations: CVP, central venous pressure; PAP, pulmonary artery pressure; LAP, left atrial pressure; SVC, superior vena cava; PA, pulmonary artery; AV, atrioventricular; PEEP, positive end-expiratory pressure; VSD, ventricular septal defect*
For all residents and doctors working in a pediatric intensive care unit (PICU), following is a recommended format of general admitting orders from author’s experience.

**General Admitting Orders**

Following is a guideline to be used by residents and fellows for admission and daily PICU rounds with consultant/faculty.

Admitting orders on an unstable patient (shock, mechanical ventilation, encephalitis, etc.) admitted to the PICU includes:

1. Admit to Dr in PICU Level III
2. Diagnosis
3. Condition critical/labile/stable
4. Vital signs per PICU routine on cardiorespiratory monitor heart rate, respirations, blood pressure (NBP at regular cycles or intra-arterial continuous)
5. Pulse oximetry continuous
6. Hourly temperature, urine output
7. Nasogastric tube
8. Foley catheter
9. Investigations: CBC, platelets, PT, PTT, Na, K, Ca $$^+$$, urea, Cr, blood culture, LFTs, ABG now
10. Chest X-ray now and every am
11. Initial ventilator settings: Tidal volume, peak pressure, PEEP, inspiratory time, rate, FiO$_2$
12. Inform the intensivist if:
   - BP > or <
   - CVP > or <
   - pH > or <
   - PO$_2$ > or <
   - PCO$_2$ > or <
13. Paracetamol 15 mg/kg PO/PR for temp > 101 sos Q 4 hourly
14. Arterial line flush 2 ml/hr NS 1:1 heparinized saline
15. Fluids bolus with NS/LR 20 ml/kg as ordered
16. Maintenance fluids D5 (1/2) NS with 20 mEq, kcl/L after voids
17. Sedation midazolam drip at...
18. Muscle relaxants. Vecuronium/pancuronium at...
19. Other medications such as ranitidine/antibiotics/aerosols/vasoactive drips, epinephrine, dopamine, dobutamine, norepinephrine
20. Blood/fresh frozen plasma/platelets
21. Feeds/nil by mouth
22. Consults with other subspecialists.

Information to be presented on daily PICU rounds by registrar or fellow: Systematic approach by problems

- Over all condition critical/improved
- Vital signs
- Respiratory O₂ saturation, lung examination, FiO₂ and ventilatory settings, ABG, chest X-ray, endotracheal tube position
- Assessment and plan
- Cardiovascular CVP, ABP, capillary refill, pressor drip doses. Assessment and plan
- Fluids total intake/output total fluid rate IV + oral
- Hematological HB, WBC, platelets, PT, PTT need for blood, blood products
- Gastrointestinal/nutrition feeds Cal/kg/day enteral or parenteral, abdominal girth bowel sounds, aspirates/residuals
- Stool/vomiting
- Electrolytes Na/K/Cl/HCO₃⁻/Ca²⁺/Mg assessment and plan
- Renal urine output ml/kg/hr Urea/Cr
- Hepatic liver functions, albumin, bilirubin, liver enzymes, amylase
- Infectious disease fever spikes, white counts, culture results blood, CSF, urine, tracheal aspirate. Antibiotics days of therapy
- Neurological alertness/pupils size and reaction/response/GCS
- EEG/last seizure/CT/MRI. Shunt related/ventriculostomy issues
- Neurosurgical issues
- Physiotherapy issues
- Social family visited, involved. Condition and medical care plan explained
- Overall plan of the day.

If you do not document, you did not see the patient. So, only defense is a well written case sheet.

Following documentation should be clear and concise

- Admission notes
- Progress notes
- Prescriptions
- Procedure notes
- Transfer notes
- Discharge summaries.

**GENERAL GUIDELINES**
- Note heading
- Date and time
- Legible
- No erasures/white out
- Strike through mistakes and write “error”
- Sign and print name, with designation
- Multi page notes—sign; rewrite date and time.

**Admission Notes**
- Clear and complete admission note essential
- Need not be long
- Preprinted format acceptable
- Document presenting complaint, history, with review of systems, allergies (even if absent), past history, family history
- Physical examination
- Investigations
- Assessment
- Plan.

**Progress Notes**
- Date and time
- Title
- Subjective, objective, assessment, and plan (SOAP) format
  - Subjective—what the patient says/expresses. May not be practical in ventilated or comatose pediatric patient. Pain score can still be performed. Mother can describe some of the problems
  - Objective—what you find on examination, including vital signs and lab results
  - Assessment
  - Plan
- Signature, printed name and designation
- Plan can be problem oriented for complicated patients.

**SOAP Note—Subjective**
- Why is the patient here? Describe problems concisely. Include only information pertinent to his complaints
- State what the patient says
- If a new patient, document past medical history, medications, allergies, family history and important social history.
PICU Rounds and Documentation

**SOAP note—Objective**
- What do you see? This is the physical examination, which should be focused on the systems related to the patient’s complaints
- Vital signs are mandatory
- Include maximum temperature in past 24 hours
- Write the examination in order, from head to toe, even if you do not perform the examination that way
- What labs or tests have been performed—list these and the results after the physical examination
- Do not list investigations that will be done later—they go under the plan.

**SOAP Note—Assessment**
- What do you think is going on
- List the diagnosis, if you are sure or possible diagnoses, if you are not
- Imagine you would not be there tomorrow and someone else has to figure out why you chose the tests or treatment that you did.

**SOAP Note—Plan**
- What are you going to do?
- What tests are planned and when?
- What medicines are being started?

**Problem Oriented Medical Record Format**
An alternate equally acceptable note can be problem oriented medical record (POMR).

**Problem by System**
In each system heading relevant problem and condition, investigations need to be documented; any clinical change since the last note should be documented, as well as consultant recommendations. Affected system can be detailed. Other unaffected systems can be brief, but all systems heads should be mentioned to be complete.

**Example**

**Note Heading**
Date time

**General Condition**
Alert/playful/remains comatose/critical/remains ventilated/somewhat improving/stable any rashes/petechiae.
Vital signs like temperature, heart rate, blood pressure, respiratory rate, pulse, oximeter saturation, capillary perfusion.

**Problem by System**

**Respiratory (Resp)**
Distress, spontaneous respiratory effort, FiO₂, ventilator settings, oxygen saturations, lung findings on examination, blood gases, end tidal CO₂, chest X-ray, respiratory medications/nebulization/respiratory therapy, any clinical change, any consultation.

**Assessment and Plan**

**Cardiovascular**
S1 S2 normal, no murmur/gallop. Blood pressure, CVP, inotropic support dopamine at…., adrenaline at….., other cardiovascular medications, echocardiogram, ECG finding, any clinical change, any consultation.

**Assessment and Plan**

**Gastrointestinal**
Hepatosplenomegaly, jaundice, ascites, diarrhea/constipation, stool number, frequency, consistency, GI bleeding, vomiting, nasogastric drainage, abdominal distension, tenderness, bowel sounds, liver function tests, GI medications, any clinical change, any consultation.

**Assessment and Plan**

**Fluid/Nutrition**
IV fluids type, rate, TPN, nil by mouth or feedings at ml/hr or second/third hourly, calorie count (Kcal/kg/day), 24-hour intake output, net fluid balance: positive/negative, how much need for diuretics/fluid restriction, related lab values, albumin, triglycerides, any change, any dietary consults.

**Assessment and Plan**

**Hematological (Heme)**
Hemoglobin/hematocrit/platelet count, DIC, D-dimer, fibrin split products, PT, PTT, INR. Any blood, fresh frozen plasma or platelet transfusions given, any clinical bleeding, severe anemia, any change, any consultations.

**Assessment and Plan**

**Infectious Disease**
Fever, WBC count differential
CRP/X-ray, ultrasound, CT/MRI findings as applicable
Blood cultures, last cultures, antimicrobials and monitoring drug levels, change in status, any consultations.

**Assessment and Plan**

**Renal (Renal)**
Urine output, urea, creatinine, blood pressure, serum potassium, acid-base status. Overall fluid balance, medications, any change, consultations, dialysis modality, peritoneal dialysis, hemodialysis, hemofiltration, kidney biopsy if applicable, laboratory results.

**Assessment and Plan**

**Metabolic**
Acid-base status, anion gap if any, lactic acidosis, diabetic ketoacidosis, sodium, potassium, calcium, magnesium, phosphorus, glucose abnormalities, insulin therapy, any change, any consultations.

**Assessment and Plan**

**Neurological**
GCS and mental status, pupils reactive, nonreactive, equal unequal, cranial nerves, motor sensory deficits, last seizure, ICP/ventriculostomy drain as applicable, CT, MRI, EEG findings. Medications including seizure medications.

Medication levels (serum), mannitol, steroids, EMG, VEP, BERA, sedation and muscle relaxation, any significant clinical change, any new consultations.

**Assessment and Plan**

**Social**
Family here, involved, parents counseled problems discussed with family

*Overall assessment*
1
2
3
4

*Overall plan* (after discussion with involved multidisciplinary consultants in complex patients)
1
2
3
4
Important Points to Remember

- Always be truthful
- If you did not examine a system, leave it blank—do not make things up
- Never add to (or delete from) an old note or chart. Make a new, corrected entry—the walls have copying machines!
- Document what you tell the family.

Procedure Notes

- Name of the procedure
- Indication
- Whether informed consent obtained (obtain a written consent)
- Site
- What sedation and analgesia were used
- Actual procedure, including equipment used
- Condition after the procedure
- Complications
- Any confirmatory tests performed
- Include: Intubation, arterial and central lines, chest tubes, LP, bone marrow aspiration
- Write a note even if procedure is unsuccessful.

Transfer Note

- Transfer from ICU to ward or to another hospital
- Especially important if long length of stay
- Brief summary, including:
  - Presentation, treatment, hospital course, relevant investigations, procedures, complications, current medications and labs to be followed up
- Transfer orders
- Write clear and unambiguous transfer orders
- (All previous orders to be discontinued, to avoid confusion)
- When transfer to another hospital accepting physician, patient condition and mode of transfer must be documented.

Discharge Summary

- Others will judge us by the quality of the discharge summary put yourself on the receiving end
- Brief clinical summary, including presentation, relevant investigations, diagnosis, treatment, hospital course, procedures, complications, outcome
- Discharge medications and advice
- Follow-up.
**Summary**

**General recommendations for documentation**
Use complete sentences
Avoid unnecessary detail
Pay attention to spelling and grammar
Avoid wordiness and jargon
“Clouded sensorium with decreased mental status” = confused
“Integumentary apparatus” = skin
“Ambulate” = walk
“Experienced a rapidly fatal outcome” = died

**Appendix**

**Informed consent: Pediatric intensive care unit**

Name age sex M F

Reg. no

I ……..father/mother of above named patient hereby authorize Dr ……. and those/whom/he may designate as associates or assistants to perform following emergency procedures in the pediatric intensive care unit.

Resuscitation
Endotracheal intubation
Initiation of mechanical ventilation
Central line placement
Arterial line placement

*I further consent to administration of intravenous sedation to assist in above mentioned procedures.

*I further consent to administration of such drugs, infusions, plasma or blood products transfusion or any other treatment or procedure deemed necessary in the judgement of medical staff.

*I consent to have my/my wards blood tested for infectious diseases such as hepatitis B, C and, HIV.

*The nature and purpose of the procedures, the necessity thereof, the possible alternative methods of treatment, the risks involved and the possibility of complication in the treatment of my/my wards condition have been fully explained to me and I understand the same.

I certify that I have read and fully understood the above consent, that the explanations therein referred to were made and that all blank statements requiring insertion or completion were filled in and any inapplicable paragraphs stricken before I signed.
Signature of doctor/witness | Name………………………….
---|---
| Relationship to patient………
Date | Time | Signature of patient/person authorized to consent for the patient…………….

**Glossary**

<table>
<thead>
<tr>
<th>Term</th>
<th>Abbreviation</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>NBP</td>
<td>Noninvasive blood pressure</td>
<td></td>
</tr>
<tr>
<td>CBC</td>
<td>Complete blood count</td>
<td></td>
</tr>
<tr>
<td>PT</td>
<td>Prothrombin time</td>
<td></td>
</tr>
<tr>
<td>PTT</td>
<td>Partial thromboplastin time</td>
<td></td>
</tr>
<tr>
<td>LFTs</td>
<td>Liver function tests</td>
<td></td>
</tr>
<tr>
<td>ABG</td>
<td>Arterial blood gas</td>
<td></td>
</tr>
<tr>
<td>PEEP</td>
<td>Positive end-expiratory pressure</td>
<td></td>
</tr>
<tr>
<td>CVP</td>
<td>Central venous pressure</td>
<td></td>
</tr>
<tr>
<td>HB</td>
<td>Hemoglobin</td>
<td></td>
</tr>
<tr>
<td>WBC</td>
<td>White blood cell</td>
<td></td>
</tr>
<tr>
<td>CSF</td>
<td>Cerebrospinal fluid</td>
<td></td>
</tr>
<tr>
<td>EEG</td>
<td>Electroencephalogram</td>
<td></td>
</tr>
<tr>
<td>CT</td>
<td>Computed tomography</td>
<td></td>
</tr>
<tr>
<td>MRI</td>
<td>Magnetic resonance imaging</td>
<td></td>
</tr>
<tr>
<td>TPN</td>
<td>Total parenteral nutrition</td>
<td></td>
</tr>
<tr>
<td>DIC</td>
<td>Disseminated intravascular coagulation</td>
<td></td>
</tr>
<tr>
<td>INR</td>
<td>International normalized ratio</td>
<td></td>
</tr>
<tr>
<td>ICP</td>
<td>Intracranial pressure</td>
<td></td>
</tr>
<tr>
<td>VEP</td>
<td>Visual evoked potential</td>
<td></td>
</tr>
<tr>
<td>BERA</td>
<td>Brainstem evoked response audiometry</td>
<td></td>
</tr>
<tr>
<td>ICU</td>
<td>Intensive care unit</td>
<td></td>
</tr>
<tr>
<td>LP</td>
<td>Lumbar puncture</td>
<td></td>
</tr>
<tr>
<td>CRP</td>
<td>C-reactive protein</td>
<td></td>
</tr>
<tr>
<td>GCS</td>
<td>Glasgow coma scale</td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>Intravenous</td>
<td></td>
</tr>
<tr>
<td>NS</td>
<td>Normal saline</td>
<td></td>
</tr>
</tbody>
</table>
Safe and timely transport saves lives. The decision to transfer is individual and depends on the facilities and logistics that govern local conditions.

**Who Should Be Transported Out?**

Any child that the pediatrician feels
- Cannot be given standard care within the institution or
- Will get enhanced care at another institution within safe transportable distance, or
- Needs specialized care not available at the institution for any condition major or minor, is a candidate for transport.

The benefit of transporting any particular patient, in his current condition, must outweigh the risk inherent in the transport.

Information needed by pediatrician to make this decision:
- Know the status of patient
- The capabilities of patient’s current medical setting
- The capabilities of the transport team
- The capabilities of the receiving institution.

The referring provider should not feel unduly rushed to transfer the patient to the referring hospital until they are satisfied that the child’s status is optimal under the circumstances.

**Classification**

- **Stable:** Normal vitals, no respiratory distress, saturation of peripheral oxygen ($\text{SpO}_2$) > 95% in room air.
- **Critical but stable:** Potential hemodynamic instability, altered mental status (Glasgow Coma Scale (GCS) > 8), mild-moderate respiratory distress, $\text{SpO}_2$ < 95% in room air.
- **Critical and unstable:** Inotropic support, moderate/severe respiratory distress, intubated, GCS < 8.
Some Red Flags:
Any hemodynamically unstable patient that fails to respond to initial fluid resuscitation

Respiratory system:
- Increasing $O_2$ requirement
- Worsening distress or failure to achieve expected response in reasonable time
- Frank or imminent respiratory failure

CNS including trauma:
- Seizure uncontrolled beyond phenobarbitone/phenytoin + valproate
- Glasgow Coma Scale $<9$ definitely, $<12$ may be
- Need for imaging that needs special transport and travel
- Polytrauma
- Serious head injury or moderate head injury with long time in travel

Others:
- Requirement for multiple transfusions
- Difficult to control bleeding
- Diagnostic capabilities limitations

Risks versus Benefits

Vary from center to center.

Benefits
- Referring center cannot manage the patient safely and effectively
- Referral center offers better care and patient can be safely transported
- Transport vehicle well equipped for condition of the patient
- Transport team skilled for condition of the patient.

Risks
- Environment of the transport vehicle is inherently less controlled than that of the referring hospital
- Poorly oxygenated, hemodynamically unstable patient. May deteriorate further in transport.

Mode of Transport
- *Family car*: Not acceptable mode of transport for class 2 and 3.
- *Ground ambulance*: Most common and easy mode available.
- *Helicopter*
- *Fixed-wing aircraft*
- Only one family member should preferably accompany the patient. This helps in medicolegal standing also.

Skills Needed by Physician, Nurse and Technician
- Pediatric advanced life support (PALS)/neonatal advanced life support (NALS)
- Pediatric intensive care unit (PICU) work experience
Transport Protocol

- Intubation
- Intravenous (IV) insertion/intraosseous (IO) infusion
- Chest tube insertion.

Retrieving team should stabilize the child on site before transporting: STAY AND PLAY rather than take away an unstable patient: SCOOP AND RUN.

Counseling the Family

- Reason for transport
- Transparency and honesty (do not have facility, expertise, machine, beds, etc.)
- Check bed availability
- Choice of centers
- Financial implications
- Should not go to two to three different places before reaching final care hospital.

Information provided should be reasonably complete and accurate.

- Referring MD’s name, institution, and phone number
- Patient’s name, age, weight, vital signs
- Brief history and clinical findings
- Any diagnostic or therapeutic interventions performed
- Current clinical status
- Proper communication is essential
  - Doctor to doctor
  - Nurse to nurse
- Ensure that a bed is available
- Medical advice for stabilization may be obtained when you call prior to departure, with estimated time of arrival.

Ambulance Equipment

- Portable cardiorespiratory monitor
- Pulse oximeter
- End-tidal carbon dioxide (ET CO₂) if intubated
- Infusion pumps, portable suction
- Adequate oxygen
- Nebulizer
- Transport incubator
- Ventilator optional
- Intravenous poles, pumps, fixed seat, removable stretcher, storage, pediatric equipment sizes
- Defibrillator.

- Sophistication of ambulances and skill of personnel is variable.
- Make sure you know what you have ordered when using a commercial service.

If air transport:

- Pneumothorax must be drained if already
- Gastrointestinal (GI) distress large bore nasogastric tube in place
Equipment implications
- All drains to be kept open
- Use oxygen.

**Do’s and Don’ts**

**Do’s**
- Do not transfer with an unstable airway
- Stabilize cervical spine
- If in doubt, intubate!
- Secure endotracheal tube (ETT)
- End tidal (ET) CO₂ monitoring with capnograph
- Secure all IV lines well
- Recognize tube dislodgement—ET CO₂ helps
- Keep the patient warm
- Repeated reassessment
- Move patient safely in and out of vehicle
- Do sedate if intubated.

**Don’ts**
- Do not give unrealistic expectation of referred hospital
- Do not sedate for “agitation” (unintubated).

**Documentation of Vital Signs and Physical Examination**

- Ideally, complete copy of hospital chart
- Detailed, legible transfer note
  - History, vital signs, investigations, treatment given, hospital course
  - X-ray films
  - Pending investigations with contact phone number
- Hand over patient with a note on condition during transport
- At hand over, allow one family member to accompany patient in interests of transparency.

**KEY POINTS**
- Specially trained teams should conduct interfacility transport of critically ill children
- Successful transport involves thorough preparation and anticipation of patient decompensation
- Good communication between referring and receiving institutions and the transport team is essential to the process
- Proactive steps in service organization and training can improve safety for staff and patient
- Air transports demand specific logistical and physiological considerations

Better early than late, better late than never. Discretion is the better part of valor.
Further Reading


Introduction

Given below are the definitions of levels of sedation.

- **Conscious sedation**: A drug-induced depression of consciousness during which patients respond purposefully to verbal commands, either alone or accompanied by light tactile stimulation. Reflex withdrawal from a painful stimulus is not considered a purposeful response. No interventions are needed to maintain a patent airway, and spontaneous ventilation is adequate. Cardiovascular function is usually maintained.

- **Deep sedation/analgesia**: A drug-induced depression of consciousness during which patients cannot be easily aroused, but respond purposefully after repeated or painful stimulation. The ability to independently maintain ventilatory function may be impaired. Patient may need help in maintaining a patent airway, and spontaneous ventilation may be inadequate. Cardiovascular function is usually maintained.

- **Anesthesia**: Drug-induced loss of consciousness during which patient cannot be aroused, even by painful stimulation. The ability to independently maintain ventilatory function is often impaired. Patient needs help in maintaining airway and positive pressure ventilation may be needed because of depressed spontaneous ventilation or drug induced depression of neuromuscular function. Cardiovascular function may be impaired.

Main Goals of Sedation

Sedation is given to achieve following goals:

- To attenuate fear and anxiety
- To potentiate analgesia
- To reduce unnecessary recall (amnesia).

Procedures Requiring Sedation

**Noninvasive Procedures**

- Magnetic resonance imaging (MRI)
- Computerized tomographic scan (CT scan)
Other imaging studies
Radiation therapy
Electroencephalography (EEG).

Invasive and Painful Procedures
- Bone marrow aspiration and biopsy
- Lumbar puncture or intrathecal medication administration
- Liver biopsy, renal biopsy or bone biopsy
- Dressing changes and wound or burn debridement
- Endoscopy, bronchoscopy
- Transesophageal echocardiography
- Thoracentesis or chest tube placement
- Paracentesis, pericardiocentesis
- Placement or removal of central line
- Fracture reduction or cast replacement
- Ultrasound-guided aspiration of fluid collection in chest or abdomen.

Preprocedure Fasting and Risk of Aspiration
In order to decrease the possibility of aspiration if airway reflexes are lost, proper nil by mouth (NBM) status must be assured before an elective sedation. Standard accepted American Society of Anesthesiologists (ASA) recommendations given below can be tailored to the individual patient age group as applicable:
- Preterm or newborn: No milk 2 hours prior to sedation
- 1–5 months: No milk or solids 4 hours prior to sedation
- 6–36 months: No milk or solids 6 hours prior to sedation
- > 36 months: No milk or solids 8 hours prior to sedation.

Six to eight ounces of clear liquid may be ingested up to 2 hours before the procedure by patients with normal gastric emptying. Clear liquids include apple juice, pediatric electrolyte containing fluids, water or aerated drinks. Many include breast milk in the category of clear liquids. No pulp containing juices are considered clear liquids.

Sedation Equipment
Following is a list of minimum equipment and medications required for pediatric sedation:
- Oxygen/gas supply
- Suction machine/central suction
- Bag mask ventilation
- Pediatric masks of various sizes: Neonatal, infant, and bigger pediatric
- Laryngeal mask airway: Various pediatric sizes
- C circuit (anesthesia circuit) with 0.5 L, 1 L and 2 L bag
- Laryngoscope straight and curved blades, pediatric and neonatal sizes
- Endotracheal tubes: 3, 3.5, 4.0, 4.5, 5.0, 5.5, 6.0 and 7.0 mm sizes
- Stylets
- Tapes to secure endotracheal tubes
- *Suction catheters*: Pediatric size 6, 8, 10, 12 French (Fr) sizes
- Nasogastric tubes 8, 10, 12 French sizes
- Intravenous syringes, needles and fluids
- 24 and 22 intravenous cannulae
- Medications:
  - Sedatives
    - Midazolam—Most commonly used. Safe in all. Watch for respiratory depression
    - Diazepam—Oil based, painful
    - Thiopental—Good in patient with raised ICP
    - Ketamine—Good in patient with asthma or shock
    - Propofol—Short acting, watch for hypotension, acidosis
    - Morphine—Watch for hypotension, respiratory depression
    - Fentanyl—Watch for chest wall rigidity and respiratory depression
  - Muscle relaxants
    - Succinylcholine
    - Atracurium
    - Vecuronium
  - Reversal agents
    - Neostigmine
    - Atropine
    - Naloxone
    - Flumazenil
  - Resuscitation drugs
    - Adrenaline
    - Atropine
    - Sodium bicarbonate
    - Calcium chloride
- Monitoring equipment (ordinary as well as MRI compatible)
  - Cardiorespiratory monitor
  - Noninvasive blood pressure monitor
  - Pulse oximeter with neonatal and pediatric size probes
  - End tidal CO$_2$ monitor.

**Physician Qualifications**
- Doctors responsible for administration of moderate and deep sedation to children should be trained in pediatric intensive care/anesthesia.
- Doctor should be competent in airway techniques including endotracheal intubation, appropriate monitoring during sedation, response to complications, and use of reversal agents and be trained in at least basic life support. Therefore, sedation must be administered by a doctor qualified to administer such sedation as well as to closely monitor the level
of sedation, airway, hemodynamic parameters such as heart rate, blood pressure, respirations and pulse oximetry saturations and/or end tidal CO₂ measurement as applicable

- Additional personnel: In addition to doctor administering sedation, a qualified individual (resident, nurse or an operating room technician) is responsible for providing uninterrupted monitoring of the patients physiological parameters and assistance in supportive or resuscitative measures
- Procedurist (intensivist, pediatrician, surgeon, endoscopist, bronchoscopist, or pediatric subspecialist performing the procedure on the child) shall not take responsibility for administration and monitoring of sedation.

**Monitoring and Care of Child after Sedation**

- Intensive care unit (ICU) patients will be shifted back to respective ICU.
- Positioning in lateral/postoperative position with oxygen by mask to continue.
- Bedrails must be up to prevent fall.
- Personnel should be ready to handle emesis after sedation.
- Suction oropharyngeal secretions to clear the airway.
- Vital signs every 15 minutes to include pulse, blood pressure respirations oxygen saturation until fully conscious.
- Oral clear liquids may be allowed after fully conscious. Child may be transferred to ward or home once fully alert and keeping liquids down.

**Documentation**

- Presedation assessment
- Consent form
- Sedation note by doctor
- Nursing documentation of vital signs during and after procedure.

**Further Reading**

Admission Criteria

These are general guidelines. Individual cases may require discussion with the pediatric intensivist.

- Pediatric patients who are older than 1 month and younger than 18 years may be admitted to the pediatric intensive care unit (PICU).
- The following will be appropriate admissions to the PICU.
  - All patients requiring mechanical ventilation.
  - Patients with impending respiratory failure:
    - Upper airway obstruction requiring endotracheal intubation.
    - Labile croup (laryngotracheobronchitis) requiring more than three adrenaline nebulizations or oxygen to maintain oxygen saturation above 92%.
    - Lower airway obstruction: Severe asthma requiring aerosol therapy every 20 minutes more than three times or continuously.
    - Patients in respiratory distress with signs of hypoxemia, fatigue, and lethargy due to asthma, bronchiolitis, or foreign body.
    - Alveolar disease: Severe pneumonia, empyema, acute respiratory distress syndrome, cardiogenic pulmonary edema causing respiratory distress, hypoxemia, fatigue and/or lethargy.
    - Unstable airway: Jaw fracture, facial burns, retropharyngeal abscess, severe tonsillar enlargement, or deeply comatose patients, vocal cord paralysis, neuromuscular problems such as Guillain-Barré syndrome with cranial nerve palsy involving poor suck, gag reflexes.
    - All pediatric patients after successful resuscitation.
  - Comatose patients: Glasgow Coma Score < 7 secondary to following:
    - Meningitis, encephalitis
    - Hepatic encephalopathy
    - Cerebral malaria
    - Head injury
    - Poisonings
Admission, Transfer and Discharge Criteria for Pediatric Intensive Care Unit (PICU)

- All types of shock/hemodynamic instability:
  - Septic shock with or without multiorgan failure.
  - Hypovolemic shock with hypotension, deterioration in mental status
  - Bleeding emergencies such as gastrointestinal bleeding, bleeding diathesis, disseminated intravascular coagulation
  - *Cardiogenic shock*: Myocarditis, cardiomyopathy, cardiac dysrhythmias, congenital heart disease
  - *Neurogenic shock*: Spinal cord injury, severe brain injury, severe autonomic nervous system involvement affecting blood pressure (BP), heart rate and respiratory drive.
  - Multiple trauma involving major abdominal organs such as liver, splenic injury, pancreatic injury or bladder/kidney injury, rib fractures with lung contusion, pneumothorax, myocardial contusion, cervical spine injury or suspected active internal bleeding.
  - Cardiac dysrhythmias.
  - Hypertensive emergencies.
  - Severe acid base disorders.
  - Severe electrolyte abnormalities.
  - Acute renal failure: Patients requiring acute hemodialysis, hemofiltration and peritoneal dialysis
  - Postoperative patients:
    - Requiring ventilation.
    - Unstable patients.
    - Postoperative patients after open heart surgery, neurosurgery, thoracic surgery, airway surgery and other patients after major general surgery with potential for respiratory/hemodynamic instability.
  - Patients requiring nitric oxide therapy
  - Malignant hyperpyrexia
  - Acute hepatic failure
  - Near drowning episode
  - Acute anaphylaxis
  - Status epilepticus with inadequate control.
  - All post-transplant patients (as applicable)
  - Diabetic ketoacidosis with pH < 7.2, shock and mental status deterioration.

**Admission Criteria to Level 2 Care (Unventilated)**

- All ward patients requiring close monitoring (more than hourly vital signs) due to potentially unstable conditions.
- Croup (laryngotracheobronchitis) requiring oxygen/and/or adrenaline nebulization.
- Asthma requiring 2 hourly nebulization and oxygen to maintain saturations > 92%.
All patients requiring more than 50% oxygen to maintain oxygen saturations > 92%.
- Closed head injury/skull fracture admitted for observation.
- Patients with episodes of apnea.
- Patients with significant abdominal trauma with suspected renal/splenic/hepatic injury.
- Severe dehydration with mental status change.
- Postoperative patients after major surgery with significant postoperative pain/blood loss/stress.
- Patients recovering from critical illness (Level 3 care), but requiring close monitoring.
- Electrolyte abnormalities such as severe hyperkalemia, hyponatremia, hypernatremia, hypocalcemia.

Criteria for Transfer/Discharge from the PICU (Level 3 or Level 2)

Established guidelines should be met prior to transfer/discharge from the PICU regardless of the diagnosis. Patients who are unable to meet established guidelines need to be reviewed by the pediatric intensivist and nurse incharge before transfer.

Minimum Criteria

- Cardiopulmonary stability as evidenced by physiological parameters within appropriate ranges for age and diagnosis. In accordance with pediatric advanced life support (PALS) guidelines, hemodynamically stable is defined as follows:
  - Level of consciousness appropriate for condition/age not requiring intervention.
  - Adequate peripheral perfusion as evidenced by peripheral pulses, color, capillary refill.
  - Absence of signs of impending respiratory failure (inappropriate respiratory rate for age, increased work of breathing, unmaintainable upper airway, pallor/cyanosis, abnormal arterial blood gases).
  - Blood pressure within acceptable range for age as per PALS guidelines.
    - 50th percentile (median) systolic BP 90 mm Hg + (2 × age in years)
    - 5th percentile (min) systolic BP 70 mm Hg + (2 × age in years).
  - Stable pulse rate and respiratory rate as given in Table 65.1.
- Level of oxygenation greater than 92% on room air or up to 50% of oxygen without excessive use of respiratory muscles (retractions, nasal flaring). Patency of airway without any airway intervention. Stable arterial blood gases pH 7.34–7.35; partial pressure of oxygen in arterial blood (PaO₂) 80–100 mm Hg; partial pressure of carbon dioxide in arterial blood (PaCO₂) 35–45 mm Hg; HCO₃ 23–25 mEq/L.
- Renal and metabolic parameters within normal limit:
Admission, Transfer and Discharge Criteria for Pediatric Intensive Care Unit (PICU)

- Na⁺: 133–145 mEq/L
- K⁺: 3.5–5.5 mEq/L
- Cl⁻: 95–105 mEq/L
- Glucose: 80–120 mg/dl
- Urea < 20, creatinine < 1
- Urine output > 1 cc/kg/hr
- Absence of life-threatening dysrhythmias for more than 24 hours.
- No evidence of ongoing septic process (i.e. high fever > 103°F or hemodynamic instability)
- Significant improvement from condition requiring admission.
- Interfacility transfer requires completed transfer formalities including doctor to doctor communication, nursing report, copies of chart and laboratory investigation reports/X-rays, CT scans, etc.

Table 65.1: Normal vital signs

<table>
<thead>
<tr>
<th>Normal values</th>
<th>Systolic BP/Diastolic BP (mm Hg)</th>
<th>Pulse (Beats/min)</th>
<th>Respiratory rate (Breaths/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neonate 3 kg birth</td>
<td>50–70/25–45</td>
<td>70–170</td>
<td>30–40</td>
</tr>
<tr>
<td>Neonate 96 hours</td>
<td>60–90/20–60</td>
<td>70–170</td>
<td>30–40</td>
</tr>
<tr>
<td>Infant 6 months</td>
<td>87–105/53–66</td>
<td>70–170</td>
<td>30–40</td>
</tr>
<tr>
<td>Toddler 2 years</td>
<td>95–105/53–66</td>
<td>80–130</td>
<td>25–32</td>
</tr>
<tr>
<td>School age 7 years</td>
<td>97–112/57–71</td>
<td>70–110</td>
<td>20–26</td>
</tr>
<tr>
<td>Adolescent 15 years</td>
<td>112–128/66–80</td>
<td>60–100</td>
<td>16–20</td>
</tr>
</tbody>
</table>

Further Reading

Pediatric Advanced Life Support

In contrast to adults, cardiac arrest in infants and children does not usually result from a primary cardiac cause. More often it is the terminal result of progressive respiratory failure or shock, also called an asphyxial arrest. Asphyxia begins with a variable period of systemic hypoxemia, hypercapnea, and acidosis, progresses to bradycardia and hypotension, and culminates with cardiac arrest.

Another mechanism of cardiac arrest, ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT), is the initial cardiac rhythm in approximately 5–15% of pediatric in-hospital and out-of-hospital cardiac arrests; it is reported in up to 27% of pediatric in-hospital arrests at some point during the resuscitation. The incidence of VF/pulseless VT cardiac arrest rises with age. Increasing evidence suggests that sudden unexpected death in young people can be associated with genetic abnormalities in myocyte ion channels resulting in abnormalities in ion flow (see below).

Since 2010 marks the 50th anniversary of the introduction of cardiopulmonary resuscitation (CPR), it seems appropriate to review the progressive improvement in outcome of pediatric resuscitation from cardiac arrest. Survival from in-hospital cardiac arrest in infants and children in the 1980s was around 9%. Approximately 20 years later, that figure had increased to 17%, and by 2006, to 27%. In contrast to those favorable results from in-hospital cardiac arrest, overall survival to discharge from out-of-hospital cardiac arrest in infants and children has not changed substantially in 20 years and remains at about 6% (3% for infants and 9% for children and adolescents).

It is unclear why the improvement in outcome from in-hospital cardiac arrest has occurred, although earlier recognition and management of at-risk patients on general inpatient units and more aggressive implementation of evidence-based resuscitation guidelines may have played a role. Implementation of a formal pediatric medical emergency team (MET) or rapid response team (RRT) as part of an emergency response system for a deteriorating inpatient has been shown...
to significantly decrease the incidence of cardiac and respiratory arrests, as well as hospital mortality rates in some large children’s hospitals. Such teams, often consisting of providers with expertise in assessment and initial management of acutely ill patients (critical-care nurses, respiratory therapists, and critical-care physicians), decreased the number of cardiac and respiratory arrests by as much as 72% and hospital mortality by as much as 35% in institutions where the effect was studied. Although it is possible that most of the impact is due to a decrease in respiratory arrests, this cannot be confirmed by the available published data. Implementation of a pediatric MET/RRT may be beneficial in facilities where children with high risk illnesses are present in general inpatient units (Class IIa, LOE B).

Despite the improved outcome of in-hospital CPR, a majority of children with in-hospital cardiac arrest and an even larger percentage of children with out-of-hospital cardiac arrest do not survive, or they are severely incapacitated if they do. Several studies, discussed later in this document, showed that the presence of family members during resuscitation has helped them deal with the inevitable trauma and grief following the death of a child. Therefore, whenever possible, provide family members with the option of being present during resuscitation of an infant or child (Class I, LOE B).

BLS Considerations During PALS

Pediatric advanced life support (PALS) usually takes place in the setting of an organized response in an advanced healthcare environment. In these circumstances, multiple responders are rapidly mobilized and are capable of simultaneous coordinated action. Resuscitation teams may also have access to invasive patient monitoring that may provide additional information during the performance of basic life support (BLS).

Simultaneous Actions

BLS (whether for a child or adult) is presented as a series of sequential events with the assumption that there is only one responder, but PALS usually takes place in an environment where many rescuers are rapidly mobilized and actions are performed simultaneously. The challenge is to organize the rescuers into an efficient team. Important considerations for the greatest chance of a successful resuscitation from cardiac arrest include the following:

Chest compressions should be immediately started by one rescuer, while a second rescuer prepares to start ventilations with a bag and mask. Ventilation is extremely important in pediatrics because of the large percentage of asphyxial arrests in which best results are obtained by a combination of chest compressions and ventilations. Unfortunately ventilations are sometimes delayed because equipment (bag, mask, oxygen, airway) must be mobilized. Chest compressions require only the hands of a willing rescuer. Therefore, start
CPR with chest compressions immediately, while a second rescuer prepares to provide ventilations (Class I, LOE C).

The effectiveness of PALS is dependent on high-quality CPR, which requires an adequate compression rate (at least 100 compressions/min), an adequate compression depth [at least one third of the AP diameter of the chest or approximately 1 1/2 inches (4 cm) in infants and approximately 2 inches (5 cm) in children], allowing complete recoil of the chest after each compression, minimizing interruptions in compressions, and avoiding excessive ventilation. Reasons for not performing high-quality CPR include rescuer inattention to detail, rescuer fatigue, and long or frequent interruptions to secure the airway, check the heart rhythm, and move the patient. Optimal chest compressions are best delivered with the victim on a firm surface.

While one rescuer performs chest compressions and another performs ventilations, other rescuers should obtain a monitor/defibrillator, establish vascular access, and calculate and prepare the anticipated medications.

Monitored Patients
Many in-hospital patients, especially if they are in an ICU, are monitored and some have an advanced airway and are receiving mechanical ventilation. If the patient has an indwelling arterial catheter, use the waveform as feedback to evaluate hand position and chest compression depth. A minor adjustment of hand position or depth of compression can significantly improve the amplitude of the arterial waveform, reflecting better chest compression-induced stroke volume. The arterial waveform may also be useful in identification of return of spontaneous circulation (ROSC). If the patient’s end-tidal CO₂ (PETCO₂) is being monitored, it can be used to evaluate the quality of chest compressions; it can also provide an indication of ROSC (see below).

Respiratory Failure
Respiratory failure is characterized by inadequate ventilation, insufficient oxygenation, or both. Anticipate respiratory failure if any of the following signs is present:
- An increased respiratory rate, particularly with signs of distress (e.g. increased respiratory effort including nasal flaring, retractions, seesaw breathing, or grunting)
- An inadequate respiratory rate, effort, or chest excursion (e.g. diminished breath sounds or gasping), especially if mental status is depressed
- Cyanosis with abnormal breathing despite supplementary oxygen.

Shock
Shock results from inadequate blood flow and oxygen delivery to meet tissue metabolic demands. The most common type of shock in children is hypovolemic,
including shock due to hemorrhage. Distributive, cardiogenic, and obstructive shock occur less frequently. Shock progresses over a continuum of severity, from a compensated to a decompensated state. Compensatory mechanisms include tachycardia and increased systemic vascular resistance (vasoconstriction) in an effort to maintain cardiac output and perfusion pressure respectively. Decompensation occurs when compensatory mechanisms fail and results in hypotensive shock.

Typical signs of compensated shock include:
- Tachycardia
- Cool and pale distal extremities
- Prolonged (>2 seconds) capillary refill (despite warm ambient temperature)
- Weak peripheral pulses compared with central pulses
- Normal systolic blood pressure.

As compensatory mechanisms fail, signs of inadequate end-organ perfusion develop. In addition to the above, these signs include:
- Depressed mental status
- Decreased urine output
- Metabolic acidosis
- Tachypnea
- Weak central pulses
- Deterioration in color (e.g., mottling, see below)

Decompensated shock is characterized by signs and symptoms consistent with inadequate delivery of oxygen to tissues (pallor, peripheral cyanosis, tachypnea, mottling of the skin, decreased urine output, metabolic acidosis, depressed mental status), weak or absent peripheral pulses, weak central pulses, and hypotension.

Learn to integrate the signs of shock because no single sign confirms the diagnosis. For example:
- Capillary refill time alone is not a good indicator of circulatory volume, but a capillary refill time >2 seconds is a useful indicator of moderate dehydration when combined with decreased urine output, absent tears, dry mucous membranes, and a generally ill appearance. Capillary refill time is influenced by ambient temperature, 25 site, and age and its interpretation can be influenced by lighting.
- Tachycardia is a common sign of shock, but it can also result from other causes, such as pain, anxiety, and fever.
- Pulses are weak in hypovolemic and cardiogenic shock, but may be bounding in anaphylactic, neurogenic, and septic shock.
- Blood pressure may be normal in a child with compensated shock but may decline rapidly when the child decompensates. Like the other signs, hypotension must be interpreted within the context of the entire clinical picture.
There are several sources of data that use large populations to identify the 5th percentile for systolic blood pressure at various ages. For purposes of these guidelines, hypotension is defined as a systolic blood pressure:
- <60 mmHg in term neonates (0 to 28 days)
- <70 mmHg in infants (1 month to 12 months)
- <70 mmHg + (2 × age in years) in children 1 to 10 years
- <90 mmHg in children ≥10 years of age.

Airway

Oropharyngeal and Nasopharyngeal Airways

Oropharyngeal and nasopharyngeal airways help maintain an open airway by displacing the tongue or soft palate from the pharyngeal air passages. Oropharyngeal airways are used in unresponsive victims who do not have a gag reflex. Make sure to select the correct size: an oropharyngeal airway that is too small may push the base of the tongue farther into the airway; one that is too large may obstruct the airway.

Nasopharyngeal airways can be used in children who do have a gag reflex. Pay careful attention to proper diameter and length. A nasopharyngeal airway that is too short may not maintain an open airway, while one that is too long may obstruct it. A small-diameter nasopharyngeal airway may be obstructed easily by secretions. It may therefore require frequent suctioning.

Laryngeal Mask Airway (LMA)

Although several supraglottic devices have been used in children, clinical studies of devices other than the LMA in pediatric patients are limited. When bag-mask ventilation (see “Bag-Mask ventilation,” below) is unsuccessful and when endotracheal intubation is not possible, the LMA is acceptable when used by experienced providers to provide a patent airway and support ventilation (Class IIA, LOE C). LMA insertion is associated with a higher incidence of complications in young children compared with older children and adults.

Oxygen

It is reasonable to ventilate with 100% oxygen during CPR because there is insufficient information on the optimal inspired oxygen concentration (Class IIA, LOE C). Once the circulation is restored, monitor systemic oxygen saturation. It may be reasonable, when the appropriate equipment is available, to titrate oxygen administration to maintain the oxyhemoglobin saturation ≥94%. Provided appropriate equipment is available, once ROSC is achieved, adjust the FiO₂ to the minimum concentration needed to achieve an arterial oxyhemoglobin saturation at least 94%, with the goal of avoiding hyperoxia while ensuring adequate oxygen delivery. Since an arterial oxyhemoglobin saturation of 100% may correspond to a PaO₂ anywhere between 80 and
500 mmHg, in general it is appropriate to wean the FIO₂ when saturation is 100%, provided the oxyhemoglobin saturation can be maintained ≥94% (Class IIb, LOE C). Remember that adequate oxygen delivery requires not only adequate arterial oxyhemoglobin saturation but also adequate hemoglobin concentration and cardiac output.

**Pulse Oximetry**

If the patient has a perfusing rhythm, monitor oxyhemoglobin saturation continuously with a pulse oximeter because clinical recognition of hypoxemia is not reliable. Pulse oximetry may, however, also be unreliable in patients with poor peripheral perfusion, carbon monoxide poisoning, or methemoglobinemia.

**Bag-Mask Ventilation**

Bag-mask ventilation can be as effective, and may be safer, than endotracheal tube ventilation for short periods during out-of-hospital resuscitation. In the prehospital setting it is reasonable to ventilate and oxygenate infants and children with a bag-mask device, especially if transport time is short (Class IIa, LOE B). Bag-mask ventilation requires training and periodic retraining in selecting a correct mask size, maintaining an open airway, providing a tight seal between mask and face, providing ventilation, and assessing effectiveness of ventilation.

**Precautions**

Use only the force and tidal volume needed to just make the chest rise visibly (Class I, LOE C); avoid delivering excessive ventilation during cardiac arrest (Class III, LOE C). Evidence shows that cardiac arrest victims frequently receive excessive ventilation. Excessive ventilation during cardiac arrest increases intrathoracic pressure, which impedes venous return, thus reducing cardiac output and cerebral and coronary blood flow. These effects will reduce the likelihood of ROSC. In addition, excessive ventilation may cause air trapping and barotrauma in patients with small airway obstruction. It also increases the risk of stomach inflation, regurgitation, and aspiration.

If the infant or child is not intubated, pause after 30 chest compressions (1 rescuer) or after 15 chest compressions (2 rescuers) to give 2 ventilations (mouth-to-mouth, mouth-to-mask, or bag-mask). Deliver each breath with an inspiratory time of approximately 1 second. If the infant or child is intubated, ventilate at a rate of about 1 breath every 6 to 8 seconds (8 to 10 times per minute) without interrupting chest compressions (Class I, LOE C). It may be reasonable to do the same if an LMA is in place (Class IIb, LOE C).

In the victim with a perfusing rhythm but absent or inadequate respiratory effort, give 1 breath every 3 to 5 seconds (12 to 20 breaths per minute), using the higher rate for the younger child (Class I, LOE C). One way to achieve that rate with a ventilating bag is to use the mnemonic “squeeze-release-release” at a normal speaking rate.
Two-Person Bag-Mask Ventilation

A two-person ventilation technique may be preferable when personnel are available and may be more effective than ventilation by a single rescuer if the patient has significant airway obstruction, poor lung compliance, or the rescuer has difficulty in creating a tight mask-to-face seal. One rescuer uses both hands to maintain an open airway with a jaw thrust and a tight mask-to-face seal while the other compresses the ventilation bag. Both rescuers should observe the victim's chest to ensure chest rise.

Gastric Inflation

Gastric inflation may interfere with effective ventilation and cause regurgitation, aspiration of stomach contents, and further ventilatory compromise. The risk of gastric inflation can be decreased by:

- Avoiding excessive peak inspiratory pressures by ventilating slowly and giving only enough tidal volume to just achieve visible chest rise.
- Applying cricoid pressure in an unresponsive victim to reduce air entry into the stomach (Class IIa, LOE B). This may require a third rescuer if cricoid pressure cannot be applied by the rescuer who is securing the bag to the face. Avoid excessive cricoid pressure so as not to obstruct the trachea (Class III, LOE B).
- Passing a nasogastric or orogastric tube to relieve gastric inflation, especially if oxygenation and ventilation are compromised. Pass the tube after intubation because a gastric tube interferes with gastroesophageal sphincter function, allowing regurgitation during intubation. If a gastrostomy tube is present, vent it during bag-mask ventilation to allow gastric decompression.

Ventilation with an Endotracheal Tube

Endotracheal intubation in infants and children requires special training because the pediatric airway anatomy differs from that of the adult. The likelihood of successful endotracheal tube placement with minimal complications is related to the length of training, supervised experience in the operating room and in the field, adequate ongoing experience, and use of rapid sequence intubation (RSI).

Rapid Sequence Intubation (RSI)

To facilitate emergency intubation and reduce the incidence of complications, skilled, experienced providers may use sedatives, neuromuscular blocking agents, and other medications to rapidly sedate and neuromuscularly block the pediatric patient.

Use RSI only if you are trained, and have experience using these medications and are proficient in the evaluation and management of the pediatric airway. If you use RSI you must have a secondary plan to manage the airway in the event that you cannot achieve intubation.

Actual body weight, rather than ideal body weight, should be used for some non-resuscitation medications (e.g. succinylcholine).
Cricoid Pressure During Intubation

There is insufficient evidence to recommend routine cricoid pressure application to prevent aspiration during endotracheal intubation in children. Do not continue cricoid pressure if it interferes with ventilation or the speed or ease of intubation (Class III, LOE C).

Cuffed Versus Uncuffed Endotracheal Tubes

Both cuffed and uncuffed endotracheal tubes are acceptable for intubating infants and children (Class IIa, LOE C). In the operating room, cuffed endotracheal tubes are associated with a higher likelihood of correct selection of tube size, thus achieving a lower reintubation rate with no increased risk of perioperative complications. In intensive care settings the risk of complications in infants and in children is no greater with cuffed tubes than with noncuffed tubes. Cuffed endotracheal tubes may decrease the risk of aspiration. If cuffed endotracheal tubes are used, cuff inflating pressure should be monitored and limited according to manufacturer’s instruction (usually less than 20 to 25 cm H₂O).

In certain circumstances (e.g. poor lung compliance, high airway resistance, or a large glottic air leak) a cuffed endotracheal tube may be preferable to an uncuffed tube, provided that attention is paid to endotracheal tube size, position, and cuff inflation pressure (Class IIa, LOE B).

Endotracheal Tube Size

Length-based resuscitation tapes are helpful and more accurate than age-based formula estimates of endotracheal tube size for children up to approximately 35 kg, even for children with short stature.

In preparation for intubation with either a cuffed or an uncuffed endotracheal tube, confirm that tubes with an internal diameter (ID) 0.5 mm smaller and 0.5 mm larger than the estimated size are available. During intubation, if the endotracheal tube meets resistance, place a tube 0.5 mm smaller instead. Following intubation, if there is a large glottic air leak that interferes with oxygenation or ventilation, consider replacing the tube with one that is 0.5 mm larger, or place a cuffed tube of the same size if an uncuffed tube was used originally. Note that replacement of a functional endotracheal tube is associated with risk; the procedure should be undertaken in an appropriate setting by experienced personnel.

If an uncuffed endotracheal tube is used for emergency intubation, it is reasonable to select a 3.5 mm ID tube for infants up to one year of age and a 4.0 mm ID tube for patients between 1 and 2 years of age. After age 2, uncuffed endotracheal tube size can be estimated by the following formula:

If a cuffed tube is used for emergency intubation of an infant less than 1 year of age, it is reasonable to select a 3.0 mm ID tube. For children between 1 and 2 years of age, it is reasonable to use a cuffed endotracheal tube with an internal
Verification of Endotracheal Tube Placement

There is a risk of endotracheal tube misplacement (i.e. in the esophagus, the pharynx above the vocal cords, or a mainstem bronchus) and an ongoing risk of displacement or obstruction, especially during patient transport. Since no single confirmation technique, including clinical signs or the presence of water vapor in the tube, is completely reliable, use both clinical assessment and confirmatory devices to verify proper tube placement immediately after intubation, again after securing the endotracheal tube, during transport, and each time the patient is moved (e.g. from gurney to bed) (Class I, LOE B).

The following are methods for confirming correct position:

- Look for bilateral chest movement and listen for equal breath sounds over both lung fields, especially over the axillae.
- Listen for gastric insufflation sounds over the stomach. They should not be present if the tube is in the trachea.
- Check for exhaled CO₂ (see “Exhaled or End-Tidal CO₂ Monitoring,” below).
- If there is a perfusing rhythm, check oxyhemoglobin saturation with a pulse oximeter. Remember that following hyperoxygenation, the oxyhemoglobin saturation detected by pulse oximetry may not decline for as long as 3 minutes even without effective ventilation.
- If you are still uncertain, perform direct laryngoscopy and visualize the endotracheal tube to confirm that it lies between the vocal cords.
- In hospital settings, perform a chest X-ray to verify that the tube is not in a bronchus and to identify proper position in the midtrachea.

After intubation, secure the tube; there is insufficient evidence to recommend any single method. After securing the tube, maintain the patient’s head in a neutral position; neck flexion may push the tube farther into the airway, and extension may pull the tube out of the airway.

If an intubated patient’s condition deteriorates, consider the following possibilities (mnemonic DOPE):

- Displacement of the tube
- Obstruction of the tube
- Pneumothorax
- Equipment failure.

Exhaled or End-Tidal CO₂ Monitoring

When available, exhaled CO₂ detection (capnography or colorimetry) is recommended as confirmation of tracheal tube position for neonates, infants, and children with a perfusing cardiac rhythm in all settings [e.g. prehospital, emergency department (ED), ICU, ward, operating room] (Class I, LOE C) and during intrahospital or interhospital transport (Class IIb, LOE C). Remember that a
color change or the presence of a capnography waveform confirms tube position in the airway but does not rule out right mainstem bronchus intubation. During cardiac arrest, if exhaled CO₂ is not detected, confirm tube position with direct laryngoscopy (Class IIa, LOE C) because the absence of CO₂ may reflect very low pulmonary blood flow rather than tube misplacement.

Confirmation of endotracheal tube position by colorimetric end-tidal CO₂ detector may be altered by the following:

- If the detector is contaminated with gastric contents or acidic drugs (e.g. endotracheally administered epinephrine), a consistent color rather than a breath-to-breath color change may be seen.
- An intravenous (IV) bolus of epinephrine may transiently reduce pulmonary blood flow and exhaled CO₂ below the limits of detection.
- Severe airway obstruction (e.g. status asthmaticus) and pulmonary edema may impair CO₂ elimination below the limits of detection.
- A large glottic air leak may reduce exhaled tidal volume through the tube and dilute CO₂ concentration.

**Esophageal Detector Device (EDD)**

If capnography is not available, an esophageal detector device (EDD) may be considered to confirm endotracheal tube placement in children weighing >20 kg with a perfusing rhythm (Class IIb, LOE B), but the data are insufficient to make a recommendation for or against its use in children during cardiac arrest.

**Transtracheal Catheter Oxygenation and Ventilation**

Transtracheal catheter oxygenation and ventilation may be considered for patients with severe airway obstruction above the level of the cricoid cartilage if standard methods to manage the airway are unsuccessful. Note that transtracheal ventilation primarily supports oxygenation as tidal volumes are usually too small to effectively remove carbon dioxide. This technique is intended for temporary use while a more effective airway is obtained. Attempt this procedure only after proper training and with appropriate equipment (Class IIb, LOE C).

**Suction Devices**

A properly sized suction device with an adjustable suction regulator should be available. Do not insert the suction catheter beyond the end of the endotracheal tube to avoid injuring the mucosa. Use a maximum suction force of -80 to -120 mmHg for suctioning the airway via an endotracheal tube. Higher suction pressures applied through large-bore noncollapsible suction tubing and semirigid pharyngeal tips are used to suction the mouth and pharynx.

**CPR Guidelines for Newborns With Cardiac Arrest of Cardiac Origin**

Recommendations for infants differ from those for the newly born (i.e. in the delivery room and during the first hours after birth) and newborns (during
their initial hospitalization and in the NICU). The compression-to-ventilation ratio differs (newly born and newborns – 3:1; infant two rescuer - 15:2) and how to provide ventilations in the presence of an advanced airway differs (newly born and newborns – pause after 3 compressions; infants – no pauses for ventilations). This presents a dilemma for healthcare providers who may also care for newborns outside the NICU. Because there are no definitive scientific data to help resolve this dilemma, for ease of training we recommend that newborns (intubated or not) who require CPR in the newborn nursery or NICU receive CPR using the same technique as for the newly born in the delivery room (i.e. 3:1 compression-to-ventilation ratio with a pause for ventilation). Newborns who require CPR in other settings [e.g. prehospital, ED, pediatric intensive care unit (PICU), etc.], should receive CPR according to infant guidelines: 2 rescuers provide continuous chest compressions with asynchronous ventilations if an advanced airway is in place and a 15:2 ventilation-to-compression ratio if no advanced airway is in place (Class IIb, LOE C). It is reasonable to resuscitate newborns with a primary cardiac etiology of arrest, regardless of location, according to infant guidelines, with emphasis on chest compressions (Class IIa, LOE C). For further information, please refer to Part 13, “Pediatric Basic Life Support,” and Part 15, “Neonatal Resuscitation.”

**Extracorporeal Life Support (ECLS)**

Extracorporeal life support (ECLS) is a modified form of cardiopulmonary bypass used to provide prolonged delivery of oxygen to tissues. Consider early activation of ECLS for a cardiac arrest that occurs in a highly supervised environment, such as an ICU, with the clinical protocols in place and the expertise and equipment available to initiate it rapidly. ECLS should be considered only for children in cardiac arrest refractory to standard resuscitation attempts, with a potentially reversible cause of arrest (Class IIa, LOE C). When ECLS is employed during cardiac arrest, outcome for children with underlying cardiac disease is better than the outcome for children with noncardiac disease. With underlying cardiac disease, long-term survival when ECLS is initiated in a critical-care setting has been reported even after >50 minutes of standard CPR.

**Monitoring**

**Electrocardiography**

Monitor cardiac rhythm as soon as possible so both normal and abnormal cardiac rhythms are identified and followed. Continuous monitoring is helpful in tracking responses to treatment and changes in clinical condition.

**Echocardiography**

There is insufficient evidence for or against the routine use of echocardiography in pediatric cardiac arrest. When appropriately trained personnel are available,
Echocardiography may be considered to identify patients with potentially treatable causes of the arrest, particularly pericardial tamponade and inadequate ventricular filling (Class IIb, LOE C). Minimize interruption of CPR while performing echocardiography.

**End-Tidal CO\(_2\)** (PETCO\(_2\))

Continuous capnography or capnometry monitoring, if available, may be beneficial during CPR, to help guide therapy, especially the effectiveness of chest compressions (Class IIa, LOE C). Animal and adult studies show a strong correlation between PETCO\(_2\) and interventions that increase cardiac output during CPR or shock. If the PETCO\(_2\) is consistently <10 to 15 mmHg, focus efforts on improving chest compressions and make sure that the victim does not receive excessive ventilation. An abrupt and sustained rise in PETCO\(_2\) in adults and animals is observed just prior to clinical identification of ROSC, so use of PETCO\(_2\) may spare the rescuer from interrupting chest compressions for a pulse check. PETCO\(_2\) must be interpreted with caution for 1 to 2 minutes after administration of epinephrine or other vasoconstrictive medications because these medications may decrease the end-tidal CO\(_2\) level by reducing pulmonary blood flow.

**Vascular Access**

Vascular access is essential for administering medications and drawing blood samples. Obtaining peripheral venous access can be challenging in infants and children during an emergency; intraosseous (IO) access can be quickly established with minimal complications by providers with varied levels of training. Limit the time spent attempting to establish peripheral venous access in a critically ill or injured child.

**Intraosseous (IO) Access**

IO access is a rapid, safe, effective, and acceptable route for vascular access in children, and it is useful as the initial vascular access in cases of cardiac arrest (Class I, LOE C). All intravenous medications can be administered intraosseously, including epinephrine, adenosine, fluids, blood products, and catecholamines. Onset of action and drug levels for most drugs are comparable to venous administration. IO access can be used to obtain blood samples for analysis including for type and cross match and blood gases during CPR, but acid-base analysis is inaccurate after sodium bicarbonate administration via the IO cannula. Use manual pressure or an infusion pump to administer viscous drugs or rapid fluid boluses; follow each medication with a saline flush to promote entry into the central circulation.
Venous Access
Peripheral IV access is acceptable during resuscitation if it can be placed rapidly, but placement may be difficult in a critically ill child. Although a central venous catheter can provide more secure long-term access, its placement requires training and experience, and the procedure can be time-consuming. Therefore central venous access is not recommended as the initial route of vascular access during an emergency. If both central and peripheral accesses are available, administer medications into the central circulation since some medications (e.g. adenosine) are more effective when administered closer to the heart, and others (e.g. calcium, amiodarone, procainamide, sympathomimetics) may be irritating when infused into a peripheral vein. The length of a central catheter can contribute to increased resistance, making it more difficult to push boluses of fluid rapidly through a multilumen central than a peripheral catheter.

Endotracheal Drug Administration
Vascular access (IO or IV) is the preferred method for drug delivery during CPR, but if it is not possible, lipid-soluble drugs, such as lidocaine, epinephrine, atropine, and naloxone (mnemonic “LEAN”) can be administered via an endotracheal tube. However, the effects may not be uniform with tracheal as compared with intravenous administration. One study of children in cardiac arrest demonstrated similar ROSC and survival rates regardless of the method of drug delivery, while three studies of adults in cardiac arrest demonstrated reduced ROSC and survival to hospital discharge with tracheal administration of epinephrine compared to vascular delivery. If CPR is in progress, stop chest compressions briefly, administer the medications, and follow with a flush of at least 5 ml of normal saline and 5 consecutive positive-pressure ventilations. Optimal endotracheal doses of medications are unknown; in general expert consensus recommends doubling or tripling the dose of lidocaine, atropine or naloxone given via the ETT. For epinephrine, a dose ten times the intravenous dose (0.1 mg/kg or 0.1 ml/kg of 1:1000 concentration) is recommended.

Medications for Pediatric Resuscitation
The effectiveness of endotracheal epinephrine during cardiac arrest is controversial. Some studies showed it to be as effective as vascular administration while other studies have not found it to be as effective. Animal studies suggested that a higher dose of epinephrine is required for endotracheal than for intravascular administration because the lower epinephrine concentrations achieved when the drug is delivered by the endotracheal route may produce predominant transient peripheral β2-adrenergic vasodilating effects. These effects can be detrimental, and cause hypotension, lower coronary artery perfusion pressure and flow, and a reduced potential for ROSC.
Non-lipid-soluble drugs (e.g. sodium bicarbonate and calcium) may injure the airway; they should not be administered via the endotracheal route.

**Emergency Fluids and Medications**

**Estimating Weight**

In the out-of-hospital setting, a child’s weight is often unknown, and even experienced personnel may not be able to estimate it accurately. Tapes with precalculated doses printed at various patient lengths have been clinically validated and are more accurate than age-based or observer (parent or provider) estimate-based methods in the prediction of body weight. Body habitus may also be an important consideration.

**Medication Dose Calculation**

To calculate the dose of resuscitation medications, use the child’s weight if it is known. If the child’s weight is unknown, it is reasonable to use a body length tape with precalculated doses (Class IIa, LOE C).

It is unclear if an adjustment in the calculation of resuscitation medications is needed in obese children. Use of the actual body weight in calculation of drug doses in obese patients may result in potentially toxic doses. Length-based tapes estimate the 50th percentile weight for length (i.e. ideal body weight), which may, theoretically, result in inadequate doses of some medications in obese patients. Despite these theoretical considerations, there are no data regarding the safety or efficacy of adjusting the doses of resuscitation medications in obese patients. Therefore, regardless of the patient’s habitus, use the actual body weight for calculating initial resuscitation drug doses or use a body length tape with precalculated doses (Class IIb, LOE C).

For subsequent doses of resuscitation drugs in both nonobese and obese patients, expert providers may consider adjusting doses to achieve the desired therapeutic effect. In general, the dose administered to a child should not exceed the standard dose recommended for adult patients.

**Medications**

**Adenosine**

Adenosine causes a temporary atrioventricular (AV) nodal conduction block and interrupts reentry circuits that involve the AV node. The drug has a wide safety margin because of its short half-life. Adenosine should be given only IV or IO, followed by a rapid saline flush to promote drug delivery to the central circulation. If adenosine is given IV, it should be administered as close to the heart as possible.
Amiodarone
Amiodarone slows AV conduction, prolongs the AV refractory period and QT interval, and slows ventricular conduction (widens the QRS). Expert consultation is strongly recommended prior to administration of amiodarone to a pediatric patient with a perfusing rhythm.

Precautions
Monitor blood pressure and electrocardiograph (ECG) during intravenous administration of amiodarone. If the patient has a perfusing rhythm, administer the drug as slowly (over 20 to 60 minutes) as the patient’s clinical condition allows; if the patient is in VF/pulseless VT, give the drug as a rapid bolus. Amiodarone causes hypotension through its vasodilatory property, and the severity is related to the infusion rate; hypotension is less common with the aqueous form of amiodarone. Decrease the infusion rate if there is prolongation of the QT interval or heart block; stop the infusion if the QRS widens to >50% of baseline or hypotension develops. Other potential complications of amiodarone include bradycardia and torsades de pointes ventricular tachycardia. Amiodarone should not be administered together with another drug that causes QT prolongation, such as procainamide, without expert consultation.

Atropine
Atropine sulfate is a parasympatholytic drug that accelerates sinus or atrial pacemakers and increases the speed of AV conduction.

Precautions
Small doses of atropine (<0.1 mg) may produce paradoxical bradycardia because of its central effect. Larger than recommended doses may be required in special circumstances such as organophosphate poisoning or exposure to nerve gas agents.

Calcium
Calcium administration is not recommended for pediatric cardiopulmonary arrest in the absence of documented hypocalcemia, calcium channel blocker overdose, hypermagnesemia, or hyperkalemia (Class III, LOE B). Routine calcium administration in cardiac arrest provides no benefit and may be harmful.

If calcium administration is indicated during cardiac arrest, either calcium chloride or calcium gluconate may be considered. Hepatic dysfunction does not appear to alter the ability of calcium gluconate to raise serum calcium levels. In critically ill children, calcium chloride may be preferred because it results in a greater increase in ionized calcium during the treatment of hypocalcemia. In the nonarrest setting, if the only venous access is peripheral, calcium gluconate is recommended because it has a lower osmolality than calcium chloride and is therefore less irritating to the vein.
**Epinephrine**

The α-adrenergic-mediated vasoconstriction of epinephrine increases aortic diastolic pressure and thus coronary perfusion pressure, a critical determinant of successful resuscitation from cardiac arrest. At low doses, the β-adrenergic effects may predominate, leading to decreased systemic vascular resistance; in the doses used during cardiac arrest, the vasoconstrictive α-effects predominate.

**Precautions**

- Do not administer catecholamines and sodium bicarbonate simultaneously through an IV catheter or tubing because alkaline solutions such as the bicarbonate inactivate the catecholamines.
- In patients with a perfusing rhythm, epinephrine causes tachycardia; it may also cause ventricular ectopy, tachyarrhythmias, vasoconstriction, and hypertension.

**Glucose**

Because infants have a relatively high glucose requirement and low glycogen stores, they may develop hypoglycemia when energy requirements rise. Check blood glucose concentration during the resuscitation and treat hypoglycemia promptly (Class I, LOE C).

**Lidocaine**

Lidocaine decreases automaticity and suppresses ventricular arrhythmias, but is not as effective as amiodarone for improving ROSC or survival to hospital admission among adult patients withVF refractory to shocks and epinephrine. Neither lidocaine nor amiodarone has been shown to improve survival to hospital discharge.

**Precautions**

Lidocaine toxicity includes myocardial and circulatory depression, drowsiness, disorientation, muscle twitching, and seizures, especially in patients with poor cardiac output and hepatic or renal failure.

**Magnesium**

Magnesium is indicated for the treatment of documented hypomagnesemia or for torsades de pointes (polymorphic VT associated with long QT interval). There is insufficient evidence to recommend for or against the routine administration of magnesium during cardiac arrest.

**Precautions**

Magnesium produces vasodilation and may cause hypotension if administered rapidly.
Procainamide

Procainamide prolongs the refractory period of the atria and ventricles and depresses conduction velocity.

Precautions

There is limited clinical data on using procainamide in infants and children. Infuse procainamide very slowly (over 30 to 60 minutes) while monitoring the ECG and blood pressure. Decrease the infusion rate if there is prolongation of the QT interval, or heart block; stop the infusion if the QRS widens to >50% of baseline or hypotension develops. Do not administer together with another drug causing QT prolongation, such as amiodarone, without expert consultation. Prior to using procainamide for a hemodynamically stable patient, expert consultation is strongly recommended.

Sodium Bicarbonate

Routine administration of sodium bicarbonate is not recommended in cardiac arrest (Class III, LOE B). Sodium bicarbonate may be administered for treatment of some toxidromes (see “Toxicological Emergencies,” below) or special resuscitation situations such as hyperkalemic cardiac arrest.

Precautions

During cardiac arrest or severe shock, arterial blood gas analysis may not accurately reflect tissue and venous acidosis. Excessive sodium bicarbonate may impair tissue oxygen delivery; cause hypokalemia, hypocalcemia, hypernatremia, and hyperosmolality; decrease the VF threshold; and impair cardiac function.

Vasopressin

There is insufficient evidence to make a recommendation for or against the routine use of vasopressin during cardiac arrest. Pediatric and adult case series/reports suggested that vasopressin or its long-acting analog, terlipressin, may be effective in refractory cardiac arrest when standard therapy fails. A large pediatric NRCPR case series, however, suggested that vasopressin is associated with lower ROSC, and a trend toward lower 24-hour and discharge survival. A preponderance of controlled trials in adults do not demonstrate a benefit.

Pulseless Arrest

In the text below, box numbers identify the corresponding step in the algorithm (Fig. 66.1).

- (Step 1) As soon as the child is found to be unresponsive with no breathing, call for help, send for a defibrillator (manual or AED), and start CPR (with supplementary oxygen if available). Attach ECG monitor or AED pads as
soon as available. Throughout resuscitation, emphasis should be placed on provision of high-quality CPR (providing chest compressions of adequate rate and depth, allowing complete chest recoil after each compression, minimizing interruptions in compressions and avoiding excessive ventilation).

Fig. 66.1: PALS pulseless arrest algorithm
While CPR is being given, determine the child's cardiac rhythm from the ECG or, if you are using an AED, the device will tell you whether the rhythm is "shockable" (e.g. VF or rapid VT) or "not shockable" (e.g. asystole or PEA). It may be necessary to temporarily interrupt chest compressions to determine the child's rhythm. Asystole and bradycardia with a wide QRS are most common in asphyxial arrest. VF and PEA are less common but VF is more likely to be present in older children with sudden witnessed arrest.

Nonshockable Rhythm: Asystole/PEA (Step 9)

PEA is an organized electric activity—most commonly slow, wide QRS complexes—without palpable pulses. Less frequently there is a sudden impairment of cardiac output with an initially normal rhythm but without pulses and with poor perfusion. This subcategory, formerly known as electromechanical dissociation (EMD), may be more reversible than asystole. For asystole and PEA (Step 10): Continue CPR with as few interruptions in chest compressions as possible. A second rescuer obtains vascular access and delivers epinephrine, 0.01 mg/kg (0.1 ml/kg of 1:10,000 solution) maximum of 1 mg (10 ml), while CPR is continued. The same epinephrine dose is repeated every 3 to 5 minutes (Class I, LOE B). There is no survival benefit from high-dose epinephrine, and it may be harmful, particularly in asphyxia (Class III, LOE B). High-dose epinephrine may be considered in exceptional circumstances, such as β-blocker overdose (Class IIb, LOE C).

Once an advanced airway is in place, 1 rescuer should give continuous chest compressions at a rate of at least 100 per minute without pause for ventilation. The second rescuer delivers ventilations at a rate of 1 breath every 6 to 8 seconds (about 8 to 10 breaths per minute). Rotate the compressor role approximately every 2 minutes to prevent compressor fatigue and deterioration in quality and rate of chest compressions. Check rhythm every 2 minutes with minimal interruptions in chest compressions. If the rhythm is "nonshockable" continue with cycles of CPR and epinephrine administration until there is evidence of ROSC or you decide to terminate the effort. If at any time the rhythm becomes "shockable," give a shock (Step 7) and immediately resume chest compressions for 2 minutes before rechecking the rhythm. Minimize time between chest compressions and shock delivery (i.e. check rhythm and deliver shocks immediately after compressions rather than after rescue breaths, if possible) and between shock delivery and resumption of chest compressions.

Search for and treat reversible causes.

Shockable Rhythm: VF/Pulseless VT (Step 2)

Defibrillation is the definitive treatment for VF (Class I, LOE B) with an overall survival rate of 17–20%. Survival is better in primary than in secondary VF. In adults, the probability of survival declines by 7–10% for each minute of arrest without CPR and defibrillation. Survival is better if early, high-quality CPR is provided with minimal interruptions. Outcome of shock delivery is best
if rescuers minimize the time between last compression and shock delivery, so rescuers should be prepared to coordinate (brief) interruptions in chest compressions to deliver shocks, and should resume compressions immediately after shock delivery.

**Defibrillators**

Defibrillators are either manual or automated (AED), with monophasic or biphasic waveforms. For further information see Part 6, “Electrical Therapies: Automated External Defibrillators, Defibrillation, Cardioversion, and Pacing.”

AEDs in institutions caring for children at risk for arrhythmias and cardiac arrest (e.g. hospitals, EDs) must be capable of recognizing pediatric cardiac rhythms and should ideally have a method of adjusting the energy level for children.

The following should be considered when using a manual defibrillator:

**Paddle Size**

In general, manual defibrillators have two sizes of hand-held paddles: adult and infant. The infant paddles may slide over or be located under the adult paddles. Manual defibrillators can also be used with hands-free pads that are self adhesive. Use the largest paddles or self-adhering electrodes that will fit on the child’s chest without touching (when possible, leave about 3 cm between the paddles or electrodes). Paddles and self-adhering pads appear to be equally effective. Self-adhering pads should be pressed firmly on the chest so that the gel on the pad completely touches the child’s chest. An appropriate paddle or self-adhesive pad size is:

- “Adult” size (8 to 10 cm) for children >10 kg (> approximately 1 year)
- “Infant” size for infants <10 kg.

**Interface**

The electrode–chest wall interface is part of the self-adhesive pad; in contrast, electrode gel must be applied liberally on manually applied paddles. Do not use saline-soaked pads, ultrasound gel, bare paddles, or alcohol pads.

**Paddle Position**

Follow package directions for placement of self-adhesive AED or monitor/defibrillator pads.

Place manual paddles over the right side of the upper chest and the apex of the heart (to the left of the nipple over the left lower ribs) so the heart is between the two paddles. Apply firm pressure. There is no advantage in an anterior-posterior position of the paddles.
Energy Dose

The lowest energy dose for effective defibrillation and the upper limit for safe defibrillation in infants and children are not known; more data are needed. It has been observed that in children with VF, an initial monophasic dose of 2 J/kg is only effective in terminating ventricular fibrillation 18–50% of the time, while similar doses of biphasic shocks are effective 48% of the time. Children with out-of-hospital VF cardiac arrest often receive more than 2 J/kg, and one in-hospital cardiac arrest study showed that children received doses between 2.5 J/kg and 3.2 J/kg to achieve ROSC. Energy doses >4 J/kg (up to 9 J/kg) have effectively defibrillated children and pediatric animals with negligible adverse effects. Based on data from adult studies and pediatric animal models, biphasic shocks appear to be at least as effective as monophasic shocks and less harmful.

It is acceptable to use an initial dose of 2 to 4 J/kg (Class IIa, LOE C), but for ease of teaching an initial dose of 2 J/kg may be considered (Class IIb, LOE C). For refractory VF, it is reasonable to increase the dose to 4 J/kg (Class IIa, LOE C). Subsequent energy levels should be at least 4 J/kg, and higher energy levels may be considered, not to exceed 10 J/kg or the adult maximum dose (Class IIb, LOE C).

AEDs

Many AEDs can accurately detect VF in children of all ages. They can differentiate “shockable” from “non-shockable” rhythms with a high degree of sensitivity and specificity. It is recommended that systems and institutions that have AED programs and that care for children should use AEDs with a high specificity to recognize pediatric shockable rhythms and a pediatric attenuating system that can be used for infants and children up to approximately 25 kg (approximately 8 years of age). If an AED with an attenuator is not available, use an AED with standard electrodes (Class IIa, LOE C).

In infants <1 year of age a manual defibrillator is preferred. If a manual defibrillator is not available, an AED with a dose attenuator may be used. An AED without a dose attenuator may be used if neither a manual defibrillator nor one with a dose attenuator is available (Class IIb, LOE C).

Integration of Defibrillation With Resuscitation Sequence

The following are important considerations:

- Provide CPR until the defibrillator is ready to deliver a shock; after shock delivery, resume CPR, beginning with chest compressions. Minimize interruptions of chest compressions. In adults with prolonged arrest and in animal models, defibrillation is more likely to be successful after a period of effective chest compressions. Ideally chest compressions should be interrupted only for ventilations (until an advanced airway is in place), rhythm check, and shock delivery. If a “shockable” rhythm is still present,
continue chest compressions after a rhythm check (when possible) while the defibrillator is charging (so chest compressions are delivered until shock delivery).

- (Step 3) Give 1 shock (2 J/kg) as quickly as possible and immediately resume CPR, beginning with chest compressions. If 1 shock fails to eliminate VF, the incremental benefit of another immediate shock is low, and resumption of CPR is likely to confer a greater value than another shock. CPR may provide coronary perfusion, increasing the likelihood of defibrillation with a subsequent shock. It is important to minimize the time between chest compressions and shock delivery and between shock delivery and resumption of postshock compressions.

- (Step 4) Continue CPR for about 2 minutes. In in-hospital settings with continuous invasive monitoring, this sequence may be modified at the expert provider’s discretion (see also Part 8.2: “Management of Cardiac Arrest”). If sufficient rescuers are present, obtain vascular (IO or IV) access. After 2 minutes of CPR, check the rhythm; recharge the defibrillator to a higher dose (4 J/kg).

- (Step 5) If a “shockable” rhythm persists, give another shock (4 J/kg). If rhythm is “nonshockable,” continue with the asystole/PEA algorithm (Steps 10 and 11).

- (Step 6) Immediately resume chest compressions. Continue CPR for approximately 2 minutes. During CPR give epinephrine 0.01 mg/kg (0.1 ml/kg of 1:10,000 concentration), maximum of 1 mg (Class I, LOE B) every 3 to 5 minutes. It is helpful if a third rescuer prepares the drug doses before the rhythm is checked so epinephrine can be administered as soon as possible. Epinephrine should be administered during chest compressions, but the timing of drug administration is less important than the need to minimize interruptions in chest compressions. Just prior to the rhythm check, the rescuer operating the defibrillator should prepare to recharge the defibrillator (4 J/kg or more with a maximum dose not to exceed 10 J/kg or the adult dose, whichever is lower).

Check the rhythm

- (Step 7) If the rhythm is “shockable,” deliver another shock (4 J/kg or more with a maximum dose not to exceed 10 J/kg or the adult dose, whichever is lower) and immediately resume CPR (beginning with chest compressions).

- (Step 8) While continuing CPR, give amiodarone (Class IIb, LOE C) or lidocaine if amiodarone is not available.

If at any time the rhythm check shows a “nonshockable” rhythm, proceed to the “Pulseless Arrest” sequence (Steps 10 or 11).

Once an advanced airway is in place, 2 rescuers no longer deliver cycles of CPR (i.e. compressions interrupted by pauses for ventilation). Instead, the compressing rescuer gives continuous chest compressions at a rate of at least 100 per minute without pause for ventilation. The rescuer delivering ventilation provides about 1 breath every 6 to 8 seconds (8 to 10 breaths per minute). Two or more rescuers should rotate the compressor role approximately every 2 minutes to prevent compressor fatigue and deterioration in quality and rate of chest compressions.
If defibrillation successfully restores an organized rhythm (or there is other evidence of ROSC, such as an abrupt rise in PETCO₂ or visible pulsations on an arterial waveform), check the child’s pulse to determine if a perfusing rhythm is present. If a pulse is present, continue with postresuscitation care.

If defibrillation is successful but VF recurs, resume CPR and give another bolus of amiodarone before trying to defibrillate with the previously successful shock dose.

Search for and treat reversible causes.

**Torsades de Pointes**

This polymorphic VT is associated with a long QT interval, which may be congenital or may result from toxicity with type IA antiarrhythmics (e.g. procainamide, quinidine, and disopyramide) or type III antiarrhythmics (e.g. sotalol and amiodarone), tricyclic antidepressants (see below), digitalis, or drug interactions.

**Treatment**

Torsades de pointes VT typically deteriorates rapidly to VF or pulseless VT, so providers should initiate CPR and proceed with defibrillation when pulseless arrest develops (see above). Regardless of the cause, treat torsades de pointes with a rapid (over several minutes) IV infusion of magnesium sulfate (25–50 mg/kg; maximum single dose 2 gm).

**Bradycardia**

Box numbers in the text below refer to the corresponding boxes in the PALS Bradycardia Algorithm (Fig. 66.2). This algorithm applies to the care of the infant or child with bradycardia and cardiorespiratory compromise, but a palpable pulse. If at any time the patient develops pulseless arrest, see the PALS Pulseless Arrest Algorithm.

Emergency treatment of bradycardia is indicated when the rhythm results in hemodynamic compromise.

- (Box 1) Support a patent airway, breathing, and circulation as needed. Administer oxygen, attach an ECG monitor/defibrillator, and obtain vascular access.
- (Box 2) Reassess the patient to determine if bradycardia persists and is still causing cardiorespiratory symptoms despite adequate oxygenation and ventilation.
- (Box 4a) If pulses, perfusion, and respirations are adequate, no emergency treatment is necessary. Monitor and proceed with evaluation.
- (Box 3) If heart rate is <60 beats per minute with poor perfusion despite effective ventilation with oxygen, start CPR.
- (Box 4) After 2 minutes reevaluate the patient to determine if bradycardia and signs of hemodynamic compromise persist. Verify that the support is adequate (e.g. check airway, oxygen source, and effectiveness of ventilation).
(Box 5) Medications and pacing:
- Continue to support airway, ventilation, oxygenation, and chest compressions (Class I, LOE B). If bradycardia persists or responds only transiently, give epinephrine IV (or IO) 0.01 mg/kg (0.1 ml/kg of 1:10,000 solution) or if IV/IO access not available, give endotracheally 0.1 mg/kg (0.1 ml/kg of 1:1,000 solution) (Class I, LOE B).
- If bradycardia is due to increased vagal tone or primary AV conduction block (i.e. not secondary to factors such as hypoxia), give IV/IO atropine 0.02 mg/kg or an endotracheal dose of 0.04 to 0.06 mg/kg (Class I, LOE C).
- Emergency transcutaneous pacing may be lifesaving if the bradycardia is due to complete heart block or sinus node dysfunction unresponsive to ventilation, oxygenation, chest compressions, and medications, especially if it is associated with congenital or acquired heart disease (Class IIb, LOE C). Pacing is not useful for asystole or bradycardia due to postarrest hypoxic/ischemic myocardial insult or respiratory failure.

**Tachycardia**

The box numbers in the text below correspond to the numbered boxes in the Tachycardia Algorithm (Fig. 66.3).
If there are signs of poor perfusion and pulses are not palpable, proceed with the PALS pulseless arrest algorithm (see Fig. 66.1).

(Box 1) If pulses are palpable and the patient has adequate perfusion
- Assess and support airway, breathing, and circulation
- Provide oxygen
- Attach monitor/defibrillator
- Obtain vascular access
- Evaluate 12-lead ECG and assess QRS duration (Box 2).

**Narrow-Complex (≤0.09 Second) Tachycardia**

Evaluation of a 12-lead ECG (Box 3) and the patient’s clinical presentation and history (Boxes 4 and 5) should help differentiate sinus tachycardia from...
supraventricular tachycardia (SVT). If the rhythm is sinus tachycardia, search for and treat reversible causes.

Supraventricular Tachycardia (Box 5)

- Monitor rhythm during therapy to evaluate the effect of interventions. The choice of therapy is determined by the patient’s degree of hemodynamic instability.
- Attempt vagal stimulation (Box 7) first, unless the patient is hemodynamically unstable or the procedure will unduly delay chemical or electric cardioversion (Class IIa, LOE C). In infants and young children, apply ice to the face without occluding the airway.
- In older children, carotid sinus massage or Valsalva maneuvers are safe.
- One method for performing a Valsalva maneuver is to have the child blow through a narrow straw. Do not apply pressure to the eye because this can damage the retina.
- Pharmacologic cardioversion with adenosine (Box 8) is very effective with minimal and transient side effects. If IV/IO access is readily available, adenosine is the drug of choice (Class I, LOE C). Side effects are usually transient. Administer IV/IO adenosine 0.1 mg/kg using 2 syringes connected to a T-connector or stopcock; give adenosine rapidly with 1 syringe and immediately flush with ≥5 ml of normal saline with the other. An IV/IO dose of Verapamil, 0.1 to 0.3 mg/kg is also effective in terminating SVT in older children, but it should not be used in infants without expert consultation (Class III, LOE C) because it may cause potential myocardial depression, hypotension, and cardiac arrest.
- If the patient is hemodynamically unstable or if adenosine is ineffective, perform electric synchronized cardioversion (Box 8). Use sedation, if possible. Start with a dose of 0.5 to 1 J/kg. If unsuccessful, increase the dose to 2 J/kg (Class IIb, LOE C). If a second shock is unsuccessful or the tachycardia recurs quickly, consider amiodarone or procainamide before a third shock.
- Consider amiodarone 5 mg/kg IO/IV or procainamide 15 mg/kg IO/IV for a patient with SVT unresponsive to vagal maneuvers and adenosine and/or electric cardioversion; for hemodynamically stable patients, expert consultation is strongly recommended prior to administration (Class IIb, LOE C). Both amiodarone and procainamide must be infused slowly (amiodarone over 20 to 60 minutes and procainamide over 30 to 60 minutes), depending on the urgency, while the ECG and blood pressure are monitored. If there is no effect and there are no signs of toxicity, give additional doses. Avoid the simultaneous use of amiodarone and procainamide without expert consultation.

Wide-Complex (>0.09 Second) Tachycardia (Box 9)

Wide-complex tachycardia often originates in the ventricles (ventricular tachycardia) but may be supraventricular in origin.

Because all arrhythmia therapies have a potential for serious adverse effects, consultation with an expert in pediatric arrhythmias is strongly recommended before treating children who are hemodynamically stable.
The following are important considerations in treating wide-complex tachycardia in hemodynamically stable patients:

- **Adenosine** may be useful in differentiating SVT from VT and converting wide-complex tachycardia of supraventricular origin (Box 12). Adenosine should be considered only if the rhythm is regular and the QRS is monomorphic. Do not use adenosine in patients with known Wolff-Parkinson-White syndrome and wide-complex tachycardia.

- **Electric cardioversion** after sedation using a starting energy dose of 0.5 to 1 J/kg. If that fails, increase the dose to 2 J/kg (Class IIb, LOE C) (Box 11).

- **Pharmacologic conversion** with either intravenous amiodarone (5 mg/kg over 20 to 60 minutes) or procainamide (15 mg/kg given over 30 to 60 minutes) while monitoring ECG and blood pressure. Stop or slow the infusion if there is a decline in blood pressure or the QRS widens (Box 13). Expert consultation is strongly recommended prior to administration.

- **In hemodynamically unstable patients**: Electric cardioversion is recommended using a starting energy dose of 0.5 to 1 J/kg. If that fails, increase the dose to 2 J/kg (Class 1, LOE C).

## Special Resuscitation Situations

### Septic Shock

There appears to be no clinically important difference in survival of children who are treated for septic shock with colloid compared with those who are treated with isotonic crystalloid solutions. Although colloid may be beneficial as part of a protocol-driven strategy, it is reasonable to use isotonic crystalloid solution as the initial fluid for the treatment of septic shock (Class IIa, LOE C).

Monitoring the central venous (superior vena cava) oxygen saturation (ScvO₂) may be useful to titrate therapy in infants and children with septic shock. Protocol-driven or “goal-directed” therapy, with a target ScvO₂ ≥70% appears to improve patient survival in severe sepsis (Class IIb, LOE B).

Early assisted ventilation may be considered as part of a protocol-driven strategy for septic shock (Class IIb, LOE C).

Etomidate has been shown to facilitate endotracheal intubation in infants and children with minimal hemodynamic effect, but do not use it routinely in pediatric patients with evidence of septic shock (Class III, LOE B). Adrenal suppression is seen after administration of etomidate in children and adults. In children and adults with septic shock, etomidate administration is associated with a higher mortality rate.

### Hypovolemic Shock

Use an isotonic crystalloid solution (e.g. lactated Ringer’s solution or normal saline) as the initial fluid for the treatment of shock (Class I, LOE A). There is no added benefit in using colloid (e.g. albumin) during the early phase of resuscitation.
Treat signs of shock with a bolus of 20 ml/kg of isotonic crystalloid even if blood pressure is normal (Class IIb, LOE C). Crystalloids may have an associated survival benefit over colloid for children with shock secondary to general trauma, traumatic brain injury, and burns. There is no evidence to support the use of a specific isotonic crystalloid. Give additional boluses (20 ml/kg) if systemic perfusion fails to improve. There are insufficient data to make a recommendation for or against use of hypertonic saline for shock associated with head injuries or hypovolemia.

There is insufficient evidence in infants and children to make a recommendation about the best timing or extent of volume resuscitation for children with hemorrhagic shock following trauma.

**Trauma**

Some aspects of trauma resuscitation require emphasis because improperly performed resuscitation is a major cause of preventable pediatric deaths.

Common errors in pediatric trauma resuscitation include failure to open and maintain the airway, failure to provide appropriate fluid resuscitation, and failure to recognize and treat internal bleeding. Involve a qualified surgeon early and, if possible, transport a child with multisystem trauma to a trauma center with pediatric expertise.

The following are special aspects of trauma resuscitation:

- When the mechanism of injury is compatible with cervical spinal injury, restrict motion of the cervical spine and avoid traction or movement of the head and neck. Open and maintain the airway with a jaw thrust, and do not tilt the head.
- If the airway cannot be opened with a jaw thrust, use a head-tilt–chin-lift because you must establish a patent airway. Because of the disproportionately large head of infants and young children, optimal positioning may require recessing the occiput or elevating the torso to avoid undesirable backboard-induced cervical flexion.
- Do not routinely hyperventilate even in case of head injury (Class III, LOE C). Intentional brief hyperventilation may be used as a temporizing rescue therapy if there are signs of impending brain herniation (e.g. sudden rise in measured intracranial pressure, dilation of one or both pupils with decreased response to light, bradycardia, and hypertension).
- Suspect thoracic injury in all thoracoabdominal trauma, even in the absence of external injuries. Tension pneumothorax, hemothorax, or pulmonary contusion may impair oxygenation and ventilation.
- If the patient has maxillofacial trauma or if you suspect a basilar skull fracture, insert an orogastric rather than a nasogastric tube (Class IIa, LOE C).
- In the very select circumstances of children with cardiac arrest from penetrating trauma with short transport times, consider performing resuscitative thoracotomy (Class IIb, LOE C).
- Consider intra-abdominal hemorrhage, tension pneumothorax, pericardial tamponade, and spinal cord injury in infants and children, and intracranial hemorrhage in infants, as causes of shock.
Single Ventricle

Standard prearrest and arrest resuscitation procedures should be followed for infants and children with single ventricle anatomy following Stage I palliation or in the infant or neonate with a univentricular heart and a shunt to augment pulmonary blood flow. Heparin may be considered for infants with a systemic-pulmonary artery shunt or right ventricular-pulmonary artery shunt. Following resuscitation from cardiac arrest, oxygen administration should be adjusted to balance systemic and pulmonary blood flow, targeting an oxyhemoglobin saturation (SpO₂) of approximately 80%. End-tidal CO₂ (PETCO₂) in the single-ventricle patient during cardiac arrest may not be a reliable indicator of CPR quality because pulmonary blood flow changes rapidly and does not necessarily reflect cardiac output during CPR.

Neonates in a prearrest state due to elevated pulmonary-to-systemic flow ratio prior to Stage I repair might benefit from a PaCO₂ of 50–60 mm Hg, which can be achieved during mechanical ventilation by reducing minute ventilation, increasing the inspired fraction of CO₂ or administering opioids with or without chemical paralysis (Class IIb, LOE B). Neonates in a low cardiac output state following stage I repair may benefit from systemic vasodilators such as α-adrenergic antagonists (e.g. phenoxybenzamine) to treat or ameliorate increased systemic vascular resistance, improve systemic oxygen delivery, and reduce the likelihood of cardiac arrest (Class IIa, LOE B). Other drugs that reduce systemic vascular resistance (e.g. milrinone or nipride) may also be considered for patients with excessive Qp:Qs (Class IIa, LOE B). Following Stage I repair, evaluation of oxygen delivery and extraction [e.g. using central venous oxygen saturation (ScvO₂) and near-infrared spectroscopy] may help identify evolving changes in hemodynamics that may herald impending cardiac arrest. During cardiopulmonary arrest, it is reasonable to consider extracorporeal membrane oxygenation (ECMO) for patients with single ventricle anatomy who have undergone Stage I procedure (Class IIa, LOE B).

Hypoventilation may improve oxygen delivery in patients in a prearrest state with Fontan or hemi-Fontan/bidirectional Glenn (BDG) physiology (Class IIa, LOE B). Negative-pressure ventilation may improve cardiac output (Class IIa, LOE C). During cardiopulmonary arrest, it is reasonable to consider extracorporeal membrane oxygenation (ECMO) for patients with Fontan physiology (Class IIa, LOE C). It is unclear at this time whether patients with hemi-Fontan/BDG physiology in cardiac arrest might benefit from ECMO.

Pulmonary Hypertension

Standard PALS, including oxygenation and ventilation, should be provided to patients with pulmonary hypertension and a cardiopulmonary arrest. It may be beneficial to attempt to correct hypercarbia. Administration of a bolus of isotonic fluid may be useful to maintain preload to the systemic ventricle. If intravenous or inhaled therapy to decrease pulmonary hypertension has been interrupted,
reinstitute it (Class IIa, LOE C). Consider administering inhaled nitric oxide (iNO) or aerosolized prostacyclin or analogue to reduce pulmonary vascular resistance (Class IIa, LOE C). If iNO is not available, consider giving an intravenous bolus of prostacyclin (Class IIa, LOE C). ECMO may be beneficial if instituted early in the resuscitation (Class IIa, LOE C).

**Children with Special Healthcare Needs**

Children with special healthcare needs may require emergency care for chronic conditions (e.g. obstruction of a tracheostomy), failure of support technology (e.g. ventilator failure), progression of their underlying disease, or events unrelated to those special needs.

For additional information about CPR see Part 13: “Pediatric Basic Life Support.”

**Ventilation With a Tracheostomy or Stoma**

Parents, school nurses, and home healthcare providers should know how to assess patency of the airway, clear the airway, replace the tracheostomy tube, and perform CPR using the artificial airway in a child with a tracheostomy.

Parents and providers should be able to ventilate via a tracheostomy tube and verify effectiveness by assessing chest expansion. If, after suctioning, the chest does not expand with ventilation, remove the tracheostomy tube and replace it or insert a same-sized endotracheal tube, if available, into the tracheal stoma. If a clean tube is unavailable, perform mouth-to-stoma or mask-to-stoma ventilations. If the upper airway is patent, bag-mask ventilation via the nose and mouth may be effective if the tracheal stoma is manually occluded.

**Toxicological Emergencies**

Overdose with local anesthetics, cocaine, narcotics, tricyclic antidepressants, calcium channel blockers, and β-adrenergic blockers may require specific treatment modalities in addition to the usual resuscitative measures.

**Local Anesthetic**

Local anesthetics are used topically, intravenously, subcutaneously, and in epidural or other catheters for delivery of regional analgesia. The toxicity of local anesthetics is well recognized in children; they may cause changes in mental status, seizures, arrhythmias, or even cardiac arrest in settings of overdose or inadvertent vascular administration. Multiple case reports, including some pediatric reports, have described successful treatment of local anesthetic toxicity with intravenous lipid emulsion.

**Cocaine**

Acute coronary syndrome, manifested by chest pain and cardiac rhythm disturbances (including VT and VF), is the most frequent cocaine-related reason
for hospitalization in adults. Cocaine also may prolong the action potential and QRS duration and impairs myocardial contractility.

Treatment

- Hyperthermia, which may result from cocaine-induced hypermetabolism, is associated with an increase in toxicity; therefore treat elevated temperature aggressively.
- For coronary vasospasm consider nitroglycerin (Class IIa, LOE C), a benzodiazepine, and phentolamine (an α-adrenergic antagonist) (Class IIb, LOE C).
- Do not give β-adrenergic blockers (Class III, LOE C),
- For ventricular arrhythmia, consider sodium bicarbonate (1 to 2 mEq/kg) administration (Class IIb, LOE C) in addition to standard treatment.
- To prevent arrhythmias secondary to myocardial infarction, consider a lidocaine bolus followed by a lidocaine infusion (Class IIb, LOE C).

Tricyclic Antidepressants and Other Sodium Channel Blockers

- Toxic doses cause cardiovascular abnormalities including intraventricular conduction delays, heart block, bradycardia, prolongation of the QT interval, ventricular arrhythmias (including torsades de pointes, VT, and VF), hypotension, seizures, and a depressed level of consciousness.

Treatment

- Give 1 to 2 mEq/kg intravenous boluses of sodium bicarbonate until arterial pH is >7.45; then provide an infusion of 150 mEq NaHCO₃ per liter of D5W to maintain alkalosis. In cases of severe intoxication increase the pH to 7.50 to 7.55. Do not administer Class IA (quinidine, procainamide), Class IC (flecainide, propafenone), or Class III (amiodarone and sotalol) antiarrhythmics, which may exacerbate cardiac toxicity (Class III, LOE C).
- For hypotension, give boluses (10 ml/kg each) of normal saline. If hypotension persists, epinephrine and norepinephrine are more effective than dopamine in raising blood pressure.
- Consider ECMO if high-dose vasopressors do not maintain blood pressure.

Calcium Channel Blockers

Manifestations of toxicity include hypotension, ECG changes (prolongation of the QT interval, widening of the QRS, and right bundle branch block), arrhythmias (bradycardia, SVT, VT, torsades de pointes, and VF), seizures, and altered mental status.

Treatment

- Treat mild hypotension with small boluses (5 to 10 ml/kg) of normal saline because myocardial depression may limit the amount of fluid the patient can tolerate.
- The effectiveness of calcium administration is variable (Class IIb, LOE C). Infuse 20 mg/kg (0.2 ml/kg) of 10% calcium chloride intravenously over
5–10 minutes; if there is a beneficial effect, give an infusion of 20–50 mg/kg per hour. Monitor serum ionized calcium concentration to prevent hypercalcemia. It is preferable to administer calcium chloride via a central venous catheter; use caution when infusing into a peripheral IV because infiltration can cause severe tissue injury. If no central venous catheter is available, infuse calcium gluconate through a secure peripheral IV. For bradycardia and hypotension, consider vasopressors and inotropes such as norepinephrine or epinephrine (Class IIb, LOE C).

There are insufficient data to recommend for or against an infusion of insulin and glucose or sodium bicarbonate.

**Beta-Adrenergic Blockers**

Toxic doses of β-adrenergic blockers cause bradycardia, heart block, and decreased cardiac contractility, and some (e.g. propranolol and sotalol) may also prolong the QRS and the QT intervals.

**Treatment**

- High-dose epinephrine infusion may be effective (Class IIb, LOE C).
- Consider glucagon (Class IIb, LOE C). In adolescents infuse 5 to 10 mg of glucagon over several minutes followed by an IV infusion of 1 to 5 mg/hour.
- Consider an infusion of glucose and insulin (Class IIb, LOE C).
- There are insufficient data to make a recommendation for or against using calcium (Class IIb, LOE C).
- Calcium may be considered if glucagon and catecholamines are ineffective (Class IIb, LOE C).

**Opioids**

Narcotics may cause hypoventilation, apnea, bradycardia, and hypotension in addition to depressed responsiveness.

**Treatment**

- Support of oxygenation and ventilation is the initial treatment for severe respiratory depression from any cause (Class I).
- Naloxone reverses the respiratory depression of narcotic overdose (Class I, LOE B), but in persons with long-term addictions or cardiovascular disease, naloxone may markedly increase heart rate and blood pressure and cause acute pulmonary edema, cardiac arrhythmias (including asystole), and seizures. Ventilation before administration of naloxone appears to reduce these adverse effects. Intramuscular administration of naloxone may lower the risk by slowing the onset of drug effect.

**Postresuscitation Stabilization (Post-Cardiac Arrest Care)**

The goals of postresuscitation care are to preserve neurologic function, prevent secondary organ injury, diagnose and treat the cause of illness, and enable the patient to arrive at a pediatric tertiary-care facility in an optimal physiologic state.
Frequent reassessment of the patient is necessary because cardiorespiratory status may deteriorate.

**Respiratory System**

Data suggest that hyperoxemia (i.e. a high PaO₂) enhances the oxidative injury observed following ischemia-reperfusion. Therefore, one goal of the postresuscitation phase is to reduce the risk of oxidative injury while maintaining adequate oxygen delivery. A practical way to achieve that goal is to reduce the FiO₂ to reduce the PaO₂ while ensuring adequate arterial oxygen content. Specifically, use the lowest inspired oxygen concentration that will maintain the arterial oxyhemoglobin saturation ≥94%. Provided appropriate equipment is available, once ROSC is achieved, adjust the FiO₂ to the minimum concentration needed to achieve transcutaneous or arterial oxygen saturation at least 94%, with the goal of avoiding hyperoxia while ensuring adequate oxygen delivery. Since an arterial oxyhemoglobin saturation of 100% may correspond to a PaO₂ anywhere between ~80 and 500 mmHg, in general it is appropriate to wean the FiO₂ for a saturation of 100%, provided the oxyhemoglobin saturation can be maintained ≥94%.

In addition to the usual clinical signs of adequate perfusion, laboratory parameters of adequate oxygen delivery over time include resolution of metabolic acidosis, reduced lactate concentration, and normalization of venous oxygen saturation.

Assist ventilation if there is significant respiratory compromise (tachypnea, respiratory distress with agitation or decreased responsiveness, poor air exchange, cyanosis, hypoxemia). If the patient is already intubated, verify tube position, patency, and security. In the hospital setting, consider obtaining arterial blood gases 10 to 15 minutes after establishing the initial mechanical ventilator settings and make appropriate adjustments. Ideally, correlate blood gases with capnographic end-tidal CO₂ concentration (PETCO₂) to enable noninvasive monitoring of ventilation.

Control pain and discomfort with analgesics (e.g. fentanyl or morphine) and sedatives (e.g. lorazepam or midazolam). Neuromuscular blocking agents (eg, vecuronium or pancuronium) with analgesia or sedation, or both, may improve oxygenation and ventilation in case of patient-ventilator dyssynchrony or severely compromised pulmonary function. Neuromuscular blockers, however, can mask seizures and impede neurologic examinations.

Monitor exhaled CO₂ (PETCO₂), especially during transport and diagnostic procedures (Class IIa, LOE B).

Insert a gastric tube to relieve and help prevent gastric inflation.

**Cardiovascular System**

Monitor heart rate and blood pressure. Repeat clinical evaluations at frequent intervals until the patient is stable. Consider monitoring urine output with an indwelling catheter. A 12-lead ECG may be helpful in establishing the cause of the cardiac arrest.
Remove the IO access after alternative (preferably 2) secure venous catheters are placed. Monitor venous or arterial blood gas analysis and serum electrolytes, glucose, and calcium concentrations. A chest X-ray should be performed to evaluate endotracheal tube position, heart size, and pulmonary status. Consider obtaining arterial lactate and central venous oxygen saturation to assess adequacy of tissue oxygen delivery.

Drugs Used to Maintain Cardiac Output

**Medications to Maintain Cardiac Output and for Postresuscitation Stabilization**

Myocardial dysfunction and vascular instability are common following resuscitation from cardiac arrest. Systemic and pulmonary vascular resistances are often increased initially, except in some cases of septic shock. The postarrest effects on the cardiovascular system may evolve over time, with an initial hyperdynamic state replaced by worsening cardiac function. Therefore in infants and children with documented or suspected cardiovascular dysfunction after cardiac arrest, it is reasonable to administer vasoactive drugs titrated to improve myocardial function and organ perfusion.

There are no studies evaluating the benefit of specific vasoactive agents after ROSC in infants and children. In animal studies after resuscitation from cardiac arrest and post-cardiac surgical experience in children and adults, hemodynamic improvement was associated with administration of selected vasoactive agents. Each drug and dose must be tailored to the patient because clinical response is variable. Infuse all vasoactive drugs into a secure IV line. The potential adverse effects of catecholamines include local ischemia and ulceration, tachycardia, atrial and ventricular tachyarrhythmias, hypertension, and metabolic changes (hyperglycemia, increased lactate concentration, and hypokalemia).

**Epinephrine**

Low-dose infusions (<0.3 mcg/kg per minute) generally produce β-adrenergic actions (tachycardia, potent inotropy, and decreased systemic vascular resistance). Higher-dose infusions (>0.3 mcg/kg per minute) cause α-adrenergic vasoconstriction. Because there is great interpatient variability in response, titrate the drug to the desired effect. Epinephrine or norepinephrine may be preferable to dopamine in patients (especially infants) with marked circulatory instability and decompensated shock.

**Dopamine**

Dopamine can produce direct dopaminergic effects and indirect β- and α-adrenergic effects through stimulation of norepinephrine release. Titrate dopamine to treat shock that is unresponsive to fluids and when systemic vascular resistance is low (Class IIb, LOE C). Typically a dose of 2 to 20 mcg/kg per minute is used. Although low-dose dopamine infusion has been frequently recommended to maintain renal blood flow or improve renal
function, data do not show benefit from such therapy. At higher doses (>5 mcg/kg per minute), dopamine stimulates cardiac β-adrenergic receptors, but this effect may be reduced in infants and in patients with chronic congestive heart failure. Infusion rates >20 mcg/kg per minute may result in excessive vasoconstriction. In one study in single ventricle postoperative cardiac patients, dopamine increased oxygen consumption while not improving blood pressure or cardiac output.

**Dobutamine Hydrochloride**

Dobutamine has a relatively selective effect on β1- and β2-adrenergic receptors due to effects of the two isomers; one is an α-adrenergic agonist, and the other is an α-adrenergic antagonist. Dobutamine increases myocardial contractility and can decrease peripheral vascular resistance. Titrate the infusion to improve cardiac output and blood pressure due to poor myocardial function.

**Norepinephrine**

Norepinephrine is a potent vasopressor promoting peripheral vasoconstriction. Titrate the infusion to treat shock with low systemic vascular resistance (septic, anaphylactic, spinal, or vasodilatory) unresponsive to fluid.

**Sodium Nitroprusside**

Sodium nitroprusside increases cardiac output by decreasing vascular resistance (afterload). If hypotension is related to poor myocardial function, consider using a combination of sodium nitroprusside to reduce afterload and an inotrope to improve contractility. Fluid administration may be required secondary to vasodilatory effects.

**Inodilators**

Inodilators (inamrinone and milrinone) augment cardiac output with little effect on myocardial oxygen demand. It is reasonable to use an inodilator in a highly monitored setting for treatment of myocardial dysfunction with increased systemic or pulmonary vascular resistance (Class Ila, LOE B). Administration of fluids may be required secondary to vasodilatory effects.

Inodilators have a long half-life with a delay in reaching a steady-state hemodynamic effect after the infusion rate is changed (18 hours with inamrinone and 4.5 hours with milrinone). In cases of toxicity the cardiovascular effects may persist for several hours even after the infusion is discontinued.

**Neurologic System**

A primary goal of resuscitation is to preserve brain function. Limit the risk of secondary neuronal injury by adhering to the following precautions:

- Do not routinely provide excessive ventilation or hyperventilation. Hyperventilation has no benefit and may impair neurologic outcome by
adversely affecting cardiac output and cerebral perfusion. Intentional brief hyperventilation may be used as temporizing rescue therapy in response to signs of impending cerebral herniation (e.g., sudden rise in measured intracranial pressure, dilated pupil(s) not responsive to light, bradycardia, hypertension).

- Therapeutic hypothermia (32°–34°C) may be considered for children who remain comatose after resuscitation from cardiac arrest (Class IIb, LOE C). It is reasonable for adolescents resuscitated from sudden, witnessed, out-of-hospital VF cardiac arrest (Class IIa, LOE C). Although there are no randomized studies in the pediatric population on the effect of therapeutic hypothermia, it is of benefit in adults following witnessed out-of-hospital VF arrest and in asphyxiated newborns.

- The ideal method and duration of cooling and rewarming are not known. Prevent shivering by providing sedation and, if needed, neuromuscular blockade, recognizing that this can mask seizure activity. Closely watch for signs of infection. Other potential complications of hypothermia include diminished cardiac output, arrhythmia, pancreatitis, coagulopathy, thrombocytopenia, hypophosphatemia, hypovolemia from cold diuresis, hypokalemia, and hypomagnesemia.

- Monitor temperature continuously, if possible, and treat fever (>38°C) aggressively with antipyretics and cooling devices because fever adversely influences recovery from ischemic brain injury (Class IIa, LOE C).

- Treat postischemic seizures aggressively; search for a correctable metabolic cause such as hypoglycemia or electrolyte imbalance.

- Avoid rewarming from 32–34°C faster than 0.5°C per 2 hours unless the patient requires rapid rewarming for clinical reasons.

Renal System

Decreased urine output (<1 ml/kg per hour in infants and children or <30 ml/hour in adolescents) may be caused by prerenal conditions (e.g., dehydration, inadequate systemic perfusion), renal ischemic damage, or a combination of factors. Avoid nephrotoxic medications and adjust the dose of medications excreted by the kidneys until you have checked renal function.

Interhospital Transport

Ideally postresuscitation care should be provided by a trained team from a pediatric tertiary care facility. Contact such a team as early as possible during the resuscitation attempt and coordinate transportation with the receiving unit. Transport team members should be trained and experienced in the care of critically ill and injured children and supervised by a pediatric emergency medicine or pediatric critical care physician. The mode of transport and composition of the team should be established for each system based on the care required by each patient. Monitor exhaled CO₂ (qualitative colorimetric detector or capnography) during interhospital or intrahospital transport of intubated patients (Class IIa, LOE B).
Family Presence During Resuscitation

Family presence during CPR is increasingly common, and most parents would like to be given the opportunity to be present during resuscitation of their child. Studies show that family members who are present at a resuscitation would recommend it to others. Parents of chronically ill children are comfortable with medical equipment and emergency procedures, but even family members with no medical background who were at the side of a loved one to say goodbye during the final moments of life believe that their presence was beneficial to the patient, comforting for them, and helpful in their adjustment and grieving process. Standardized psychological examinations suggest that, compared with those not present, family members present during attempted resuscitations have less anxiety and depression and more constructive grieving behavior. Parents or family members often fail to ask, but healthcare providers should offer the opportunity in most situations. Whenever possible, provide family members with the option of being present during resuscitation of an infant or child (Class I, LOE B).

Family presence during resuscitation, in general, is not disruptive, and does not create stress among staff or negatively affect their performance. If the presence of family members creates undue staff stress or is considered detrimental to the resuscitation, then family members should be respectfully asked to leave (Class IIa, LOE C). Members of the resuscitation team must be sensitive to the presence of family members, and one person should be assigned to remain with the family to comfort, answer questions, and support the family.

Termination of Resuscitative Efforts

There are no reliable predictors of outcome to guide when to terminate resuscitative efforts in children.

Clinical variables associated with survival include length of CPR, number of doses of epinephrine, age, witnessed versus unwitnessed cardiac arrest, and the first and subsequent rhythm. None of these associations, however, predict outcome. Witnessed collapse, bystander CPR, and a short interval from collapse to arrival of professionals improve the chances of a successful resuscitation. Intact survival has been documented after unusually prolonged in-hospital resuscitation.

Sudden Unexplained Deaths

Increasing evidence demonstrates that some cases of sudden infant death syndrome (SIDS) and sudden death in older children and young adults may be associated with genetic mutations causing cardiac ion channelopathies. Channelopathies are dysfunctional myocyte ion channels that result in abnormal movement of electrolytes into and/or out of the cell and predispose the heart to arrhythmia. Mutations causing cardiac ion channelopathies are found in 2–10%
of victims and in 14–20% of young adults with sudden death in whom the cause of death is not evident in a routine autopsy. Clinical and laboratory (e.g. ECG, molecular-genetic screening) investigations of first- and second-degree relatives of patients with sudden unexplained death reported inherited, arrhythmogenic disease in 22–53% of families.

Therefore when sudden unexplained cardiac arrest occurs in children and young adults, obtain a complete past medical and family history (including a history of syncopal episodes, seizures, unexplained accidents or drownings, or sudden unexpected death at <50 years old) and review previous ECGs.

All infants, children, and young adults with sudden unexpected death should, where resources allow, have an unrestricted, complete autopsy, preferably performed by a pathologist with training and experience in cardiovascular pathology. Consider appropriate preservation and genetic analysis of tissue to determine the presence of a channelopathy. Refer families of patients that do not have a cause of death found on autopsy to a healthcare provider or center with expertise in arrhythmias (Class I, LOE C).

Class of Evidence

I: Large, randomized trials with clear-cut results; low risk of false-positive (α) error or false-negative (β) error.

II: Small, randomized trials with uncertain results; moderate to high risk of false-positive (α) error and/or false-negative (β) error.

III: Nonrandomized, contemporaneous controls.

IV: Nonrandomized, historical controls and expert opinion.

V: Case series, uncontrolled studies, and expert opinion.

Levels of Evidence (LOE)

A: Supported by at least two class I investigations.

B: Supported by only one class I investigation.

C: Supported by class II investigations only.

D: Supported by at least one class III investigation.

E: Supported by class IV or class V investigations only.


The American Heart Association requests that this document be cited as follows:


In the PICU, many machines, devices, and procedures are used which are relatively uncommon in other parts of the hospital. Each device has a particular job or purpose. There are reasons (indications) to use each device or procedure for assessment or treatment. Most have some risk (possible complications) as well as potential benefit, about which a physician should be aware and also equally important, is to inform parents and take written consent.

Here we have tried to approach these common PICU procedures in a stepwise manner for better understanding and performance by end user.

**Intubation**

**Introduction**

*Step 1: When to intubate a child*

- Inadequate oxygenation or ventilation
- Inability to maintain and/or protect airway
- Hemodynamic instability
- Neuromuscular dysfunction
- Failure of Central Nervous System Regulation of ventilatory drive
- Other indications—prolonged diagnostic studies or patient transport.

*Step 2: What are the contraindications*

Assessment and management of the airway is always the first priority in caring for acutely ill or injured children. Thus, there are no absolute contraindications for endotracheal intubation (ETI) by appropriately trained providers.

*Precautions*—To exclude difficult airway

*Anatomic Consideration*—There are several anatomic features in infants and children that may impact advanced airway management. These differences, most evident in children less than two to three years of age, include the following:

- A large occiput affects positioning
- A large tongue and small mouth may make laryngoscopy difficult.
The larynx may be harder to locate with the laryngoscope because it is higher and more anterior than in an adult. The epiglottis is large and floppy and may difficult to control.

**Step 3: Preparation**

Success in airway management depends on careful patient assessment, implementation of an appropriate endotracheal intubation (ETI) plan, and gathering and testing of all necessary equipment.

1. **Rapid assessment:** The clinician should perform a focused assessment of the child’s history and physical findings to identify conditions and clinical features that will impact bag-mask ventilation, laryngoscopy, and/or ETI. Examples include:
   - Congenital abnormalities associated with airway difficulties (eg, Pierre-Robin, Treacher-Collins)
   - Known difficult endotracheal intubation in the past
   - Anatomic characteristics associated with difficult airway management, such as poor mouth opening, large tongue or tonsils, small chin, short mandible, or decreased neck mobility
   - Some clinicians advocate for the use of the Mallampati score or LEMON (Look externally, Evaluate 3-3-2, Mallampati, Obstruction/obesity, Neck mobility) approach although their use has not been validated in children.

2. **Intubation plan:** Rapid sequence intubation has been shown to be safe and effective in children. However, in any child in whom laryngoscopy and intubation may be more difficult, an alternative plan that involves assistance from specialists (anesthesiologists, otolaryngologists) and/or intubation with sedation but without paralysis should be employed.

3. **Patient counseling/Informed consent:** When emergent endotracheal intubation is performed for life-threatening circumstances, consent is implied. Whenever possible, the procedure should be explained to both the parents and the child prior to intubation with emphasis on the indications for intubation and benefits of the procedure. Key components of the discussion include:
   - Medications will provide sedation and pain control throughout the procedure.
Table 67.1: Rapid overview of rapid sequence intubation in children

<table>
<thead>
<tr>
<th>Preoxygenation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Begin preoxygenation as soon as the decision to intubate is considered.</td>
</tr>
<tr>
<td>Administer oxygen at the highest concentration available.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Preparation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Identify conditions that will affect choice of medications.</td>
</tr>
<tr>
<td>Identify conditions that will predict difficult intubation or bag-mask ventilation.</td>
</tr>
<tr>
<td>Assemble equipment and check for function.</td>
</tr>
<tr>
<td>Develop contingency plan for failed intubation.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Pretreatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine: All children ≤1 year, children &lt;5 years receiving succinycholine, and older children receiving a second dose of succinylcholine. Dose: 0.02 mg/kg IV (maximum single dose 0.5 mg, minimum 0.1 mg; if no IV access, can be given IM).</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sedation</th>
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</thead>
<tbody>
<tr>
<td>Lidocaine: Optional for increased intracranial pressure. Dose: 1.5 mg/kg IV (maximum dose 100 mg). Give 2 to 3 minutes before intubation.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sedation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Etomidate: Safe with hemodynamic instability, neuroprotective, transient adrenal corticosuppression. Do not use in patients with septic shock or focal seizures. Dose: 0.3 mg/kg IV.</td>
</tr>
<tr>
<td>Thiopental: Neuroprotective. Do not use with hemodynamic instability. Dose: 3 to 5 mg/kg IV.</td>
</tr>
<tr>
<td>Ketamine: Safe with hemodynamic instability if patient is not catecholamine depleted. Use in patients with bronchospasm and septic shock. Use with caution in patients with increased intracranial pressure. Dose: 1 to 2 mg/kg IV. (If no IV access, can be given IM dose: 3 to 7 mg/kg).</td>
</tr>
<tr>
<td>Midazolam: Time to clinical effect is longer, inconsistently induces unconsciousness. May cause hemodynamic instability at doses required for sedation. Dose: 0.2 to 0.3 mg/kg IV (maximum dose 2 mg, onset of effect requires 2 to 3 minutes).</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Paralytic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Succinycholine: Do not use with chronic myopathy or denervating neuromuscular disease; 48 to 72 hours after burn, crush, or denervating injury; malignant hyperthermia; or pre-existing hyperkalemia. Dose: infants and young children: 2 mg/kg IV, older children: 1 to 1.5 mg/kg IV. (If IV access unobtainable, can be given IM, dose: 3 to 5 mg/kg).</td>
</tr>
<tr>
<td>Rocuronium: Use for children with contraindication for succinylcholine. Use with extreme caution for patients who may be difficult to intubate. Suggested dose: 1 mg/kg IV (range 0.6 to 1.2 mg/kg).</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Protection and positioning</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maintain manual cervical spine immobilization during intubation in the trauma patient.</td>
</tr>
<tr>
<td>If cervical spine injury is not potentially present, put the patient in the “sniffing position” (ie, head forward so that the external auditory canal is anterior to the shoulder and the nose and mouth point to the ceiling). Apply cricoid pressure when the child is unconscious. Remove cricoid pressure if it causes airway obstruction or difficulty viewing the larynx.</td>
</tr>
<tr>
<td>If used, maintain cricoid pressure until tracheal tube position is verified.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Positioning, with placement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Confirm tracheal tube placement with end-tidal CO2 detection and auscultation.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Postintubation management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest radiograph for tracheal tube placement; provide ongoing sedation, analgesia (eg, fentanyl 1 mcg per kilogram), and paralysis.</td>
</tr>
</tbody>
</table>
– Endotracheal intubation may not be successful.
– The subsequent planned actions if the child cannot be successfully intubated.

4. **Materials, equipment, and personnel:** Functioning airway equipment in a full range of sizes from neonate to adolescent/adult should be readily available wherever critically ill or injured children receive medical care (Table 2). Equipment should always be checked prior to performing this procedure.

**Preintubation Supplies**

1. **Personnel:** Ideally, at least three practitioners are present during emergency intubation. In addition to the laryngoscopist/intubator, an assistant can be utilized to hold cricoid pressure (when used), pass equipment, and watch the monitor, and an additional provider can be assigned to infuse medications

**Table 67.2: Airway equipment for pediatric patients**

<table>
<thead>
<tr>
<th>Supplemental oxygen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nasal cannulae (infant, child, and adult)</td>
</tr>
<tr>
<td>Clear oxygen masks (standard and nonrebreathing - infant, child, and adult)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Suction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suction catheters (6 through 16 French)</td>
</tr>
<tr>
<td>Yankauer suction tip (two sizes)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Bag-mask ventilation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Masks (neonate, infant, child, adult)</td>
</tr>
<tr>
<td>Self-inflating resuscitator bag (450 and 1000 mL)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Artificial airways</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oropharyngeal airways (sizes 0 through 5)</td>
</tr>
<tr>
<td>Nasopharyngeal airways (12 through 30 French)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Intubation equipment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endotracheal tubes (uncuffed and cuffed, 2.5 through 8.0 mm internal diameter)</td>
</tr>
<tr>
<td>Stylets (infant, pediatric, and adult)</td>
</tr>
<tr>
<td>Laryngoscope handle (pediatric and adult)</td>
</tr>
<tr>
<td>Laryngoscope blades: straight (sizes 0, 1, 2, and 1.5 Wis-Hipple) and curved (sizes 2 and 3)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Rescue airway devices</th>
</tr>
</thead>
<tbody>
<tr>
<td>Laryngeal mask airway (sizes 1, 1.5, 2, 2.5, 3, 4, and 5)</td>
</tr>
<tr>
<td>Combitube (37 and 41 French)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Miscellaneous</th>
</tr>
</thead>
<tbody>
<tr>
<td>End-tidal CO2 detector</td>
</tr>
<tr>
<td>Magill forceps (pediatric and adult)</td>
</tr>
<tr>
<td>Bulb suction</td>
</tr>
</tbody>
</table>
when RSI is utilized. When possible, the resuscitation leader should not be the provider performing the intubation.

2. **Monitoring equipment**: Any patient undergoing ET intubation should be placed on continuous monitoring equipment including heart rate, respiratory rate, blood pressure, and continuous oxygen saturation monitoring. Capnography should be employed when available.

3. **Oxygen supply**: Supplemental oxygen must be available, either from a wall source or a portable tank with a flow meter that allows at least 10 L/min.

4. **Suction**: Wall suction or portable devices should be available. Pressures should be limited to 80 to 120 mmHg to decrease the risk of trauma to airway mucosa. Yankauer or wide-bore tonsil tip catheters are most appropriate for suctioning particulate matter (e.g., thick secretions and vomitus). Flexible suction catheters can be used for thin secretions in the nose, mouth, and hypopharynx, as well as for deep suctioning through the ET tube.

5. **Bag and mask**: Selecting an appropriately sized bag and mask for bag-mask ventilation (BMV) (Table). BMV can provide a temporizing means for oxygenation and ventilation while preparing for intubation in the child in respiratory failure. In addition, children desaturate more quickly than adults during rapid sequence intubation and may require assisted ventilation after administration of sedatives or neuromuscular blockade agents and prior to laryngoscopy and endotracheal intubation (ETI).

Bag-mask ventilation (BMV) is as effective as ETI and ventilation for providing temporary respiratory support. However, BMV does not provide a secure airway and may result in gastric distention, which increases the risk for vomiting and aspiration. Thus, any child requiring prolonged respiratory support is best managed with endotracheal intubation, especially when performed by appropriately trained providers in the emergency department or other critical care settings.

6. **Artificial airways**: Oro- and nasopharyngeal airways should be available to facilitate bag-mask ventilation in case it is necessary during the process of intubation, or in the event that an endotracheal tube cannot be passed successfully.

![Figure 67.2: Oropharyngeal and nasopharyngeal airway](image)
7. **Endotracheal tube**

   **i. Cuffed versus uncuffed**—Anesthesia literature, as well as Pediatric Advanced Life Support Guidelines, now supports that, beyond the newborn period, cuffed endotracheal (ET) tubes are equally as safe as uncuffed tubes, and are favored in some clinical circumstances, such as:
   - Children at risk for aspiration
   - Burn victims
   - Children with severe lung disease who may require high ventilator pressures (e.g., bronchiolitis, status asthmaticus, chronic lung disease)
   When using cuffed tubes, care must be taken to avoid cuff pressures greater than 20 cm H2O, which can increase the risk of tracheal mucosal ischemia. Clinical assessment of cuff pressure is often inaccurate, therefore an ET cuff manometer should be considered in any patient requiring prolonged intubation.

   **ii. Endotracheal tube size**—The size of the ET tube is determined by the internal diameter, measured in millimeters (mm). Available sizes range from 2.5 mm (suitable for a preterm infant) to adult sizes of 7.0 mm or more. The appropriate size ET tube for any given patient should be small enough to pass easily through the vocal cords, but large enough to minimize resistance to airflow. Uncuffed tubes should fit snugly in the subglottic trachea to minimize air leak, while cuffed tubes allow for some adjustment through cuff inflation to provide appropriate endotracheal fit. For uncuffed ET tube sizing, the age-based formula $4 + \left(\frac{\text{age in years}}{4}\right)$ has been shown to be effective and accurate in children. When using a cuffed ET tube, selecting a tube one full size smaller than determined by the above formula was accurate 99 percent of the time, though with the development of newer, lower profile, thinner walled cuffed ET tubes, using a tube one half size smaller than the age-based calculation is recommended (Table 67.3).

   **Table 67.3**: Age-based formula for selecting endotracheal tube size (internal diameter in mm)

<table>
<thead>
<tr>
<th>Uncuffed tube</th>
<th>Cuffed tube</th>
</tr>
</thead>
<tbody>
<tr>
<td>$4 + \left(\frac{\text{age in years}}{4}\right)$</td>
<td>$3.5 + \left(\frac{\text{age in years}}{4}\right)$</td>
</tr>
</tbody>
</table>

Additional tubes one size larger and one size smaller than calculated should also be available.

8. **Stylet**: The clinician should generally use a stylet during emergent endotracheal intubation to reinforce the rigidity of the ET tube and allow the operator to direct the tube into the glottic opening. The largest diameter stylet that fits through the ET tube should be used. Tubes greater than 5.5 mm usually accommodate a larger diameter (i.e., adult) stylet. Two small (pediatric) stylets may improve rigidity in smaller tubes if a single small stylet does not provide significant rigidity. If the stylet does not have a friction reducing
surface coating, the provider should lubricate it with water soluble lubricant to facilitate removal. Bending the styletted ET tube into a hockey stick configuration enhances the ability to direct the ET tube anteriorly through the glottis (Fig. 67.3).

To avoid injury to the tracheal mucosa, care must be taken to ensure that the tip of the stylet does not pass beyond the distal end of the ET tube. Bending the proximal end of the stylet over the adapter at the proximal end of the tube prevents inadvertent movement during intubation.

9. Laryngoscope handle and blade—There are two components to the laryngoscope, the handle and the blade. Pediatric and adult sized handles are available that differ in diameter and length, though either size can be used depending on the clinician's preference. Laryngoscope blades are either curved or straight. The choice of curved or straight blade is best made based on the experience and preference of the laryngoscopist. Curved blades have a large flange which facilitates displacement of the tongue, and a curve that allows easy placement in the vallecula (Fig. 67.4). A straight blade allows direct lifting of the epiglottis to expose the glottic opening, which may be preferred in infants and young children under two years of age in whom the epiglottis is often larger and more acutely angled (Fig. 67.5).

Fig. 67.3: Hockey trick configuration for endotracheal tube versus gentle curve

Fig. 67.4: Direct laryngoscopy using a curved blade
A straight blade may also be preferred in patients in whom cervical spine injury is suspected because laryngoscopy with a straight blade may result in less motion of the cervical spine.

Laryngoscope blades range in size from 00 for the extremely premature infant to 4 for large adults. The appropriate size blade for a given patient is one that is large enough to control the tongue and to reach the glottic structures (Table 67.4). Generally, size 0 or 1 blades are used in average-sized newborns, and size 1 blades for most infants beyond the immediate newborn period. The Wis-Hipple is available in a 1.5 size, which is convenient for children one to three years of age. The phrase “switch to size 2 at age two (years)” helps to remember this important changeover point for laryngoscope blade sizing.

Anatomic landmarks also help identify the appropriate laryngoscope blade size. In a prospective observational study, intubation was more consistently successful on the first attempt when the length of the blade used for laryngoscopy was within one centimeter of the distance between the upper incisors and the angle of the mandible.
**Step 4: Confirm tube placement**

**Confirmation devices**

1. Colorimetric end-tidal CO2 devices or capnographic monitors should be available for ET tube placement confirmation in any setting in which intubation is performed and are the most accurate means for confirming tracheal intubation in patients who are not in cardiac arrest.

   Disposable qualitative devices use colorimetric methods to detect CO2 in the ET tube during the exhalation phase of positive pressure ventilation. Once the trachea is intubated and the colorimetric detector is attached, six positive pressure breaths are delivered. The device will change color (typically from purple to yellow) during exhalation when CO2 is present. This confirms placement of the ET tube in the tracheobronchial tree if the patient has a perfusing cardiac rhythm.

   Capnography confirms ventilation by producing a continuous tracing of CO2 levels. The presence of a regular waveform indicates successful ventilation. It is the most accurate method for confirming ET tube placement.

2. In patients in cardiac arrest, gas exchange in the lungs is markedly reduced and CO2 may not be detectable, despite proper positioning of the ET tube. In such situations, an esophageal bulb may be used to confirm tracheal placement in children who weigh more than 20 kg. It relies on the principle that the esophagus is collapsible under negative pressure, whereas the trachea (which is rigid) is not. The bulb is deflated and then placed on the

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**Table 67.4: Equipment sizing**

<table>
<thead>
<tr>
<th>Age</th>
<th>Blade size and type</th>
<th>Uncuffed tube size (mm ID)</th>
<th>Cuffed tube size (mm ID)</th>
<th>Stylet size</th>
<th>Insertion depth (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Newborn</td>
<td>1 straight</td>
<td>3.3-3.5</td>
<td>N/A</td>
<td>Pediatric</td>
<td>9-10</td>
</tr>
<tr>
<td>6 months</td>
<td>1 straight</td>
<td>3.5-4.0</td>
<td>3.0-3.5</td>
<td>Pediatric</td>
<td>11-12</td>
</tr>
<tr>
<td>1 years</td>
<td>1.5-2 straight</td>
<td>4.5</td>
<td>4.0</td>
<td>Pediatric</td>
<td>12</td>
</tr>
<tr>
<td>2 years</td>
<td>2 straight or curved</td>
<td>5.0</td>
<td>4.5</td>
<td>Pediatric x 2*</td>
<td>14-15</td>
</tr>
<tr>
<td>5 years</td>
<td>2 straight or curved</td>
<td>6.0</td>
<td>5.5</td>
<td>Adult</td>
<td>16-18</td>
</tr>
<tr>
<td>8 years</td>
<td>2-3 straight or curved</td>
<td>6.5</td>
<td>6.0</td>
<td>Adult</td>
<td>18-20</td>
</tr>
<tr>
<td>10 years</td>
<td>2-3 straight or curved</td>
<td>7.0</td>
<td>6.5</td>
<td>Adult</td>
<td>20-21</td>
</tr>
<tr>
<td>12 years</td>
<td>3 straight or curved</td>
<td>7.0</td>
<td>6.5</td>
<td>Adult</td>
<td>20-21</td>
</tr>
</tbody>
</table>

mm ID: internal diameter in mm.

* Use two pediatric stylets in children of this size to ensure adequate endotracheal tube stiffness.
end of the ET tube following intubation. The bulb will remain deflated when the ET tube is in the esophagus, but will reinflate with gas from the trachea and lungs when the ET tube is correctly positioned in the non-collapsible trachea.

3. Alternative airway supplies: Alternative strategies and appropriate equipment for providing oxygenation and ventilation must be considered for the child who may be difficult to intubate with direct laryngoscopy. These techniques may be temporizing (such as laryngeal mask airway, Combitube, or a surgical airway) or provide alternative approaches to tracheal intubation (as with fiberoptic intubation, gum elastic bougie, a lighted stylet, or a video laryngoscope.

4. Miscellaneous supplies:
- Tape or a commercial holder to secure the endotracheal tube
- Tincture of benzoin to enhance the holding power of the tape
- Gauze or cotton-tipped applicator for benzoin application
- 5 to 10 mL syringe for cuff inflation
- A nasogastric or orogastric tube to decompress the stomach following intubation. Insufflated air from BMV or residual gastric contents should be removed to decrease the risk of aspiration and improve diaphragmatic excursion.

Procedure

Direct laryngoscopy and endotracheal intubation are complex processes. Developing a systematic and reproducible approach to this procedure will improve success.

1. Monitoring: Continuous cardiorespiratory monitoring and pulse oximetry prior to intubation are essential. Capnography should be utilized to confirm and monitor endotracheal tube position after intubation.

2. Preoxygenation: Preoxygenation with 100 percent inspired oxygen creates an oxygen reservoir, primarily by washing nitrogen out of the functional residual capacity of the lungs and replacing it with oxygen.

3. Suction: Two suction devices (e.g., Yankauer or wide-bore tonsil tip catheters) should be immediately available at the bedside and attached to a wall unit suction that is turned on and limited to a maximum of 120 mmHg.

4. Positioning: Proper positioning aligns the pharyngeal, tracheal, and oral axes into the “sniffing position” (Fig. 67.6).

A. To align the pharyngeal and tracheal axes, the chin is moved anteriorly with respect to the shoulders, such that the external auditory canal is anterior to the shoulder. This may be accomplished in children by placing a towel or roll under the occiput. In infants, because of a prominent occiput, the towel must be placed under the shoulders to achieve this position.

B. To align the oral axis with the pharyngeal and tracheal axes, the head is then extended on the neck, such that the nose and mouth are pointing
toward the ceiling. This may be accomplished by placing the palm of the right hand on the patient's forehead with the fingers extending onto the occiput, cupping the head and gently rotating the head posteriorly (Fig. 7). This maneuver also opens the patient's mouth, facilitating insertion of the laryngoscope.

**Figs 67.6A to C:** Proper positioning for ventilation and intubation for children older than two years of age: (A) The oral (O), tracheal (T), and pharyngeal (P) axes are in divergent planes; (B) A towel under the occiput brings the external auditory canal anterior to the shoulder, aligning the T and P axes; (C) The extension of the head on the neck, with the mouth and nose facing the ceiling, aligns the O axis with the T and P axes.

**Fig. 67.7:** Endotracheal intubation position in a child- Positioning the head and neck for insertion of the laryngoscope blade. Posterior pressure on the forehead extends the neck and usually causes the mouth to open.
C. Cervical spine immobilization—For the child with a suspected cervical spine injury, neck movement must be minimal during positioning and laryngoscopy. Initially, the airway can be opened with the jaw thrust maneuver (Fig. 67.8). If a cervical collar is in place, the front should be opened to allow complete mouth opening and displacement of the chin and mandible. Manual in-line stabilization should be maintained by an assistant during laryngoscopy and intubation (Fig. 67.9).

5. **Sedation and neuromuscular blockade**: Rapid sequence intubation (RSI) typically achieves optimal conditions for laryngoscopy in children requiring emergent intubation (Table 67.1). RSI involves the delivery of a sedative and neuromuscular blocking agent to sedate and pharmacologically paralyze so that movement and protective airway reflexes do not interfere with endotracheal intubation. RSI may be modified in the following circumstances:

![Figure 67.8: Jaw thrust maneuver](image)

![Figure 67.9: Manual stabilization of the pediatric spine](image)
i. Sedative agents may be omitted in obtunded or comatose patients.

ii. Neuromuscular blockade should be avoided in patients with a predicted difficult airway unless a back-up approach is available.

iii. Once rapid sequence intubation medications are provided, the clinician should make every effort to avoid BMV because of the increased risk of vomiting and aspiration that can occur with gastric distention. However, in patients who cannot be adequately preoxygenated, BMV with small tidal volumes and cricoid pressure is preferable to intubating a hypoxic patient. Nasal cannula oxygen delivery to the apneic patient following administration of RSI medications can also decrease the likelihood of hypoxemia.

6. **Cricoid pressure**: Cricoid pressure has historically been used in rapid sequence intubation to prevent gastric insufflation and passive regurgitation of gastric contents. In this technique, often referred to as the Sellick maneuver, the thumb and fore or middle finger are used to apply pressure over the anterior neck at the cricoid cartilage to compress the esophagus between the cricoid cartilage and the anterior surface of the C6 vertebral body (Figure 67.10).

   Increased pressure should be applied after the sedative is administered and prior to complete neuromuscular blockade. It should be maintained until endotracheal tube position is confirmed. Pediatric data exist to suggest that cricoid pressure may decrease the risk of gastric insufflation; however, conflicting evidence exists regarding the effectiveness of cricoid pressure for preventing regurgitation. Therefore, cricoid pressure may be used initially with RSI, but should be removed if airway obstruction occurs when ventilation is required or if there is difficulty viewing the larynx.

7. **Laryngoscopy**: Laryngeal exposure with visualization of the glottis is the main determinant of success or failure with endotracheal intubation (ETI).

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**Figure 67.10**: Cricoid pressure (Sellick’s maneuver)—Posterior displacement of the airway cartilages occludes the compliant esophagus. In infants and young children, the tracheal cartilage is also very compliant and excessive force while applying cricoid pressure may impair airway patency.
Direct laryngoscopy is most easily performed with the clinician standing at the patient's head, and the bed adjusted to the level of the laryngoscopist's xiphoid. The endotracheal (ET) tube, with stylet in place, and suction equipment should be easily accessible. ET tubes that are one size above and below the estimated size for age should also be available. Whenever possible, an assistant should stand to the right of the patient's head to assist with optimal positioning and to hand items to the intubator. Once the child is completely relaxed, the following steps are performed:

i. **Opening the mouth**—The mouth is opened using either of two techniques: a scissor technique in which the thumb of the operator's right hand pushes the lower incisors (or mandibular gum) caudad while the index finger (placed posterior to the thumb) pushes the upper incisors (or maxillary gum) cephalad (Fig. 67.11), or in a patient without cervical spine restrictions, extension of the head will naturally open the mouth. This can be augmented by applying caudal pressure on the chin using the fifth finger of the left hand.

ii. **Inserting the laryngoscope**—The laryngoscope is held in the left hand, regardless of the practitioner's hand dominance. The most common approach is to insert the blade into the right side of the patient's mouth, taking care not to catch the lower lip against the teeth, which may lacerate the lip. Within the oral cavity, the blade is passed under direct visualization along the base of the tongue following the natural contour of the pharynx. The tongue is swept to the left as the laryngoscope is advanced into midline of the hypopharynx (Fig. 67.12). An alternative

![Fig. 67.11: Scissor technique for opening the mouth during endotracheal intubation](image)
approach is to pass the blade down the midline. The advantage of this method is that it may avoid the blade getting caught on pharyngeal folds and may also allow for easier identification of recognizable anatomic structures such as the epiglottis.

iii. Retracting the tongue and soft tissues—Once in the midline of the oropharynx, the laryngoscope blade should be used to lift the mandibular block. This can be accomplished by applying force away from the laryngoscopist along the long axis of the handle. The laryngoscope should not be “rocked” backward, using the upper palate or incisors as the fulcrum for leverage. This improper technique will decrease the space within the oral cavity, making it difficult to pass the ET tube under direct visualization. In addition, injury to the mouth, gingiva, or teeth can occur when the blade is levered against these structures. Keeping the wrist straight will help prevent inadvertent levering.

iv. Identifying glottic structures—As the laryngoscope blade is advanced into the pharynx, the epiglottis will often come immediately into view (Fig. 67.13). This is the landmark that is most useful when identifying the remainder of the glottic structures. At this point, suction is often needed to remove saliva, blood, or debris and optimize the glottic view.

Figs 67.12A to C: Tongue sweep during laryngoscopy—Tongue control during laryngoscopy: (A and B) Poor visualization of the cords due to incorrect positioning of the blade. Note how the tongue folds over the blade and obscures the view; (C) Correct positioning of the blade to control and move the tongue to the left, providing an optimal view for intubation

Fig. 67.13: Laryngeal inlet
A number of laryngoscopic adjustments can be made if the epiglottis is not seen immediately:
- The epiglottis may be lying flat against the posterior pharyngeal wall or folded on itself, making it difficult to distinguish from surrounding mucosal surfaces. Additional elevation of the mandibular block may help separate the epiglottic rim from surrounding tissue.
- The laryngoscope blade may not be midline, often as a result of challenges in sweeping the tongue from right to left. Repositioning the blade using the uvula as a midline reference point may be helpful.
- If neither the epiglottis nor the glottic structures are visible, the laryngoscope blade can be advanced fully, placing the blade tip in the esophagus. The blade is then pulled back slowly. The first structure to fall into view will be the glottis, followed by the epiglottis. Some experts recommend routine use of this approach with blind insertion beyond the larynx and locating identifiable structures as the blade is withdrawn, particularly when intubating infants.

v. **Elevating the epiglottis**—After the epiglottis has been identified, it needs to be elevated to expose the underlying vocal cords and glottic opening. The technique employed varies based on the type of blade being used.

When using a straight blade, the tip of the blade is positioned under the epiglottis to lift it directly (Figs 67.4 and 5). The epiglottis is frequently large, floppy, and covered with airway secretions and therefore may easily slide off the blade. If this occurs, the blade should be repositioned beneath the epiglottis and it should be carefully lifted again.

When using a curved blade, the tip of the blade is pressed against the deepest portion of the vallecula to place tension on the hyoepiglottic ligament, which will help suspend the epiglottis. Once the blade (straight or curved) is positioned correctly, force is applied upward and forward along the long axis of the laryngoscope handle at approximately 45 degrees. This will lift the mandibular block and the epiglottis to expose the glottic opening.

vi **Adjusting for suboptimal view**—Ideally, with appropriate positioning and laryngoscopy, the vocal cords and glottic aperture will be quickly identified. If little or none of the glottic opening is visualized, external laryngeal manipulation (ELM) may improve the view. An assistant can be asked to adjust cricoid pressure (if already being utilized) or to apply backward-upward-rightward pressure (BURP) to the larynx.

8. **Passing the endotracheal tube**: Once the glottic opening has been identified, the final step is passage of the endotracheal (ET) tube. While maintaining a view of the glottic opening, the intubator receives the tube in his/her right hand from a previously assigned assistant. The tube is held like a pencil, between the thumb and first two fingers.
The tube enters the right side of the mouth and is advanced toward the larynx in a horizontal plane. Passage of the tube directly along the barrel of the laryngoscope blade will obscure the view of the glottic opening, and should be avoided. The ET tube should be passed through the vocal cords under direct visualization. Once the tip has passed through the vocal cords, the tube is rotated to the upright position. Although the tendency is for the intubator to move closer to the patient to improve view, this may compromise binocular vision and depth perception.

9. **Depth of insertion:** The ideal location for the endotracheal (ET) tube tip is at the midpoint between the thoracic inlet and the carina. There are a number of ways to guide the proper depth of insertion for the ET tube:
   - Placing the double line on the ET tube at the glottis
   - Using the depth provided by the length-based resuscitation tape
   - Inserting the tube until the centimeter marking at the lip is three times the internal diameter of the ET tube
   - This last calculation will result in the ET tube being correctly positioned more than 80 percent of the time, when using an appropriately size ET tube.
   - Additional techniques, less commonly used during emergent intubation, include deliberately advancing the ET tube to create an endobronchial intubation and then withdrawing the tube 2 cm beyond the passage of the carina, and palpation of the tube tip at the suprasternal notch.

10. **Initiate positive pressure ventilation:** The laryngoscope can now be removed from the mouth while the tube is held securely against the roof of the mouth, or by grasping the tube using the index finger and thumb with the remaining three fingers holding the patient’s face. If a cuffed tube is being used, the cuff should be inflated at this time.
   - Positive pressure ventilation should be initiated with 100 percent inspired oxygen via a resuscitation bag with a carbon dioxide detector attached to the endotracheal (ET) tube. The bag should initially be squeezed with enough force and volume to provide chest wall movement.
   - Subsequent ventilatory strategies can be made based on noninvasive measures of oxygenation and ventilation or based on results of blood gas analyses. If a large air leak is noted at this time, cuff inflation may be adjusted accordingly. However, persistent leak may require the tube to be changed to a larger size.
   - If an uncuffed ET tube is in place, then the air leak pressures should ideally occur at less than 25 cm H2O, while still allowing for effective ventilation. Air leak pressures up to 40 cm H2O have been shown to be safe.

11. **Confirming tube position —** Immediately following intubation, placement of the endotracheal (ET) tube in the trachea must be confirmed. Clinical assessment for appropriate tube position includes:
   - Visible chest wall rise
   - Auscultation of breath sounds in both axillae and not heard over the stomach
Continuous pulse oximetry should confirm adequate oxygenation.
Mist should be present in the ET tube.
However, because clinical evaluation is not completely accurate, confirmatory devices should be used.
End-tidal CO₂ should be detected using either a colorimetric device or capnography and is the most definitive method of confirming that the ET tube is in the trachea.
A self-inflating bulb may also be used for children weighing more than 20 kg, and may be particularly useful for confirming tube position in patients in cardiac arrest.

12. **Securing the tube:** After correct tube position is confirmed, cricoid pressure can be released if it had been utilized during the intubation. The ET tube must be firmly secured. The most common approach is to tear longitudinally down the midline of a length of cloth or silk tape, creating a Y-shape. One segment is wrapped around the tube and the base segment is placed across the cheek. Preparing the underlying skin with a layer of benzoin, which is allowed to air dry, can provide additional adherence. Alternatively, commercial tracheal tube holders may be utilized to secure the ETT. These devices have been shown to be rapidly applied in adults and to have reasonable resistance to extubation forces, although in most instances, tape is stronger. However, commercial endotracheal tube holders have not been specifically studied in infants and young children.

**Step 5: Post-intubation Care**

1. **Post-intubation imaging:** An anterior-posterior chest radiograph should be obtained to confirm the location of the tip of the ET tube. Optimal position is located at a minimum of one to three centimeters above the carina and below the thoracic inlet. Tube depth may be adjusted based on radiographic position. Preliminary evidence suggests that bedside ultrasound, performed by clinicians experienced with this technique, may also be useful for directly determining the position of the ET tube within the trachea, or confirming appropriate position by documenting bilateral lung sliding. However, this is not common practice at this time and should not replace radiography.

2. **Gastric decompression:** An orogastric or nasogastric tube should be placed following intubation to decompress the stomach. Gastric distension can occur secondary to crying or insufflation following BMV. In addition, because emergent intubation may be performed on non-fasted patients, residual gastric contents may be present and should be evacuated. Gastric decompression can decrease the risk of aspiration around the ETT, as well as improve diaphragmatic excursion and patient ventilation.

3. **Minimize head movement:** Care must be taken to avoid significant head movement in patients who have been intubated. Flexion of the neck may result in the tube advancing into an endobronchial position with resultant
limited ventilation of one lung, while neck extension can lead to unintended extubation.

4. **Positive pressure ventilation**: Ventilation strategies vary based on underlying disease process, whether ongoing sedation and neuromuscular blockade is needed, and subsequent management plans.

**Step 6: Complications**

Acute complications from laryngoscopy and intubation can occur at multiple points during the procedure.

1. **Gastric distension**: BMV ventilation may cause gastric distension, leading to diminished lung capacity and increased risk of regurgitation.

2. **Hypoxemia**: Sustained periods of inadequate oxygen delivery may lead to ischemic brain injury, the most significant complication of ET intubation. Inadequate preoxygenation will shorten safe apnea time. Prolonged laryngoscopy (even with adequate preoxygenation) will lead to hypoxemia. Monitoring via continuous pulse oximetry is paramount to recognizing inadequate oxygenation. If desaturation occurs, the intubation attempt should be discontinued. Before another attempt is initiated, the patient should receive BMV until oxygen saturations improve.

3. **Bradycardia**: Profound bradycardia can occur during laryngoscopy and intubation as follows:
   - Vagal response from stimulation of the hypopharynx, lifting the epiglottis, or rarely, the use of succinylcholine can lead to bradycardia, particularly in infants and young children.
   - Hypoxemia can also result in secondary bradycardia in infants.
   - Atropine may help prevent vagal mediated bradycardia but will be ineffective in cases of hypoxemia. Recommendations support prophylactic use of atropine in infants and young children under five years of age receiving succinylcholine as well as older children who are receiving a second dose of succinylcholine.

4. **Increased intracranial pressure (ICP)**: Intracranial pressure may increase during laryngoscopy as a result of increased cerebral arterial pressure during laryngoscopy.

5. Increases in ICP are of paramount importance in patients who already have elevated pressures. Adequate use of sedatives and premedication may help attenuate increases in pressure.

6. **Mechanical trauma**: Soft tissue injury can occur anywhere along the extrathoracic airway.

7. **Aspiration**: Aspiration of oral or gastric contents during laryngoscopy or intubation can occur. The severity of any subsequent pneumonitis is related to the acidity, volume, and presence of particulate matter in the aspirated material.
8. **Tube obstruction**: Obstruction of an ET tube can occur when the tube tip is against a mucosal surface, from intraluminal inspissated secretions, or if the tube kinks. Inadequate air flow can result in hypoxemia and hypercapnia.

9. **Barotrauma**: By definition, positive pressure ventilation puts patients at risk for pulmonary barotrauma, including pneumomediastinum and pneumothorax.

10. **Post-obstructive pulmonary edema**: When intubation is performed to relieve upper airway obstruction, the resultant changes in intrathoracic pressure can lead to pulmonary edema.

11. **Adverse events from medications**: Complications related to medications for sedation and neuromuscular blockade may also occur. This may include medication-specific adverse reactions as well as complications from dosing outside the therapeutic window.

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**Central Line Placement**

**Introduction**

Central venous cannulation (CVC) involves percutaneous placement of a vascular catheter with its tip in the lumen of a major, high-flow vein of the abdomen or thorax. It remains a cornerstone of resuscitation and critical care in casualty and pediatric intensive care unit. Advanced hemodynamic monitoring, rapid fluid infusion, parenteral nutrition, and selected multiple medications all require reliable central venous access. Promising advances in this technique, most notably real-time ultrasound guidance, have emerged since the early 2000s, which have improved success rates and decreased complication rates.

**Step Wise Approach**

**Indication for Central Line Access**

**Therapeutic**

- Appropriate fluid management (shock, intra-operative)
- Difficult peripheral vascular access
- Administration of vasoactive medications and multiple infusions.
- Administration of hypertonic fluids (parenteral nutrition, chemotherapy) or concentrated electrolytes (potassium)
- Access for Swan-Ganz catheter or pacemaker placement Long-term vascular access
- For procedures like apheresis, hemofiltration and hemodialysis

**Diagnostic**

- Measurement of venous vascular pressures (CVP)
- Measurement of mixed venous blood gases (ScvO2)
- Repeated blood sampling
Rule Out Contraindications

There are no absolute contraindications, but relative contraindications being:

**General:** Local site infection, burns, distorted local anatomy, suspected proximal vascular injury

**Subclavian vein:** Avoid in bleeding or clotting disorders as local pressure application is not possible to stop bleeding. Chest wall deformities and pneumothorax on contra-lateral side are other relative contraindications.

**Femoral vein:** hamper patients movement.

appropriate Site Selection

Assess patient’s requirement and co-morbid conditions.

Coagulopathy: prefer femoral vein > IJV (internal jugular vein)>subclavian vein

To decrease infection risk: prefer subclavian vein > IJV>femoral vein

TPN: prefer subclavian vein or IJV

CVP and ScvO2 Monitoring: prefer subclavian vein or IJV.

Appropriate Catheter Selection:

a. Single lumen or multi-lumen catheters:
   - More the number of lumens, more is the risk of infection associated with it
   - More the number of lumens, smaller becomes the diameter
   - If rapid infusion is required, as in trauma – single or double lumen preferred.
   - If number of infusions is more, three or four lumen catheters are preferred.

b. *Antimicrobial-impregnated catheters:* Use of chlorhexidine/silver sulfadiazine or minocycline/rifampicin impregnated CVC is used in that PICU settings where catheter related infection is high despite implementation of comprehensive preventive measures to reduce rate of infections.

c. Size selection (approximation, no fixed rule)

<table>
<thead>
<tr>
<th>Age (yrs)</th>
<th>IJ</th>
<th>SC</th>
<th>Femoral</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–0.5</td>
<td>3F</td>
<td>3F</td>
<td>3F</td>
</tr>
<tr>
<td>0.5–2</td>
<td>3F</td>
<td>3F</td>
<td>3–4F</td>
</tr>
<tr>
<td>3–6</td>
<td>4–5F</td>
<td>4–5F</td>
<td>4–5F</td>
</tr>
<tr>
<td>7–12</td>
<td>4–7F</td>
<td>4–7F</td>
<td>5–8F</td>
</tr>
</tbody>
</table>

**Right IJ and Right SC Depth of insertion:**

If Height < 100cm then Initial Catheter Depth (cm) = Ht (cm)/10 -1 cm

If Height > 100 cm then Initial Catheter Depth (cm) = Ht (cm)/10 -2 cm

These formulas will place 98% of catheters above R atrium, but insertion point can change the outcome. Therefore this is just an approximation, not fixed. Many new calculations are available in many indexed journals, but all are just an approximation to guide us.
**Know the Anatomy**

Three sites are commonly used for pediatric CVC placement: femoral, internal jugular, and subclavian.

a. **Subclavian vein**: After crossing the first rib, the vein lies posterior to the medial third of the clavicle. Posterior to the vein, separating it from the subclavian artery lays the anterior scalene muscle. The vein lies in close proximity to dome of pleura. In infants, anatomic factors make subclavian vein entry less attractive than entry at other sites as the subclavian vein arches more superiorly as it courses toward the atrium, resulting in acute angles that may obstruct catheter placement. The subclavian arch takes on the more typical horizontal position within the chest only after 1 year of age. Additionally, in young infants, the notch on the inferior aspect of the first rib, a landmark commonly employed to identify the percutaneous entry site for the subclavian vein, is difficult to identify. Finally, the diameter of the subclavian vein is smaller than that of the internal jugular vein in this age group. ([Fig. 67.14A](#))

b. **Internal jugular vein**: The traditional approach to IJ vein cannulation uses external anatomic structures to locate the vein. A common approach identifies a sedillot’s triangle subtended by the two heads of the sternocleidomastoid muscle and the clavicle. A needle placed at the apex of this triangle and directed toward the ipsilateral nipple should encounter the IJ usually 1.5 cm beneath the skin surface. ([Fig. 67.14B](#))

c. **Femoral vein**: Important landmark is the femoral artery pulse, because the common FV typically lies medial to the common femoral artery within the femoral sheath. The femoral artery lies at the midpoint of the inguinal ligament connecting the anterior superior iliac spine to the pubic tubercle, while the common FV is typically located medial to the common femoral artery. This side-by-side relationship of the common femoral artery and FV occurs in close proximity to the inguinal ligament, but significant vessel overlap may occur, particularly in children. ([Fig. 67.14C](#))

**Take Informed Consent**

**Keep Equipment Ready**

a. Aseptic technique - povidone-iodine or chlorhexidine solution, sterile gauze, sterile towels, drapes, sterile gloves, sterile gowns, masks

b. Catheter kit with intravascular catheter, finder needle, flexible guidewire (straight or j-tip; make sure it fits the catheter), saline solution syringes, 3 or 5 ml, silk suture (3.0 or 4.0), needle holder

c. Monitoring and other equipment - pulse oximeter, cardiac monitor, blood pressure monitor, capnometer, handheld ultrasound device

d. Medications - lidocaine 1% (without epinephrine), midazolam 0.1 mg/kg, and morphine sulfate 0.1 to 0.15 mg/kg, oxygen, suction, airway.
Set up Pressure Transducing System

Central Line Placement

- Wear cap and mask
- Wash hands with chlorhexidine based soap solution for 3-5 minutes
- Put on sterile gown and gloves – maximum sterile barrier
- Positioning of patient – shoulder roll and Trendelenburg position with head facing other side for subclavian and IJV while hip roll with legs slightly abducted and externally rotated for femoral.
- Clean the skin of patient with chlorhexidine in alcohol solution for children > 2 months of age, else use povidine-iodine, alcohol.
- Give a frictional scrub in a circular manner from inner outside.
- Place large sterile drape over insertion site. Do not occlude air supply of patient.
- Use catheter-over-the-wire (Seldinger) technique.
  - The central vein is first punctured using a needle and syringe.
  - The syringe is removed and the guidewire is inserted through the needle into the vein.
  - The needle is removed and the wire is left in place.
  - A small incision is made at the entry site to facilitate insertion of the catheter. This step is omitted if child has bleeding issues.
  - This tract is dilated with a dilator in rotating motion
  - The catheter is inserted over the guidewire and the wire is removed
  - Never lose control of guidewire – a part of it should be always visible.

Subclavian Vein Cannulation (Fig. 67.14A)

Place the patient in a 10- to 15-degree Trendelenburg position, with head turned towards opposite side and with a shoulder roll. The ipsilateral arm should be at the child’s side; gentle downward traction of the ipsilateral arm may also be helpful. The operator is positioned at the patient’s side when using the
Procedures in PICU

infraclavicular approach or at the head of the bed when using the supraclavicular approach. The infraclavicular approach is usually preferred, because this method is thought to carry a lower risk of pneumothorax and other complications. (Table 67.5)

**Internal Jugular Vein Cannulation (Fig. 67.14B)**

Internal jugular catheterization can be achieved via multiple approaches. Right-sided approaches are preferred due to potential injury to the thoracic duct on the left side. The carotid artery should be palpated, as it lies medial to the internal jugular vein within the carotid sheath. For all approaches, the patient should be positioned supine and in a slight (15-30 degree) Trendelenburg position, with a roll under the shoulders and with the head turned away from the puncture site. (Table 67.6)

In all approaches, the needle should be advanced during exhalation to minimize the chance of pneumothorax, and the syringe should be aspirated as the needle is advanced. When the vein is entered and free flow of venous blood is established, the needle should be stabilized and the syringe removed while the hub of the needle is covered to prevent air entrainment. Obtain chest radiograph.

**Table 67.5: Approach for subclavian vein cannulation**

<table>
<thead>
<tr>
<th>Insertion Landmark</th>
<th>Infraclavicular (Preferred)</th>
<th>Supraclavicular</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angle with skin</td>
<td>0-5</td>
<td>45</td>
</tr>
<tr>
<td>Aim Toward</td>
<td>Sternal Notch</td>
<td>Contralateral nipple</td>
</tr>
<tr>
<td>Depth from skin</td>
<td>Just deep to clavicle</td>
<td>Just under clavicle</td>
</tr>
</tbody>
</table>

**Table 67.6: Approach for internal jugular vein cannulation**

<table>
<thead>
<tr>
<th>Insertion Landmark</th>
<th>Central (Preferred)</th>
<th>Anterior</th>
<th>Posterior</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angle with skin</td>
<td>30</td>
<td>45</td>
<td>30-45, dive under the border of sternocleidomastoid muscle</td>
</tr>
<tr>
<td>Aim towards</td>
<td>Ipsilateral nipple</td>
<td>Ipsilateral nipple</td>
<td>Sternal notch</td>
</tr>
<tr>
<td>Depth from skin</td>
<td>Within 1-3 cm</td>
<td>Within 1-3 cm</td>
<td>Within 2-4 cm</td>
</tr>
</tbody>
</table>
Femoral Vein Cannulation (Fig. 67.14c)

The lower extremity should be positioned with slight external rotation at the hip and flexion at the knee (frog-leg appearance). A rolled towel under the buttock may facilitate successful venous access, particularly in smaller children. The femoral artery should be located by palpation or ultrasound or, in the pulseless patient, assumed to be at the midpoint between the pubic symphysis and anterior superior iliac spine. The area over the intended puncture site should be infiltrated with local anesthetic. The needle should be inserted 1-2 cm below the inguinal ligament, just medial to the femoral artery, and slowly advanced while negative pressure is applied to a syringe attached to the introducer needle. The needle should be directed at a 15-45 degree angle toward the umbilicus, depending on the size of the child, with a flatter approach used in infants than in older children. Once the free flow of venous blood is observed, the syringe should be removed while the needle is carefully stabilized and the guidewire is introduced gently.

Ultrasound Use for Vascular Access

Advantages:
- Fewer complications
- Fewer attempts for successful cannulation
- Fewer failed procedures
- Used in difficult access, obesity, short neck, in patients with coagulopathy

Disadvantage:
- Cost and maintenance of equipment
- Special USG training
- Difficulty to maintain sterility during procedure.

Transducer Selection

- B mode for vascular anatomy with high frequency transducer (5-7 Hz)

Technique

A. Static guidance suggests that USG has been placed over the anatomic area, and the area is marked while angle and distance information is noted.
B. Dynamic guidance describes procedure performed with real-time US visualization of the needle entering the anatomic area.

View

A. Transverse, where the vein appears as a circular structure on the screen (Figure 67.15)
B. Longitudinal, where the vein appears as a tubular structure along the width of the screen. (Figure 67.16)
Procedure IJV

The patient is positioned appropriately - trendelenberg.

The head should be rotated slightly contra-laterally, with the neck extended.

The ultrasound machine should be placed by the ipsilateral side of the bed

The patient’s skin can now be prepped in sterile fashion and full barrier precautions

A sterile ultrasound sheath should be placed on the sterile field for when an assistant hands the operator the ultrasound transducer.

After the patient is prepped and draped, the catheter is set up per normal routine.

All ports should be flushed with bacteriostatic saline to remove air and to test for occlusion caused by manufacturing defects.
The operator acquires the transducer, places it in the sterile cover, and secures it on the sterile field.

The transducer can either be “picked up” by the operator whose gloved and sterile hand is inside the transducer cover like a puppet or, alternatively, an assistant can insert the transducer in the open end of the cover. The end sheath is then extended to cover the transducer cord.

Confirm vessel location with USG and reconfirm orientation of probe with help of identification mark on probe or alternatively finger can be rubbed on side of transducer surface to produce an image.

Can apply chlorhexidine over transducer to make image clear

Ensure the vessel to be punctured is in centre of screen, so that the vessel is lying just deep to centre of transducer. (Figure 67.152b)

A mock poke can be done by applying pressure and visualising acoustic disturbance in subcutaneous tissue, to ensure proper placement of your needle (Figure 67.15c).

The skin puncture should be proximal to the transducer, which in most cases will result in visualization of the needle tip entering the vessel without having to move the probe much. If the needle tip cannot be visualized, the operator moves the probe along the axis of the vessel while slightly “agitating” the needle; this will accentuate the image of the needle and tip.

Needle tip should always be visualized

If done properly, the needle tip should be seen entering the lumen at about the same time as the flash of blood is obtained in the syringe.

Once the vessel has been successfully cannulated, the operator sets aside the transducer and proceeds with wire placement. Intravascular position of the wire can be confirmed with ultrasound.

Examine with probe for pneumothorax – look for sliding pleura.

**Confirm Central Line Location and Documentation of Procedure**

Routine chest radiograph is recommended for IJV and subclavian line.

Ideally the catheter tip should lie within superior vena cava, parallel to vessel wall, just outside right atrium or cavo-atrial junction. Rule out pneumothorax.

For femoral line – routine check x ray is not recommended, but if abdominal radiograph is taken, make sure catheter has not been advanced retrograde to the veins draining major organs (e.g., renal, hepatic, or jugular veins). Furthermore, at no time should a catheter tip be allowed to remain within the right atrium. Atrial wall perforation with hemopericardium is a rare but catastrophic CVC complication.

**Port Designation (for Multi-lumen Catheters)**

The ports of a multiple lumen central venous catheter should be labelled for designated use, and the information should be entered into the patient’s information sheet. There is a lack of scientific data to support many protocols.
for the specific use of ports. Most choices have been made using deductive reasoning and the following designations are examples for port usage and in no way represent the only way the lumens can be used:

a. Proximal
   - Blood Sampling
   - Medications
   - Blood Administration. The proximal port is often designated for blood sampling. This choice is made because the rapid flow of blood within the large central vein quickly carries the infusates from the more distal lumens, that might affect laboratory tests, away from the proximal sampling port. As an additional safeguard against erroneous lab results, it is recommended that all other infusions be turned off prior to blood sampling.

b. Medial
   - Parenteral Nutrition
   - Medications (only if TPN use is not anticipated). Another designation that has gained widespread acceptance is the need to reserve one lumen exclusively for total parenteral nutrition (TPN). The rationale for this designation is the prevention of catheter-related infections. When using a triple lumen catheter, the middle port is often chosen.

c. Distal:
   - CVP Monitoring
   - Blood Administration
   - High Volume or Colloids. The distal port is usually used for central venous pressure monitoring. The reasons given for this choice is that the distal lumen is the largest lumen and it is closest to the heart

*Catheter Dressing*
Acceptable dressings include transparent polyurethane adhesive dressings and standard gauze and tape dressings. The transparent dressings help secure the device, permit continuous visual inspection of the catheter site, permit careful bathing, and require less frequent changes. If blood is oozing from the catheter insertion site, gauze dressing might be preferred.

Replace catheter site dressing if the dressing becomes damp, loosened, or visibly soiled.

Do not use topical antibiotic ointment or creams on insertion sites (except when using dialysis catheters) because of their potential to promote fungal infections and antimicrobial resistance

*Complications*
Major noninfectious complications include pneumothorax, arterial puncture, and thromboembolism. The most important factors related to all complications
associated with this procedure are operator technique during placement and subsequent care.

Removal of Line

- As soon as it is not required
- Induration, frank pus from insertion site
- Confirmed CRBSI
- Catheter occlusion/thrombosis

Steps to Minimize CVC-Related Infection

a. Site preparation with approved chlorhexidine-based preparation, unless contraindicated
b. Maximal barrier precautions during catheter insertion
c. Use of mobile procedure carts, safety checklist, empowerment of staff
d. Strict protocols for catheter maintenance (including bandage and tubing changes)
e. Appropriate site selection, avoiding heavily colonized areas
f. For anticipated duration of catheterization exceeding 96 hours, use of silver-impregnated cuff, sustained release chlorhexidine gluconate patch, and/or antibiotic/antiseptic-impregnated catheters in those PICU where catheter-related infection is not controlled despite all preventive measures
g. Prompt removal of any catheter which is no longer required
h. Replace any catheter not placed with sterile precautions within 48 hours (i.e., catheter placed in emergency)
i. Use multilumen catheters only when indicated; remove when no longer needed
j. Avoid routine guidewire exchanges

Arterial Line Placement

Introduction

Arterial access is frequently used in the care of critically ill infants and children for arterial blood gas and other blood sampling, as well as continuous blood pressure monitoring. Arterial catheterization in children is technically complex and should be performed by health care providers specifically trained in the technique, usually physicians.

Conventions to Remember While Measuring Cardiovascular Pressures

- All pressures are expressed as millimeters of mercury, except CVP, which may be expressed as cmH2O. 1 mmHg = 1.36 cmH2O
- All cardiovascular pressures are referenced to the heart, in what is known as the phlebostatic axis or plane.
- All pressures, because the transducer is zeroed to atmosphere before measurement, are pressures above the ambient atmospheric pressure
Components of the System used for Measurement

- An intravascular catheter to access the patient’s blood vessels.
- Connecting tubing and stopcocks to connect the intravascular catheter and patient’s blood vessels to the monitoring system.
- A pressure transducer to convert the mechanical impulse of a pressure wave into an electrical signal through movement of a displaceable sensing diaphragm. Modern transducers use a silicon wafer as the diaphragm, which is a stiff, low compliance pressure sensing device capable of bending and creating a small volume change in response to applied pressure. The transducers function on the principle of the Wheatstone bridge.
- A continuous flush device fills the pressure tubing with fluid and helps prevent blood from clotting in the catheter by continuously flushing fluid through the system at a rate of 1-3 ml/hr. by keeping a flush bag at pressure of 300 mmHg. While some unit protocols heparinise the flush bag to prevent cannula thrombosis, many have concluded that this is unnecessary.
- An amplifier increases the low voltage signal from the pressure transducer (usually measured in micro volts) to a signal that can be displayed on an oscilloscope or other display device. Most amplifiers include electronic filters to filter out unwanted physiologic “noise”.
- An oscilloscope (to display waveforms) and a digital readout (to display numerical data).
- A processor or microcomputer which is used to calculate various haemodynamic parameters based upon the measured variables.
- A recorder – a printer, strip chart recorder, or other device which allows a paper record of the data measured to be saved.

Indications for Arterial Line Placement

- Hemodynamic monitoring
  - Acutely hypertensive or hypotensive patients
  - Continuous cardiac output monitoring
  - Use of vasoactive drugs
- Multiple blood sampling
  - Ventilated patients
  - Limited venous access
- Intraaortic balloon pump use

Check for Relative Contra-indications

- Extremities with full thickness burns/trauma
- Skin infection over insertion site
- Uncontrolled Coagulopathy

Site Selection

- The ideal artery has extensive collateral circulation that will maintain the viability of distal tissues if thrombosis occurs.
The most commonly used sites for arterial cannulation in paediatrics are the radial, femoral, ulnar, posterior tibial, dorsalis pedis and axillary arteries.

All sites are at risk of complications, ischemia due to small calibre (radial, dorsalis pedis), ischemia due to lack of good collateral (brachial artery), air embolism (retrograde bubble may enter into cerebral circulation in axillary artery).

Infectious complications may be higher in femoral, but this may be the only palpable artery in hypotensive patients.

**Check Collateral Circulation**

Controversial issue: Some authorities recommend verifying and documenting collateral circulation through the palmar arch, if we are cannulating radial/ulnar artery via the use of the modified Allen's test. There is lack of evidence that this test can predict hand ischemia after radial artery cannulation.

Steps
- First, raise patients hand above heart level
- The patient clenches the fist to exsanguinate the hand.
- Firm digital pressure is then used to occlude both the radial and ulnar arteries at the pulse over the wrist.
- The hand is opened without hyperextending the fingers, and the occlusion of the ulnar artery is released.
- The open hand is observed for return of perfusion (rubor).
- The Allen test is normal if pallor resolves and rubor returns within 5 seconds, indicating adequate collateral flow.
- The Allen test is considered abnormal if pallor persists beyond 5 seconds, indicating inadequate palmar arch collateral circulation, and radial arterial puncture should not be performed at that site.
- Finally, the test should be repeated, this time with release of digital pressure over the radial artery to assess radial arterial flow and thereby ensure that the radial artery pulsation is not due solely to ulnar flow.

The operator should document the impression of test in procedure note.

**Keep equipment ready**

- Dressing tray
- Wrist board or roller pad under wrist
- Arterial catheter (pre-packed): 2 types commonly used
  - Catheter with needle
  - Catheter with needle and guidewire
- Size selection:
  - For <10 kg – 24G (Yellow cannula), French 3-4
  - 10 – 40 kg – 20 G (Blue cannula), French 4-5
  - > 40 kg – 18 G, French 5.
- Needle holder with suture
- Sterile dressing
Arterial connector
A pressure transducing system (fluid-filled noncompliant tubing with stopcocks; transducer; a constant flush device and electronic monitoring equipment)

Arterial Line Cannulation
- Establish a sterile field and maintain aseptic technique throughout the procedure.
- Use a finger of non-dominant hand to locate the most superficial (easily palpable) course of the artery or USG.
- Insert the needle at an angle 30 to 45 degrees from the horizontal; for catheterization.
- Once there is blood return, the needle is advanced slightly further to ensure catheter has entered vessel.
- For “over the needle” technique: advance the catheter over the needle; do not fully withdraw the needle until the hub of the catheter is at the skin.
- For the “catheter over a guidewire” technique:
  - Insert the needle until flashback is seen.
  - Insert the wire through the needle into the artery.
  - Remove the needle, controlling the wire at all times.
  - Thread the catheter over the wire into the artery.
  - Remove the wire.
- Secure the catheter to the skin with nylon or silk suture material (3.0 or 4.0) and a sterile transparent dressing.
- Connect the catheter to an appropriate extension or pressure tubing.
- Use plain or heparinized (2 units/mL) saline at 3ml/hour or use 500ml saline bottle encased in a bag pressurized to 300 mmHg; to prevent clotting of the catheter.
- Label the arterial line and document the procedure
- Check perfusion of extremity.

Zero and Level The Transducer
- To obtain accurate pressure readings, the air-fluid interface must be aligned with the reference point called phlebostatic axis (junction of 4th intercoatal space and midpoint between anterior and posterior chest wall).
- A spirit level system should be used for proper levelling
- Zeroing can be checked by opening the transducer stopcock to air and aligning with the phlebostatic axis, confirming that the monitor displays zero.
- Zeroing should be repeated with patient position changes, when significant changes in blood pressure occur, and routinely every 12 to 24 hours

Check if System is Optimally Damped
- Damping is the tendency of the oscillation to die down; anything that takes energy out of system dampens the system. (Figure 17, Table 67.7 and 67.8)
- With too much damping (an “overdamped” system), however, frictional forces impede the arterial waveform such that it loses energy. Note the widened
and slurred waveform characteristic of an overdamped pressure waveform (Figure 67.17). This waveform tends to underestimate SBP and overestimate DBP (Table 67.8).

- In both setting, mean arterial pressure (MAP) remains same, hence rely on MAP when system optimization is in doubt.
- Although damping seems to be a solely a theoretical issue, under damped and over damped waveforms are encountered on a daily basis during arterial pressure monitoring in the intensive care unit. The ability to recognize when these potential sources of error or “dynamic response artifacts” are present is essential to being able to effectively analyze and apply haemodynamic measurements in the care of the critically ill and avoid potentially detrimental therapy based upon erroneous data.
- Damping can be checked by square wave test
  - A “fast-flush” or “square wave test” is performed by opening the valve of the continuous flush device such that flow through the catheter-tubing system is acutely increased to 30 mL/hr from the usual 1-3 mL/hr. This generates an acute rise in pressure within the system such that a square wave is generated on the bedside monitor. With closure of the

<table>
<thead>
<tr>
<th>Nature of waveform</th>
<th>Effect on measurements</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Narrow, peaked tracing</td>
<td>Overestimates SBP, Underestimates DBP, MAP remains unchanged</td>
<td>Long tubing, Increased vascular resistance</td>
</tr>
</tbody>
</table>

### Table 67.8: Characteristic of Overdamped Waveforms

<table>
<thead>
<tr>
<th>Nature of waveform</th>
<th>Effect on measurements</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Widened and slurred pressure tracing</td>
<td>Underestimates SBP, Overestimates DBP, MAP remains unchanged</td>
<td>Air bubbles, Overly compliant tubing, Blood clots / Fibrin, Catheter kinks, Stopcocks / Injection ports, No fluid in flush bag / Low flush bag pressure</td>
</tr>
</tbody>
</table>
valve, a sinusoidal pressure wave of a given frequency and progressively decreasing amplitude is generated.
- Optimally damped - one to two oscillations before return to tracing
- Underdamped – more than 2 oscillations before returning to tracing
- Overdamped – less than 3 oscillations before returning to zero.

Check Arterial Waveform and Interpret (Figs 67.18 and 67.19)

- The farther into the periphery blood pressure is measured the waveform appears narrower, systolic and pulse pressure increase and the diastolic pressure decrease. But MAP always remains same.

![Arterial waveform](image)

**Fig. 67.18:** Arterial waveform

![Changes of the arterial pressure waveform configuration throughout the arterial tree. Note the increasing steepness and amplitude of the systolic upstroke and the changing location of the dicrotic notch. This phenomenon is also called Distal pulse wave amplification.](image)

**Fig. 67.19:** Changes of the arterial pressure waveform configuration throughout the arterial tree. Note the increasing steepness and amplitude of the systolic upstroke and the changing location of the dicrotic notch. This phenomenon is also called Distal pulse wave amplification.
Systolic blood pressure variations seen in hypovolemia
Steep slope of upstroke suggests good contractility
Position of dicrotic notch – low (low systemic vascular resistance) and high (high afterload)
Slope of decent – steep (low systemic vascular resistance)

Optimize Natural Frequency of System
- Use wide-bore, high-pressure tubing no longer than 122 cm (48 in)
- Avoid tubing extensions and minimize stopcocks
- Ensure that all connections are tightened
- Eliminate air from the flush fluid and air bubbles from the tubing system
- Keep continuous flush bag filled and keep external pressure cuff at 300 mm Hg pressure
- Keep cannulated extremity in a neutral or slightly extended position to prevent catheter kinking.

Arterial Catheter Maintenance and Documentation
- Label arterial line
- Check perfusion of extremities regularly
- Check dressing daily and change if dirty or collection underneath
- Check insertion site for infection/inflammation
- The tubing and the transducer should be changed at 96-hr intervals.

Watch for Complications
- The most serious complication of arterial puncture is permanent damage to the artery, interrupting the arterial supply to the distal extremity. With selection of an appropriate puncture site and proper technique, this is an exceedingly rare occurrence
- Injury to the developing femoral head can occur in children during cannulation or puncture of the femoral artery.
- Hematoma, bleeding
- Nerve damage
- Pseudoaneurysm
- Thrombosis, embolisation
- CRBSI (Catheter related blood stream infection)

Treat Ischemia Complications (if they occur)
- Remove catheter
- Consider arterial duplex sonography or angiography (if condition of patient permits)
- Operative interventions, use of thrombolytics and sympathetic block (involve vascular surgeons)

When to Remove Arterial Catheter
- Remove it as soon as it is no longer needed or earlier if there are any complications
Intra Cranial Pressure Monitoring

Introduction

Intracranial pressure monitoring (ICP) monitoring is a common practice when treating intracranial pathology with risk for elevated ICP. The main objective for monitoring ICP is to assess cerebral perfusion to avoid secondary injury. The only method to reliably measure cerebral perfusion pressure (CPP) and assess for cerebral hypoperfusion or intracranial hypertension is to continuously monitor ICP and blood pressure. ICP data are very useful to help predict outcomes and worsening intracranial pathology, such as cerebral edema or hemorrhage, and is useful for guiding therapy.

Step 1: Indication for monitoring

- Severe traumatic brain injury
- Intracranial hemorrhage
- Post-craniotomy
- Space-occupying lesions such as epidural and subdural hematomas, tumors, abscesses, or aneurysms
- Reye syndrome patients who develop coma, posturing, and abnormal responses to obnoxious stimuli
- Encephalopathy from lead ingestion, hypertensive crisis, or hepatic failure
- Meningitis/encephalitis

Step 2: Monitoring Techniques of ICP

There are multiple techniques: invasive as well as noninvasive. The noninvasive techniques (transcranial Doppler, tympanic membrane displacement, optic nerve sheath diameter, CT scan/MRI and fundoscopy) can be used as reliable alternatives to the invasive techniques (ventriculostomy and microtransducers). However, ventriculostomy is considered the gold standard in terms of accurate measurement of pressure, although microtransducers generally are just as accurate. Both invasive techniques are associated with a minor risk of complications such as hemorrhage and infection. Furthermore, zero drift is a problem with selected microtransducers. The non-invasive techniques are without the invasive methods' risk of complication, but fail to measure ICP accurately enough to be used as routine alternatives to invasive measurement.

Step 3: Preparation for Invasive Intracranial Pressure Monitoring

1. Device Accuracy and Stability
   a. External ventricular drain- Invasive monitoring using the EVD technique, where a catheter is placed into one of the ventricles through a burr hole, is considered the gold standard of ICP monitoring. In addition to measuring ICP, this technique can also be used for drainage of CSF and administering of medicine intrathecally, for example, antibiotic administration in cases...
of ventriculitis, possibly resulting from EVD placement itself. Depending on ventricular size, EVD placement may be difficult, especially in younger patients with a very slender ventricular system.

b. Microtransducer ICP monitoring devices- This group of invasive ICP monitoring devices can be divided into fiber optic devices, strain gauge devices, and pneumatic sensors.

Fiber optic devices, such as the Camino ICP Monitor, transmit light via a fiber optic cable towards a displaceable mirror. Changes in ICP will move the mirror, and the differences in intensity of the reflected light are translated to an ICP value.

The Codman MicroSensor, the Raumedic Neurovent-P ICP sensor, and the Pressio sensor belong to the group of piezoelectric strain gauge devices. When the transducer is bent because of the ICP, its resistance changes, and an ICP can be calculated.

Pneumatic sensors (Spiegelberg) use a small balloon in the distal end of the catheter to register changes in pressure, and additionally allows quantitative measurement of intracranial compliance.

It is worth mentioning, that the Neurovent-P sensor, the Spiegelberg sensor and the Codman MicroSensor are compatible with magnetic resonance imaging (MRI) without any danger to the patient. The Camino monitor and Pressio sensor contain ferromagnetic components, and therefore patients with these devices cannot undergo MRI.

2. Optimal Intracranial Location of Monitor - Depending on the technique, monitoring can be done in the intraventricular, intraparenchymal, epidural, subdural, or subarachnoidal compartment (figure 67.20).

![Diagram of probe locations](image-url)
The ICP microtransducers most widely used, are those measuring ICP intraparenchymally, usually placed in the right frontal region at a depth of approximately 2 cm. Epidurally placed ICP monitors overestimates ICP values, which is most likely due to physiologically different pressures in the two compartments and not due to technical aspects. The subdurally placed pressure sensors showed lower ICP values. So, intraparenchymal or intraventricular monitors should be considered the standard choice.

3. Complications-
- Recalibration: Generally, several of the above studies concluded that when it comes to measuring ICP, microtransducers are just as accurate as the EVDs. However, microtransducers share a common disadvantage, in that no recalibration is possible after placement; though the Spiegelberg catheter is an exception from this rule, as it recalibrates itself every hour. The EVDs, on the other hand, have the advantage of that they can be recalibrated at any time, simply by resetting the transducer to atmospheric pressure at the level of the so called zero reference point (Foramen of Monro/Tragus). The difference between the starting ICP value when the sensor is calibrated (0 mmHg), and the ICP value that is measured when the sensor is removed is termed “zero drift.” A large difference between these two ICP measurements indicates, that the ICP measured while the device was implanted in the patient, was not the “true” ICP at any given moment.
- Infection: Review articles on this subject have shown a frequency of catheter-related infections in the range of 0–27%; A better definition is bacterial colonization of the device rather than infection since there have been no reports in large prospective studies of clinically significant intracranial infections associated with ICP monitoring devices (There are no pediatric data on the use of prophylactic antibiotics to prevent infectious complications.
- Hemorrhage: There are no pediatric reports documenting the incidence of significant brain injury, hemorrhage, or seizures as a result of ICP monitoring.

Step 4: Preparation for ICP Catheter Insertion

**General care**

a. Obtain informed consent from the patient’s family to place a monitoring system.

b. Prior to placement of the ICP catheter, it is extremely important to have the following laboratory values: CBC with Platelet Count, PT / PTT, and Electrolytes. These values must be within normal limits, and all coagulopathies MUST be corrected prior to placement.
c. A copy of the patient’s CT Scan at the bedside to refer to, if needed, during insertion.

d. The patient’s dominant hemisphere should be determined. The dominant hemisphere controls the speech center and motor pathways. This is important because with the insertion of the ICP catheter brain tissue is destroyed. As a rule, the majority of the population is left hemisphere dominant, as demonstrated by being right-handed. Therefore, the ICP catheter should be placed on the right side of the brain.

**Equipment Necessary for ICP Catheter Insertion**

A sterile environment, sterile equipment – necessary for all types of catheters. The following equipment is needed for all types of catheter insertions. Additional equipment is listed under each of the catheter types.

- Gown, Mask, Sterile gloves, Towels 4 x 4, clippers,
- Betadine, Lidocaine with epinephrine, Benzoin, Betadine ointment
- Bath towels for head elevation
- Suture (3-0 nylon preferable)

**Patient Preparation for Procedure**

- Monitoring the ABC’s (airway, breathing, circulation).
- Positioning is also important. The patient must be in a supine position with the head elevated on a folded towel, keeping the head midline. The patient should be at the top edge of the bed with the headboard of the bed removed. The head of the bed should be elevated to 30 degrees.
- The patient must be prepped prior to insertion. This is done by shaving the patient’s hair. Then, using sterile gloves, scrub the area with a Betadine scrub brush or Betadine soaked 4 x 4’s for several minutes. Then allow to air dry.
- The patient cannot move his head during the insertion of the ICP catheter. Therefore, sedation and/or neuromuscular blockade agent may be required prior to and during the procedure.

**Step V: Procedure**

**ICP Catheter Insertion**

- The physician will apply a mask, hat, sterile gown, and new sterile gloves. Anyone in the room during the insertion procedure must also wear a hat, mask, and gloves to maintain the sterile environment.
- The physician prepares the ICP catheter and ICP kit. The physician then instills a local anesthetic in the area the procedure is going to be performed. Usually Lidocaine 1% with Epinephrine (1:100,000) is used to prevent bleeding of the scalp veins/arteries. It is important to avoiding bleeding since even a small amount of scalp blood can track down the ICP catheter and increase the pressure inside the cranial vault.
- The physician then makes a hole in the skull. During this, the nurse will need to hold the patient’s head still to avoid any movement.
The physician then makes an incision about 2-3 inches away from the hole. He then inserts the catheter and places the catheter and tracks the catheter under the skin to the hole. This helps in the prevention of infection.

The procedure is completed by suturing the burr hole site, applying Betadine ointment at the catheter exit, prepping the skin with tincture of Benzoin, covering the site with a sterile 2 X 2, and securing with foam tape.

**Monitoring Equipment Set Up (Figure 67.21)**

a. Each monitoring system has its own catheters, sensors, cables, and monitors

b. The appendix of this packet will provide a guide for how to set up several of the common systems. However, always follow the manufacturer’s guidelines and your institution’s policy and procedures when setting up monitoring equipment.

c. In general, components of ICP pressure monitoring systems are:
   - Non-compressible tubing
   - 3-way stopcock
   - Transducer
   - Preservative free normal saline fluid
   - Monitor
   - Connecting cable

d. The transducer converts pressure into a digital signal for display on the monitor.

e. Zeroing the monitor eliminates the influence of atmospheric pressure and increases accuracy. When zeroed, the transducer must be level to the foramen of Monro to eliminate the effects of gravity. These two actions will validate the measurement obtained.

![Fig. 67.21: Intraventricular ICP Monitoring System](image)
Step VI: Documentation of Insertion and Post-Insertion

- It is important for both the nurse and physician to document regarding the insertion procedure.
  - Pre-procedure neurological assessment
  - Medications given to patient prior to and during procedure
  - Conditions under which ICP catheter is inserted
  - Type of procedure/catheter placed (reference number, if applicable)
  - Catheter location (i.e. right or left, frontal, parietal, or occipital)
  - Post-procedure neurological assessment
  - Opening pressures
  - Calculation of cerebral perfusion pressure (CPP)
  - Characteristics of CSF (color, clarity, flow and quantity)
  - If ICP elevated, what treatment was ordered by MD
  - If CPP < 60, what treatment was ordered by MD
  - Waveform
    - Level of the transducer – foramen of Monro (outer canthus of eye, top of ear, or tragus of ear)
    - Level of the bag (place the “O”-Zero on the scale level with the foramen of Monro, slide the chamber on the drainage bag to the prescribed level – usually 20 mm Hg, make sure collection bag is clamped off).
    - Condition of the dressing
    - Any procedure performed on the drainage system during insertion, such as irrigation and/or sampling

Step VII: Interpretation of ICP monitoring

It is conventional to calibrate ICP monitoring apparatus in units of mmHg to permit a direct comparison of ICP with blood pressure and to enable the difference between the two pressures (CPP) to be calculated. ICP records offer two main kinds of information, the baseline level and variations of the pressure, i.e. waves. In other words, raised ICP may be steady or periodic.

Baseline Pressure

Normal ICP is pulsatile due to intracranial arterial pulsations reflecting the cardiac and respiratory cycles. Based on largely intuitive considerations, the normal level of mean ICP is 0–10 mmHg and it is abnormal over 15 mmHg. Lundberg suggested that mean levels above 20 mmHg are moderately elevated and that sustained levels above 40 mmHg are severely increased (Lundberg, 1960). In head injury, it is more common to observe a rise in baseline pressure, rather than waves of raised ICP. If the bone flap has been removed surgically, pressure readings can be unreliable.

Pressure waves

Lundberg identified three different types of ICP variations, ‘A’, ‘B’ and ‘C’ waves (Figure 67.22). Plateau waves (‘A’ waves) are clinically very important because
they indicate dangerously reduced intracranial compliance. They rise steeply from near normal or slightly raised ICP to 50 mmHg or more and persist for 5–20 minutes before falling precipitously, even to below the original level. Although named for their rather flat tops, there may be irregularities and peaks.

The most frequent type of pressure wave, although of less adverse clinical significance than the plateau wave, is the ‘B’ wave. These are rhythmic oscillations, sharply peaked and occurring once every 1–2 minutes, in which mean ICP rises in a crescendo manner from a variable baseline to a level 20–30 mmHg higher, and then falls abruptly with no intervening period of sustained intracranial hypertension. ‘C’ waves seem to be of little clinical significance.

**Intracranial Pressure Waveform**

The ICP wave has a pulsatile quality at two different frequencies – one synchronous with the arterial pulse while the other is slower, in time with breathing (Figure 67.23). The vascular waves are caused by arterial pulsations in the large vessels within the brain, producing an oscillation in the volume of the ventricular system. The shape of the CSF pressure wave is similar to that of systemic blood pressure and it has three fairly consistent components, the ‘percussion wave’ (P1), ‘tidal wave’ (P2) and ‘dicrotic wave’ (P3; Figure 67.24). The dicrotic notch between P2 and P3 corresponds to the dicrotic notch of the arterial pulsation. The respiratory wave is synchronous with alterations in central venous
pressure, reflecting intrathoracic pressure. They are seen prominently in patients on ventilators. Normally, the amplitude of the cardiac pulse is about 1.1 mmHg, and the combined cardiac and respiratory variation is approximately 3.3 mmHg.

**Step 8: EVD Removal**

An EVD is a temporary solution or treatment for patients with increased ICP. An EVD is usually in place for 5–10 days. This time period gives the time to assess the cause and apply a more long-lasting solution or treatment. An EVD may be removed for the following reasons:

- ICP monitoring is no longer necessary
- Infection risk is increased
- Ventriculoperitoneal shunt placement
- Hydrocephalus resolution.

**Chest Tube Placement**

**Introduction**

Chest tube insertion (tube thoracostomy) involves placement of a sterile tube into the pleural space to evacuate air or fluid into a closed collection system to restore negative intrathoracic pressure, promote lung expansion, and prevent potentially lethal levels of pressure from developing in the thorax. Insertion and
care of chest tubes are common issues not only in the intensive care unit but throughout the hospital and have become a required component of the training for Pediatric Advanced Life Support.

**Step 1: Understand some terms**

**Chylothorax:** Collection of lymph fluid in the pleural space

**Haemothorax:** Collection of blood in the pleural space

**Pneumothorax:** Collection of air in the pleural space

**Tension Pneumothorax:** One way valve effect allowing air to enter the pleural space, but not to leave. Air builds up forcing a mediastinal shift. This leads to decreased venous return to the heart and lung collapse/compression causing acute life-threatening respiratory and cardiovascular compromise. It is a clinical diagnosis with hypotension and respiratory impairment as essential component’s, in a high risk individual. Radiology will confirm pneumothorax and not tension.

**Pleural effusion:** Exudate or transudate in the pleural space

**Under Water Seal Drain (UWSD):** Drainage system of either 1 or 2 or 3 chambers consisting of water seal with optional suction control and drainage collection chamber. UWSD are designed to allow air or fluid to be removed from the pleural cavity, while also preventing backflow of air or fluid into the pleural space.

**Flutter valve (e.g. pneumostat, Heimlich valve):** One way valve system that is small and portable for transport or ambulant patients. Allows air or fluid to drain, but not to backflow into pleural cavity.

**Step 2: Indications for Insertion (Table 67.9)**

**Needle Thoracentesis**
- Diagnostic and therapeutic purposes (e.g., pleural effusions of unknown cause)
- To evacuate air or fluid from the pleural space when symptoms do not permit time for chest tube placement (e.g., tension pneumothorax)

**Table 67.9:** Indications for thoracentesis and site preference

<table>
<thead>
<tr>
<th>Indication for Thoracentesis</th>
<th>Insertion Site</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Emergent situation (e.g. tension pneumothorax)</td>
<td>2nd intercostal space, superior edge of rib, mid-clavicular line</td>
</tr>
<tr>
<td>B. Gas accumulation with x-ray confirmation. (gas collects in the uppermost areas of the chest)</td>
<td>3rd, 4th or 5th intercostal space, superior edge of rib, anterior or mid axillary line.</td>
</tr>
<tr>
<td>C. Fluid accumulation with x-ray confirmation. (fluid accumulates in the most dependent areas.)</td>
<td>5th, 6th or 7th intercostal space, superior edge of rib, posterior or mid axillary line.</td>
</tr>
</tbody>
</table>
Percutaneous Thoracostomy

- Moderate and large pleural effusion or air leak
- Repeated thoracocentesis
- Recurrent effusion
- Thoracic surgeries, e.g. esophageal repair
- Pneumothorax related to trauma for those who may be transported by air or may be mechanically ventilated
- Hemothorax
- Chylothorax
- Empyema
- Other considerations (e.g., preventative measure after surgery to drain blood and prevent cardiac tamponade)

**Step 3: Rule Out Contraindications**

There is no absolute contraindication in emergency situations.

Relative contraindications are:

- Bleeding diasthesis (PT or ApTT more than twice normal, platlets < 50,000) should be corrected in non-emergency settings.
- Lung densely adherent to chest wall throughout hemithorax.
- Caution is required when there is history of thoracic surgery or pleurodesis on side of proposed chest tube insertion.
- Local skin infection.

**Step 4: Pre-drainage Assessment**

- Correct bleeding diasthesis in non-emergency settings
- A careful radiological differentiation between pneumothorax and bullous disease or collapse or effusion, is required
- If doubt, ask experts.

**Step 5: Preparation**

**For procedure:**

- Pillow or bed table
- Sterile gloves, mask, head cap and gown
- Chlorhexidine in alcohol or povidone-iodine solution
- Sterile gauze sponges
- Sterile basin for skin preparation
- Sterile towels or drape
- 2,5,10-ml syringe
- Needle size (dependent upon patient size)
  - Infant - #23 - 25 g butterfly needle or #22 - 24 g IV catheter
  - Child - # 18 g needle or #18 g angiocath
  - Adult - # 14 g needle (2” catheter-over-needle)
- 1% lidocaine
- Hemostat or metal spring
Instrument for blunt dissection (e.g., curved clamp)
Scalpel or # 15 blade (pointed)
Guide wire with dilator

Chest tube (as per age – approximation, not fixed)

<table>
<thead>
<tr>
<th>Chest tube size</th>
<th>Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>8FR -10FR</td>
<td>New born, Infants</td>
</tr>
<tr>
<td>10FR-12FR</td>
<td>Infants and Young children</td>
</tr>
<tr>
<td>12FR-16FR</td>
<td>Children (&lt;20kg)</td>
</tr>
<tr>
<td>16FR-32FR</td>
<td>Children (&gt;20kg) and adult sizes</td>
</tr>
</tbody>
</table>

Three-way stopcock
Intravenous tubing
ICD or Vacuum bottle
Sterile dressing
Tape to secure bandage
Monitors
Suture (e.g., “2-3” silk)
Resuscitation cart

For specimen collection:
- Aerobic and anaerobic culture bottles
- Sterile tubes for mycobacterial or fungal cultures
- Sterile tube for cytology
- Blood gas syringe
- Specimen tube for hematology
- Specimen tube for chemistry
- Other specimen tubes as needed

Step 6: Consent and Premedication

- Written and informed consent should be taken before procedure in non-emergency settings. If time permits take verbal consent in emergency settings.
- The physician and nurse should explain the procedure to the child in an age-appropriate manner, with the family included in the discussion
- Peripheral IV access, analgesic or mild sedation as needed or decided by physician

Step 7: Prepare Drainage System and Drain Size

- The nurse should fill the drainage bottle with sterile water to the fill line using an aseptic non-touch technique (ANTT).
- Ensure the drainage tube is 2cm below the water level - this ensures minimum resistance to drainage of air and will maintain the underwater seal even following a large inspiratory effort.
- Drain size depends on age (as mentioned already in preparation) or it depends on underlying pathology (no evidence supports it, but traditional teaching advocates -)
Small bore drains are more comfortable and are preferred in pneumothorax or pleural effusion

Large bore drains are usually preferred for empyema and acute hemothorax.

**Step 8: Patient Position (Figure 67.25)**

- The most commonly used position in PICU settings is with the patient lying at 45° with their arm raised behind the head to expose the axillary area or in a forward lean position.
- Procedure can also be done while the patient is sitting upright leaning over on bed side table with a pillow or in lateral decubitus position.
- Assist operator with positioning and restraining. Patient should be positioned and restrained in a manner that enhances visualization of the chest field requiring chest tube insertion.

**Step 9: Site Selection**

- Correct side and site should be rechecked before chest tube insertion by reviewing clinical signs and chest radiograph.
- Ultrasonography can be used as adjunctive guides to site of tube placement.
- General rule is to insert chest tube in ‘safe triangle’ through mid axillar line in 4th to 6th intercostal space.
- The safe triangle is bordered by the anterior border of latissimus dorsi, the lateral border of the pectoralis major muscle, a line superior to the horizontal level of the nipple, and an apex below the axilla.
- This position minimizes risk to underlying structures and avoids damage to muscle and breast tissue resulting in unsightly scarring.
- A more posterior position may be chosen if suggested by the presence of a locule but, while this is safe, it is more uncomfortable for the patient to lie on after insertion and there is a greater risk of the drain kinking.

**Step 10: Procedure**

**General rules:**

- Explain procedure and take consent
- Maximum sterile barrier

*Fig. 67.25: Patient position for chest tube insertion A. lying with hands above head B. sitting and leaning C. lateral decubitus*
Provide supplemental oxygen if required, secure intravenous access and attach all monitors
Ensure adequate lighting
Arrange all equipments on a sterile workplace
Once the insertion site is identified, cleanse the area with Chlorhexidine 2% thoroughly; working from the identified site of insertion out to at least 3-5 cm. Allow Chlorhexidine to dry for at least 1 minute. (In neonates use povidine-iodine solution)
Drape the desired anatomical region with sterile towels to create a surgical field. Avoid covering the patient’s head. Be careful to leave head and neck visible.
If patient condition permits, anaesthetize the insertion site subcutaneously first, and then advance to muscle and pleura using 1% Lidocaine. This infiltrates the muscle, periosteum and parietal pleura in the area of the tube’s passage. Wait 2 minutes to allow anaesthetic to take effect.

Three methods are described
- Guidewire tube thoracostomy
- Trocar tube thoracostomy
- Operative tube thoracostomy

A. Guidewire tube thoracostomy / Seldinger (small bore) chest drains
- Insert the introducer needle, just superior to the appropriate rib. Stop just at point where air or fluid is aspirated
- Direct the bevel appropriately; the bevel allows the drain to be directed basally for effusions or apically for pneumothorax.
- Remove the syringe. Straighten the J tip of the guide wire and pass the wire through the needle; there should be no resistance and the wire should pass freely.
- Remove the needle over the wire.
- Make a small nick at entry site and pass the dilator over the wire until into the thorax. The dilator should not be inserted further than 1cm beyond the depth from the skin to the pleural space.
- Remove the dilator over the wire, preventing the wire from being pulled out of, or pushed into, the thorax.
- Insert the drain over the wire, to a length, where all the side holes in tube are comfortably in pleural space.
- Remove the wire and inner tube from the drain.
- Attach the 3 way tap, leaving it closed until the drain is secured and attached to the drainage system.
- Secure the drain to the skin with a suture. It is recommended that the knot around the drain is tightened so that the drain tubing is slightly indented.

B. Trocar tube thoracostomy
- An outdated method because of the risks involved
- Make an incision parallel to the rib where the drain is to be inserted, after local anesthesia. Ensure that this is big enough for the drain (approximately 2-3cm) and goes through all the layers of the skin only
This method uses a chest tube with a trocar positioned inside the tube. The chest tube with the trocar is inserted between the ribs into the pleural cavity, directed towards opposite shoulder with flat edge of the stylet tip cephalad to prevent damage to intercostals vessels. Because significant force is often required to insert the trocar, the hand not applying the force should be placed next to patient's chest wall to control depth of penetration. Once pleural cavity is entered, the inner trocar is gradually removed from the chest tube. When the proximal end of trocar clears the chest wall, a clamp is placed between the trocar and chest wall until the trocar can be completely withdrawn and the tube attached to water seal drainage.

C. Operative tube thoracostomy / Blunt dissection technique (Fig. 67.27)

- Using the scalpel, make a small incision (0.5 cm) through the skin at the anesthetic wheal.
- Using the curved mosquito clamp, bluntly dissect down to the underlying rib. Some people feel that carrying the dissection through the subcutaneous tissue provides a tunnel that assists in fixation of the tube and helps assure an adequate seal. This subcutaneous tunnel may be carried superiorly over the next rib, anteriorly (for pneumothorax) or posteriorly (for effusion) parallel to the ribs, or obliquely.
- Entering the pleural space directly under the skin incision is thus an alternative to the subcutaneous tunnel.
- With the clamp curved over the superior margin of the rib, pressure is applied until the clamp is forced through the intercostal muscle and parietal pleura.
This is signaled by a sudden loss of resistance, and is often accompanied by an audible surge of air.

- The intercostal (pleural) opening is enlarged slightly by spreading the clamp. The clamp is then removed.
- Grasp the curved haemostat which is holding the catheter and direct the catheter through the incision into the pleural cavity. Once this is achieved, stabilize the catheter with the opposite hand and release it from the haemostat. Slowly remove the haemostat from the chest and advance the catheter to the desired position.
- The tube should be directed parallel to the lung surface and chest wall to help avoid inadvertent insertion of the tube into the lung parenchyma. The tube should also be directed either anteriorly (for pneumothorax) or posteriorly (for effusion), as indicated, by turning the clamp.
- Its entry site should be palpated to ensure that it is not in the subcutaneous tissue.

**Step 11: Fixing the Tube (Fig. 67.28)**

- The chest drain incision should be closed by a non-absorbable suture to narrow the linear incision around the edge of the chest drain.

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**Fig. 67.27:** Operative tube thoracostomy; (A) Use blunt tipped straight or curved hemostat to make a tunnel, (B) the end of the chest tube is grasped with a Kelly clamp and guided with a finger through the chest incision, the clamp is rotated 180 degrees to direct the tube toward the apex.

**Fig. 67.28:** Fixation of chest tube; (A) simple stay suture to secure the drain, (B) horizontal mattress suture for later wound closure.
The use of a "purse string" suture is controversial. Some believe they should not be used as they convert a linear wound into a circular one which is painful and may leave an unsightly scar. The alternative view is that a "purse string" suture is the best way of securing a drain.

The drain must be well secured after insertion to prevent it falling out. A stay suture can be placed through the skin and then criss-crossed up the drain ensuring it is not too tight or it can occlude a soft drain.

An omental tag of tape has also been described which allows the tube to lie a little away from the chest wall to prevent tube kinking and tension at the insertion site.

**Step 12: Verification of Chest Tube Placement and Documentation**
- The location of the tube should be confirmed by observing flow of air (seen as condensation within the tube) or fluid from the tube.
- Attach the end of the drain onto the underwater drainage system (making sure that the drain is 2-3 cm below the water level) and place the chest drain bottle below the patient.
- Ideally both PA and lateral view to be taken as ectopic locations may be missed in PA view.
- Documentation

**Step 13: Drainage Systems**

**One bottle drainage system (Fig. 67.29)**
- In this system, one bottle serves as both collection container and a water seal.
- In the 1-bottle system the chest drain is connected by collecting tubing to a tube approximately 2-3 cm under water (the seal) in the underwater-seal bottle while another vent tube is always open to atmosphere.
- In this system pleural pressure greater than 2-3 cm water will force air or fluid from the pleural space into the bottle while negative pressure in the pleural space will suck fluid up the tube.
- As long as the underwater-seal bottle is well below the patient (e.g., on the floor beside the patient), the hydrostatic pressure of the fluid column in the tube will counterbalance the negative pleural pressure and prevent water from being sucked into the pleural space.
- This is best for uncomplicated effusion or moderate pleural effusion.

![Fig. 67.29: One-bottle drainage system.](image)
If substantial amount of fluid is draining from patient’s pleural space, the level of fluid will rise in one bottle system, and therefore, patient will have to generate more pressure to allow additional fluid or air to exit the pleural system. So, in such a case 2 bottle drainage system is advocated.

**Two bottle drainage system (Fig. 67.30)**
The working principle of the 2-bottle system is the same as the 1-bottle system except a trap bottle is interposed between the drain tube and the underwater-seal bottle. So any amount of fluid can be collected in trap bottle, but water seal is unaffected.

**Three bottle drainage system (Fig 67.31)**
The system has a trap bottle (attached from patient’s side) and a water-sealed bottle, along with a manometer (pressure-regulating) bottle. This
bottle helps the system maintain a measured, constant negative pressure and negative flow.

- To obtain a suction of -20 cm of water, set the tip of the tube in manometer bottle to 20 cm below the surface of the fluid. Now, increase the vacuum gradually until air bubbles gently and constantly through the atmospheric vent in the water during both phases of respiration. A constant pressure of -20 cm of water is now transmitted to the underwater seal and on to the chest drain.

**Step 14: Care of Chest Tube**

- Bubbling chest drains are **NEVER** clamped – a tension pneumothorax will quickly develop
- 2 clamps must be kept at the bedside in case of accidental disconnection. It is not necessary to clamp the drain for moving or transferring the child
- ‘Milking’ the tube is no longer recommended as it creates exceedingly high negative pressures that can cause trauma to the lung
- Check Patient
  - Temperature
  - Pulse
  - Blood Pressure
  - Respiratory rate
  - Oxygen Satuations

- Chest drain observations
  - **Swinging** (tidaling) on inspiration indicates tube patent.
  - **Bubbling** only occurs if pneumothorax present – will stop when lung fully inflated. Excessive bubbling may indicate an air leak.
  - **Static** – examine the tubing for kinks/blockage. Ask patient to cough (increased pressure should cause fluid to move).
  - Drainage – record type and volume Ensure tubing remains below water level
  - Keep drainage bottle approx 30cm below child’s chest
  - Keep bottle upright
  - Do not allow dependent loops of tubing to form
  - Ensure connections are secure
  - The drain should be clamped for 1 hour once 10ml/kg is drained to prevent re-expansion pulmonary oedema.
  - If there is sudden cessation of fluid draining, a blockage must be suspected in empyma. Check for kinks: if none, obstruction with thick pus must be suspected and medical advice sought (the drain may need flushed with 10ml sodium chloride 0.9%).
  - Intra-pleural Fibrinolytics: Are recommended for any complicated parapneumonic effusion (thick fluid with loculations) or empyma (overt pus)
- Urokinase should be given twice daily for 3 days (6 doses in total) using 40,000 units in 40 ml 0.9% saline for children aged 1 year or above, and 10,000 units in 10 ml 0.9% saline for children aged under 1 year.
- The drain is usually clamped for 4 hours post-instillation and the patient turned to aid dispersion. Physiotherapy is often timed to coincide with this.

**Step 15: Guidelines for Removal**

- Fully expanded lung
- Resolution of air leak for 24 hours For empyma or effusion – clinical (fall in fever, better appetite) and biochemical resolution (fall in acute phase reactants) with a fall in pleural drainage (controversial issue: amount less than 2ml/kg/day for 2-3 days, don't wait for complete cessation, check USG chest can be done to rule out significant or loculated collection)
- Clamping of the drain before removal is generally unnecessary.
- Tube removal is often preceded by oral or parenteral analgesia at an appropriate time interval.
- The suture holding the tube to the skin is cut.
- As the patient takes deep breaths, the tube is removed during inspiration or valsava maneuver (traditional teaching) and the hole simultaneously covered with an occlusive gauze dressing at peak inspiration (at which point only positive pressure can be generated in the pleural space, minimizing the possibility of drawing air in).
- Removal is with a brisk movement while assistant pulls mattress suture
- A chest radiograph is performed immediately to check for a pneumothorax and is repeated 24 hours later to rule out reaccumulation of air or fluid

**Step 16: Complications**

- Local site bleeding
- Hematoma
- Hemothorax from intercostals vessel injury
- Misplaced tube
- Non functional tube
- Subcutaneous emphysema
- Laceration of lung, liver, heart

**Intra Abdominal Pressure Monitoring**

**Introduction**

The concept of IAH was proposed in the late 1800s, forgotten after World War I, and rediscovered near the end of the 20th century. In 2004, a group of international physicians and surgeons formed the World Society of the Abdominal Compartment Syndrome (WSACS). The goal of this new organization was to develop a cohesive approach to the management of IAH and ACS, foster education and research, and develop consensus statements and definitions. WSACS has developed evidence-based definitions, guidelines, and treatment
algorithms and has identified evidence-based devices and methods to measure intra-abdominal pressure (IAP).

**Step 1: Understand the Definition, Cause, Classification of Intra-abdominal Pressure**

**Intra-abdominal Pressure (IAP):** is the steady-state pressure within the abdominal cavity. In healthy persons, IAP is 0 to 5 mm Hg and varies inversely with intrathoracic pressure during normal breathing. Various factors, such as coughing, sneezing, and loud singing, can cause IAP to increase drastically for short periods and then return easily to baseline.

**Intra-abdominal Hypertension (IAH):** is a sustained or repeated pathological elevation of IAP of 12 mm Hg or greater. WSACS has developed grades of IAH (Table 67.1).

**Abdominal Perfusion Pressure:** Abdominal perfusion pressure (APP) is a measure of the relative adequacy of abdominal blood flow. APP is calculated by subtracting the IAP from the mean arterial pressure (MAP): MAP-IAP=APP. The APP in patients with IAH or ACS should be maintained at 60 mm Hg or higher.

**Abdominal Compartment Syndrome (ACS):** is a sustained IAP greater than 20 mm Hg (with or without an APP <60 mm Hg) associated with new organ dysfunction or failure.

The World Society of the Abdominal Compartment Syndrome (WSACS) grading system

**Causes of ACS**

WSACS categorizes conditions that cause ACS as primary (surgical), secondary (medical), and recurrent (Table 67.11). Primary conditions are ones that need surgical or interventional radiological treatment. Secondary conditions are due to medical causes that do not require surgery or radiological intervention as an initial therapy. Recurrent conditions are ones in which ACS redevelops after surgical or medical treatment of primary or secondary causes of ACS.

**Step 2: Measurement of Intra Abdominal Pressure**

Research results have shown no correlation between abdominal girth and IAP measurements. Serial measurements of abdominal girth are not sensitive

**Table 67.10: Grades of IAH as defined by WSACS (World Society of the Abdominal Compartment Syndrome)**

<table>
<thead>
<tr>
<th>Grade</th>
<th>WSACS Classification</th>
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<tbody>
<tr>
<td>I</td>
<td>12–15</td>
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<tr>
<td>II</td>
<td>16–20</td>
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<tr>
<td>III</td>
<td>21–25</td>
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<tr>
<td>IV</td>
<td>&gt; 25</td>
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</table>
or specific enough to detect IAH. A distended abdomen that has increased slowly over time, as in chronic ascites or pregnancy, will not necessarily have an elevated IAP. Conversely, clinically important IAH can occur in the absence of a distended abdomen with the onset of an acute condition.

Various methods are used for direct and indirect measurements of IAP. While direct intraperitoneal catheter determinations are ideal, a variety of less invasive techniques for determining IAP have been devised including measurement of intravesicular (bladder), intragastric, intracolonic, and intrauterine pressure. Measuring IAP indirectly via the urinary bladder is currently considered the gold standard. Either a transducer technique or a manometer technique can be used.

Bladder pressure may be measured by a self-constructed kit using a conventional pressure transducer system connected to the patient’s urinary drainage system or through a commercially manufactured kit designed for the purpose of measuring IAP. Two commercial kits are available for the transducer method. The AbViser AutoValve (Wolfe Tory Medical, Inc, Salt Lake, Utah; Figure 67.32) has a valve that automatically opens 1 to 3 minutes after the saline has been instilled, adding a measure of safety to this device. Another IAP transducer kit is the Bard intra-abdominal pressure monitoring device (Bard Medical

<table>
<thead>
<tr>
<th>Table 67.11: Causes of increased intrabdominal hypertension</th>
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<tbody>
<tr>
<td>Primary</td>
</tr>
<tr>
<td>Blunt/penetrating trauma</td>
</tr>
<tr>
<td>Liver transplantation</td>
</tr>
<tr>
<td>Ruptured abdominal aortic aneurysm</td>
</tr>
<tr>
<td>Postoperative bleeding</td>
</tr>
<tr>
<td>Retropertitoneal hemorrhage</td>
</tr>
<tr>
<td>Mechanical intestinal obstruction</td>
</tr>
<tr>
<td>Postoperative closure of the abdomen under tension</td>
</tr>
<tr>
<td>Bleeding pelvic fractures</td>
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</table>

Fig. 67.32: The Ab Viser Auto valve - commercially manufactured kit for measuring IAP
Division, Covington, Georgia). Regardless of the transducer setup used, the way in which an IAP measurement is obtained should remain the same.

**Step 3: Preparation**

Regardless of the technique utilized, several key principles must be followed to ensure accurate and reproducible measurements from patient to patient.

- It is measured at end-expiration after ensuring that abdominal muscle contractions are absent.
- As head of bed elevation appears to significantly increase IAP measurements, the patient should be in the complete supine position.
- The transducer zeroed in the mid-axillary line at the level of the iliak crest.
- A maximal instillation volume of 25 mL of sterile saline (3 mL/kg for children) should be used for the intravesical technique as recent studies have demonstrated that larger volumes of fluid can lead to falsely elevated IAP measurements.
- Room temperature saline significantly increases IAP, presumably due to bladder detrusor contraction. As a result IAP determination should be performed 30-60 seconds after instillation of the priming fluid to allow bladder detrusor muscle relaxation.

**Step 4: Different Methods**

Modified method for intrabladder pressure monitoring by transducer (figure 67.33)

**Procedure**

- Patient in supine position.
- Zero pressure module at the midaxillary line at the level of the iliak crest (mark for future reference) by turning the proximal stopcock onto the air and the transducer.
- At rest, the three stopcocks are turned ‘off’ to the IV bag, the syringe, and transducer giving an open way for urine to flow into the urometer.
- To measure IBP, the urinary drainage tubing is clamped distal to the ramp, and the third stopcock is turned ‘on’ to the transducer and the patient.
- The first stopcock is turned ‘off’ to the patient and ‘on’ to the IV infusion bag.
- The second stopcock is turned ‘on’ to the IV bag and the 60-ml syringe.
- Aspirate 20-25 ml of saline from the IV bag into the syringe.
- The first stopcock is turned ‘on’ to the patient, and the 20-25 ml of normal saline is instilled in the bladder.
- The first and second stopcocks are then turned ‘on’ to the patient and thus turned ‘off’ to IV tubing and the syringe.
- The third stopcock already being turned ‘on’ to the transducer and patient allows then immediate IBP reading on the monitor.
- After IAP determination, the clamp is removed, the bladder allowed to drain, and the volume of saline utilized subtracted from the patient’s urinary output for that hour.
Set up

- Using sterile scissors, the drainage tubing is cut 40 cm after the culture aspiration port after desinfection.
- A ramp with three stopcocks is connected to a conical connection piece at each side with a male/male adaptor and inserted.
- A standard intravenous (IV) infusion set is connected to a bag of 500 ml of saline and attached to the first stopcock
- A 60-ml syringe is connected to the second stopcock
- The third stopcock is connected to a pressure transducer via rigid pressure tubing
- The system is flushed with normal saline.

Intrabladder pressure monitoring with the FoleyManometerLV. (figure 67.34)
This technique that uses the patient’s own urine as pressure transmitting medium is a simple, reliable, and cost-effective clinical tool.

The disposable FoleyManometer provides a closed sterile circuit which connects between the patient’s Foley catheter and the urine collection device. Each IAP determination takes about 10 s, and no subsequent correction of urine output is required.

The technique uses a low bladder infusion volume, has a needle-free sampling port, and can measure IAP in a range from 0 to 40 mmHg.

Procedure (Figure 67.33 a,b,c)

Initial setup (Figure 67.33 a): -
- Open the FoleyManometer LV pouch and close the tube clamp
- Place the urine collection device under the patient’s bladder and tape the drainage tube to the bed sheet
- Insert the FoleyManometer between catheter and drainage device
- Prime the FoleyManometer with 20 ml of sterile saline through its needle-free injection/sampling port.
- Prime only once at initial setup

Urine drainage (Figure 67.33 b)
- Let the urine drain in between IBP measurements
- Urine sampling from the needle-free port is facilitated by temporarily opening the red clamp. Remember to close clamp afterward
- Avoid a U-bend of the large urimeter drainage tube (which will impede urine drainage)
- Replace the FoleyManometer whenever the Foley catheter or the urine collection device is replaced, or at least every 7 days.
Intravesical pressure monitoring (Figure 67.33 c):
- Place the '0-mmHg' mark of the manometer tube at the midaxillary line at the level of the iliac crest (mark for future reference) and elevate the filter vertically above the patient.
- Open the bio-filter clamp and read IBP (end-expiration value) when the meniscus has stabilized after about 10 s
- Close clamp after IBP measurement and place the FoleyManometer in its drainage position.

Complications
- Urinary tract infection if asepsis is not maintained.
- Respiratory or haemodynamic compromise secondary to supine positioning.

Step 5: Interpreting the Result
- Bladder pressure should be measured in mm Hg.
- Sustained IAP greater than 12 mm Hg is considered IAH, whereas IAP greater than 20 mm Hg with organ dysfunction is considered ACS.
- ACS is no longer graded as mild, moderate, or severe on the basis of IAP measurements, instead it is present or it is not.
- Serial measurements of bladder pressure are recommended at least every 2 to 4 hours. Sustained elevations of IAP greater than 12 mm Hg are cause for concern. Ongoing assessment for rising IAP and associated organ system effects are essential to reduce organ system dysfunction and mortality.
- Conditions that may make measurements of bladder pressures inaccurate include a ruptured bladder, abdominal packing, pelvic hematomas in the lower abdomen.
- Early nonsurgical techniques to reduce IAP have been proposed to address the 4 causes of IAH and prevent progression to ACS. These include the following:
  - Improve abdominal wall compliance—supine body positioning, sedation, pharmacological paralysis
  - Evacuate bowel intraluminal contents—nasogastric decompression, rectal decompression, gastro-and colo-prokinetic agents
  - Evacuate intra-abdominal fluid collections—paracentesis, percutaneous abscess and hematoma drainage
  - Correct positive fluid balance—diuretics, colloids, and hemodialysis and ultrafiltration

Further Reading
Useful Information and Equations in the PICU

Praveen Khilnani, Partha Bhattacharya

Alveolar Air Equation
\[ \text{PaO}_2 = \frac{\text{FiO}_2 (\text{Pb} - 47) - \text{PaCO}_2}{R} \]

Alveolar Arterial Oxygen Gradient
\[ \text{A–a (O}_2) = (\text{FiO}_2 \% 100) \times (\text{Pb} - 47 \text{ mm Hg}) - (\text{PaCO}_2/0.8) - \text{PaO}_2 \]

Airway Resistance Equation
Pressure = Flow \times \text{Resistance}
\[ \text{Resistance} = \frac{8 \text{ nl}}{\pi r^4} \]
Resistance is inversely proportional to 4th power of radius

Oxygen Content (\text{CaO}_2)
\[ \text{CaO}_2 = (\text{PaO}_2 \times 0.003) + [\text{Hb (gm)} \times \% \text{ Sat} \times 1.36] \]

Oxygen Delivery (\text{DO}_2)
\[ \text{DO}_2 = \text{CI} \times \text{CaO}_2 \times 10 \]
Normal: 620 ml/min/m²

Oxygen Consumption (\text{VO}_2)
\[ \text{VO}_2 = \text{CI} \times (\text{CaO}_2 - \text{CvO}_2) \times 10 \]
Normal: 120–200 ml/min/m²

Oxygen Extraction
\[ \frac{\text{CaO}_2 - \text{CvO}_2 \times 10}{\text{CaO}_2} \]
Shunt Equation (Qs/Qt)

$$\frac{Qs}{Qt} = \frac{C_{cO_2} - C_{aO_2}}{C_{cO_2} - C_{vO_2}}$$

Systemic to Pulmonary Shunt (Qp/Qs)

$$\frac{Qp}{Qs} = \frac{S_{aO_2} - S_{mV_{O_2}}}{S_{pV_{O_2}} - S_{pA_{O_2}}}$$

Systemic Vascular Resistance Index (SVRI)

$$SVRI = \frac{MAP - RAP \times 80}{CI}$$
Normal: 800–1200 dynes/sec/cm$^5$

Pulmonary Vascular Resistance Index (PVRI)

$$PVRI = \frac{MPAP - PCWP \times 80}{CI}$$
Normal: 20–120 dynes/sec/cm$^5$

- $P_{aO_2}$: Partial pressure of alveolar oxygen
- $A-a (O_2)$: Alveolar arterial oxygen gradient
- $F_{iO_2}$: Fraction of inspired oxygen
- $P_b$: Atmospheric barometric pressure at sea level
- $47$: Water vapor pressure
- $P_{aCO_2}$: Partial pressure of arterial carbon dioxide
- $R$: Respiratory quotient = 0.8
- $\pi$: (π) = 22/7
- $r$: Radius
- $l$: Length
- $n$: Viscosity
- $MPAP$: Mean pulmonary artery pressure: 15 mm Hg (systolic 25/diastolic 10)
- $PCWP$: Pulmonary capillary wedge pressure: 9 mm Hg (8–12 mm Hg)
- $MAP$: Mean arterial pressure: (systolic + (2 × diastolic))/3
- $RAP$: Right arterial pressure
- $S_{aO_2}$: Arterial oxygen saturation
- $S_{mV_{O_2}}$: Mixed venous oxygen saturation
- $S_{pV_{O_2}}$: Pulmonary venous oxygen saturation
- $S_{pA_{O_2}}$: Pulmonary arterial oxygen saturation
- $CI$: Cardiac index (cardiac output/surface area: 3.2–5.2 L/msq)
- $C_{cO_2}$: Pulmonary capillary oxygen content
- $C_{aO_2}$: Arterial oxygen content
- $C_{vO_2}$: Venous oxygen content
**Pharmacokinetics**

1. Concentration = \( \frac{\text{Dose} \times \text{Half life}}{\text{Vol D} \times 0.69} \)

2. Loading dose = Concentration desired \( \times \) Vol D

3. Clearance = \( \frac{\text{Rate of administration}}{\text{Steady state concentration}} \)

Vol D: Volume of distribution

Peak level (drug concentration) (e.g. usually 1 hour after gentamicin administration); if high, reduce the dose. If low, increase the dose.

Trough level (drug concentration) (e.g. before the next dose usually after three doses of gentamicin given to achieve steady state); if high, increase the dose interval. If low, decrease the dose interval.

4. Drip rate concentration = \( \frac{\text{Drip rate (New)}}{\text{Concentration (New)}} \)

**Drip Calculation**

Drug concentration (mg/100 ml) = \( \frac{6 \times \text{Desired dose (mcg/kg/min)} \times \text{kg}}{\text{Desired fluid rate (ml/hr)}} \)

\( 6 \times \text{Body weight in kg} \times \frac{\text{mg of drug in 100 ml}}{1 \text{ ml/hr}} = \frac{\text{1 mcg/kg/min}}{1} \)

**Acid-Base/Renal/Fluids**

- 10 mm Hg PaO\(_2\) = 0.08 unit pH
- 0.15 unit pH = 10 mEq/L HCO\(_3\)

Increase in pCO\(_2\) by 10 mm Hg + increase HCO\(_3\) by 1 mEq/L

Decrease in pCO\(_2\) = (acute) decrease HCO\(_3\) by 1.5 mEq/L

(chronic) decrease HCO\(_3\) by 3 mEq/L

**Serum Osmolality**

\[ \text{Serum osmolality} = 2 \times (\text{Na}) + \frac{\text{Blood glucose}}{18} + \frac{\text{BUN}}{2.8} \]

**Anion Gap**

Anion gap = Na – (Cl + HCO\(_3\))

\[ 139 – (105 + 24) = 11 (> 15 \text{ abnormal}) \]
Fractional Excretion of Na (FENa)

\[
FENa = \frac{\text{Urine} / \text{Plasma Na}}{\text{Urine} / \text{Plasma Cr}}
\]

FENa < 1: Prerenal
FENa > 1: Renal

Renal Failure Index (RFI)

\[
RFI = \frac{\text{Urine Na}}{\text{U/P Cr}}
\]

RFI < 1: Prerenal
RFI > 1: Renal
U/P Cr: Urine to plasma creatinine

Maintenance Fluids

- 10 kg 4 ml/kg/hr
- Next 10 kg 2 ml/kg/hr
- More than 20 kg 1 ml/kg/hr
  - (For 20 kg child 60 ml/hr)
- Fluid bolus 20 ml/kg up to three to four times

Temperature Conversion

Degree F (Fahrenheit) = 32 + Degree centigrade (Celsius) × 9/5 (or 1.8)
Therefore,

\[
38°C = 38 \times 9/5 + 32 = 68.5 + 32 = 100.5°F
\]

Body Surface Area (BSA)

<table>
<thead>
<tr>
<th>Wt. (kg)</th>
<th>BSA (m²)</th>
<th>Wt. (kg)</th>
<th>BSA (m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>0.12</td>
<td>25</td>
<td>0.93</td>
</tr>
<tr>
<td>3</td>
<td>0.20</td>
<td>30</td>
<td>1.07</td>
</tr>
<tr>
<td>4</td>
<td>0.23</td>
<td>35</td>
<td>1.20</td>
</tr>
<tr>
<td>5</td>
<td>0.25</td>
<td>40</td>
<td>1.32</td>
</tr>
<tr>
<td>6</td>
<td>0.29</td>
<td>45</td>
<td>1.43</td>
</tr>
<tr>
<td>7</td>
<td>0.33</td>
<td>50</td>
<td>1.53</td>
</tr>
<tr>
<td>8</td>
<td>0.36</td>
<td>55</td>
<td>1.62</td>
</tr>
<tr>
<td>9</td>
<td>0.40</td>
<td>60</td>
<td>1.70</td>
</tr>
<tr>
<td>10</td>
<td>0.44</td>
<td>65</td>
<td>1.78</td>
</tr>
<tr>
<td>15</td>
<td>0.62</td>
<td>70</td>
<td>1.84</td>
</tr>
<tr>
<td>20</td>
<td>0.79</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Biostatistics

Sensitivity = \frac{\text{True positive}}{\text{True positive} + \text{False negative}}

Specificity = \frac{\text{True negative}}{\text{True negative} + \text{False positive}}

Positive predictive value = \frac{\text{True positive}}{\text{True positive} + \text{False positive}}

Negative predictive value = \frac{\text{True negative}}{\text{True negative} + \text{False negative}}

False negative rate = \frac{\text{False negative}}{\text{True positive} + \text{False negative}}

False positive rate = \frac{\text{False positive}}{\text{True negative} + \text{False positive}}
## PICU Drug List

*Suchita Khadse, Sagar Lad, Vinayak Patki*

### Anticonvulsant Therapy

<table>
<thead>
<tr>
<th>Drug</th>
<th>Doses</th>
<th>Continuous infusion</th>
<th>Maintenance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Midazolam</td>
<td>0.05–0.2 mg/kg</td>
<td>1–18 µg/kg/min</td>
<td></td>
</tr>
<tr>
<td>Lorazepam</td>
<td>0.1 mg/kg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phenytoin</td>
<td>15–18 mg/kg</td>
<td></td>
<td>5–8 mg/kg/day</td>
</tr>
<tr>
<td>Phenobarbston</td>
<td>15–20 mg/kg</td>
<td></td>
<td>5–6 mg/kg/day</td>
</tr>
<tr>
<td>Sodium valproate</td>
<td>20 mg/kg</td>
<td></td>
<td>20 mg/kg/day (max. 60–100 mg/kg/day)</td>
</tr>
<tr>
<td>Levetiracetam</td>
<td></td>
<td></td>
<td>20 mg/kg/day (max. 60 mg/kg/day)</td>
</tr>
<tr>
<td>Thiopentone</td>
<td>1.5–5 mg/kg/dose</td>
<td>0.5–3 mg/kg/hr</td>
<td></td>
</tr>
<tr>
<td>Propofol</td>
<td>1–2 mg/kg/dose</td>
<td>2–0 mg/kg/hr</td>
<td></td>
</tr>
</tbody>
</table>

### Asthma

<table>
<thead>
<tr>
<th>Drug</th>
<th>Doses</th>
<th>Continuous infusion</th>
<th>Maintenance dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epinephrine</td>
<td>SC: 0.01 mg/kg/dose of 1:1000 solution</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Terbutaline</td>
<td>SC: 0.005–0.01 mg/kg/dose IV: 2–10 µg/kg</td>
<td>0.08–0.4 µg/kg/min (max. up to 10 µg used)</td>
<td></td>
</tr>
<tr>
<td>Aminophylline</td>
<td>5 mg/kg/dose</td>
<td>1 mg/kg/hr</td>
<td></td>
</tr>
<tr>
<td>Salbutamol</td>
<td>Resp. solution 0.5%, 0.01–0.03 mL/kg</td>
<td>Oral 0.05–0.1 mg/kg/dose q 8 hr</td>
<td></td>
</tr>
<tr>
<td>Hydrocortisone</td>
<td>10 mg/kg/dose</td>
<td>10 mg/kg/day q 6 hr</td>
<td></td>
</tr>
<tr>
<td>Methylprednisone</td>
<td>1–2 mg/kg/24 hr</td>
<td>1–2 mg/kg/24 hr q 6–12 hr</td>
<td></td>
</tr>
<tr>
<td>Dexamethasone</td>
<td>0.3 mg/kg/dose</td>
<td>0.3 mg/kg/day q 6 hr</td>
<td></td>
</tr>
</tbody>
</table>
### Inotropes

<table>
<thead>
<tr>
<th>Drug</th>
<th>Doses</th>
<th>Continuous infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dopamine</td>
<td>5–20 µg/kg/min</td>
<td></td>
</tr>
<tr>
<td>Dobutamine</td>
<td>2.5–20 µg/kg/min</td>
<td></td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>0.05–0.1 µg/kg/min, max 1–2 µg/kg/min</td>
<td></td>
</tr>
<tr>
<td>Epinephrine</td>
<td>0.1–1 µg/kg/min</td>
<td></td>
</tr>
<tr>
<td>Vasopressin</td>
<td>0.0005–0.02 µg/kg/min titrate to effect</td>
<td></td>
</tr>
<tr>
<td>Milrinone (vasodilators)</td>
<td>50–75 µg/kg</td>
<td>0.25–0.75 µg/kg/min</td>
</tr>
</tbody>
</table>

### Paralysants

<table>
<thead>
<tr>
<th>Drug</th>
<th>Doses</th>
<th>Continuous infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vecuronium</td>
<td>0.1 mg/kg/dose 0.09–0.15 mg/kg/hr</td>
<td></td>
</tr>
<tr>
<td>Pancuronium</td>
<td>0.15 mg/kg 0.03–0.1 mg/kg/hr</td>
<td></td>
</tr>
<tr>
<td>Atracurium</td>
<td>0.4–0.5 mg/kg 0.4–0.8 mg/kg/hr</td>
<td></td>
</tr>
</tbody>
</table>

### Anesthetics

<table>
<thead>
<tr>
<th>Drug</th>
<th>Doses</th>
<th>Continuous infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ketamine</td>
<td>0.5–2 mg/kg/dose</td>
<td></td>
</tr>
<tr>
<td>Fentanyl</td>
<td>1–4 µg/kg/dose 0.5–5 µg/kg/hr</td>
<td></td>
</tr>
</tbody>
</table>

### Muscle Relaxant

<table>
<thead>
<tr>
<th>Drug</th>
<th>Doses</th>
<th>Continuous infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Succinyl choline</td>
<td>1–2 mg/kg/dose</td>
<td></td>
</tr>
</tbody>
</table>

### Antiarrhythmics

<table>
<thead>
<tr>
<th>Drug</th>
<th>Doses</th>
<th>Continuous infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenosine</td>
<td>Supraventricular tachycardia 0.05 mg/kg and ↑ by 0.05 mg/kg q 2 min until clinical response or max. dose of 0.25 mg/kg or 12 mg</td>
<td>10–15 µg/kg/24 hr maintenance dose 5–10 µg/kg/24 hr maintenance dose 2–5 µg/kg/24 hr maintenance dose</td>
</tr>
<tr>
<td>Digoxin</td>
<td>Congestive cardiac failure, supraventricular tachycardia 1 month–2 year: Loading dose of 30 µg/kg 2–10 year: Loading dose of 30 µg/kg Child &gt; 10 year: Loading dose of 10 µg/kg</td>
<td></td>
</tr>
<tr>
<td>Propranolol</td>
<td>Supraventricular tachycardia Children: PO: 0.5–1 mg/kg/24 hr q 6–8 hr titrated upward to 2–5 mg/kg/24 hr, over 3–5 days</td>
<td></td>
</tr>
</tbody>
</table>

Contd...
### Drug Doses

<table>
<thead>
<tr>
<th>Drug</th>
<th>Condition</th>
<th>Doses</th>
<th>Continuous infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lidocaine</td>
<td>Ventricular tachycardia</td>
<td>1 mg/kg (may repeat q 5–10 min (max. 3 mg/kg)</td>
<td>20–50 μg/kg/min (half dose for liver disease or poor cardiac output)</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>Ventricular arrhythmias, Supraventricular tachycardia</td>
<td>5 mg/kg/dose over 1 hr</td>
<td>5–10 μg/kg/min</td>
</tr>
<tr>
<td>Procainamide</td>
<td>Ventricular tachycardia, premature ventricular contractions, paroxysmal atrial tachycardia, atrial fibrillation</td>
<td>IV: 3–6 mg/kg/dose over 5 min, not to exceed 100 mg/dose; repeat q 5–10 min as needed (max. 15 mg/kg total dose). Do not exceed 500 mg in 30 min. PO: Children: 15–50 mg/kg/24 hr, q 3–6 hr; 20–30 mg/kg/24 hr, not to exceed 4 g/24 hr</td>
<td>IV infusion of 20–80 μg/kg/min</td>
</tr>
<tr>
<td>Quinidine sulphate</td>
<td>Supraventricular tachycardia, paroxysmal ventricular tachycardia, premature atrial/ventricular contractions</td>
<td>Test dose to exclude idiosyncrasy: 20–50 mg/kg/24 hr sulphate salt q 4 hr PO</td>
<td></td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>A-V nodal block</td>
<td>0.05–2 μg/kg/min</td>
<td></td>
</tr>
</tbody>
</table>

### Cerebral Edema

<table>
<thead>
<tr>
<th>Drug</th>
<th>Doses</th>
<th>Maintenance dose</th>
<th>Continuous infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mannitol</td>
<td>IV: 200 mg/kg test dose; initial 0.5–1 gm/kg</td>
<td>0.25–0.5 gm/kg q 4–6 hr</td>
<td></td>
</tr>
<tr>
<td>Furosemide</td>
<td>1–2 mg/kg IV or 1–4 mg/kg PO q 6–24 hr</td>
<td>Start at 0.05 mg/kg/hr and adjust dose to response</td>
<td></td>
</tr>
<tr>
<td>Glycerol</td>
<td>PO: 1.5 gm/kg/day q 4 hr</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dexamethasone</td>
<td>PO, IM, IV: Loading dose of 1–2 mg/kg</td>
<td>1–1.5 mg/kg/24 hr divided q 4–6 hr, tapered over 1–6 week</td>
<td></td>
</tr>
<tr>
<td>Acetazolamide</td>
<td>5 mg/kg/24 hr IV or PO</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Cardiopulmonary Resuscitation

<table>
<thead>
<tr>
<th>Drug</th>
<th>Doses</th>
<th>Continuous infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenaline</td>
<td>• SC: 0.01 mg/kg (0.01 ml/kg/dose of 1:1,000 solution)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• IV: 0.01 mg/kg [0.1 ml/kg of 1:10,000 solution (max. 1 mg)]</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• IT: 0.1 mg/kg/dose (0.1 ml/kg of 1:1,000 solution) (max. 0.2 ml/kg)</td>
<td></td>
</tr>
<tr>
<td>Atropine</td>
<td>0.02 mg/kg (minimum: 0.1 mg); IV or intratracheal (max. 0.5 mg); may repeat 5 min later, 1×</td>
<td></td>
</tr>
<tr>
<td>Naloxone</td>
<td>IV: 0.1 mg/kg (max. 2 mg). If no response, repeat q 2–3 min until desired effect</td>
<td></td>
</tr>
<tr>
<td>Albumin</td>
<td>0.5–1 gm/kg/dose (max. 6 gm/kg/24 hr)</td>
<td></td>
</tr>
<tr>
<td>Glucose</td>
<td>0.5–1 g/kg IV/IO</td>
<td></td>
</tr>
<tr>
<td>Calcium gluconate</td>
<td>60–100 mg/kg/dose</td>
<td></td>
</tr>
<tr>
<td>Sodabicarb</td>
<td>1–2 mEq/kg/dose IV/IO slowly</td>
<td></td>
</tr>
</tbody>
</table>

Anaphylaxis

<table>
<thead>
<tr>
<th>Drug</th>
<th>Doses</th>
<th>Continuous infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenaline</td>
<td>0.01 ml/kg/dose of 1:1000 solution up to 0.3 ml IM 0.01 ml/kg/dose of 1:10,000 slow IV push</td>
<td></td>
</tr>
<tr>
<td>Diphenhydramine</td>
<td>1.25 mg/kg up to 50 mg IM</td>
<td></td>
</tr>
<tr>
<td>Cetirizine</td>
<td>0.25 mg/kg up to 10 mg PO</td>
<td></td>
</tr>
<tr>
<td>Ranitidine</td>
<td>1 mg/kg up to 50 mg IV</td>
<td></td>
</tr>
<tr>
<td>Cimetidine</td>
<td>4 mg/kg up to 200 mg IV</td>
<td></td>
</tr>
<tr>
<td>Prednisolone</td>
<td>1 mg/kg up to 75 mg PO</td>
<td></td>
</tr>
<tr>
<td>Methylprednisone</td>
<td>1–2 mg/kg up to 125 mg IV</td>
<td></td>
</tr>
<tr>
<td>Asthalin</td>
<td>Resp. solution 0.5%, 0.01–0.03 ml/kg</td>
<td></td>
</tr>
</tbody>
</table>

Antihypertensive

<table>
<thead>
<tr>
<th>Drug</th>
<th>Doses</th>
<th>Continuous infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nifedepine</td>
<td>PO/sublingual 0.25–0.5 mg/kg/dose q 4–6 hr (max 1–2 mg/kg/day)</td>
<td></td>
</tr>
<tr>
<td>Amlodipine</td>
<td>0.1–0.6 mg/kg/24 hr</td>
<td></td>
</tr>
<tr>
<td>Nicardipine</td>
<td>1–3 µg/kg/min</td>
<td></td>
</tr>
<tr>
<td>Furosemide</td>
<td>1 mg/kg/dose up to 6 mg/kg/24 hr</td>
<td></td>
</tr>
<tr>
<td>Hydrochlorothiazide</td>
<td>1–2 mg/kg/24 hrs max 200 mg/24 hr</td>
<td></td>
</tr>
</tbody>
</table>
### Contd...

<table>
<thead>
<tr>
<th>Drug</th>
<th>Doses</th>
<th>Continuous infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Captopril</td>
<td>0.15–0.5 mg/kg/dose up to 6 mg/kg/24 hr</td>
<td></td>
</tr>
<tr>
<td>Enalapril</td>
<td>0.1–0.5 mg/kg/24 hr</td>
<td></td>
</tr>
<tr>
<td>Esmolol</td>
<td></td>
<td>50–300 µg/kg/min</td>
</tr>
<tr>
<td>Phentolamine</td>
<td>0.05–0.1 mg/kg/dose, max IV q 1–2 hr</td>
<td></td>
</tr>
<tr>
<td>Phenoxybenzamine</td>
<td>0.2–2 mg/kg/24 hr PO q 8–12 hr</td>
<td></td>
</tr>
<tr>
<td>Prazosin</td>
<td>0.005–0.1 mg/kg/dose PO q 6–12 hr</td>
<td></td>
</tr>
<tr>
<td>Atenolol</td>
<td>1–2 mg/kg/24 hr PO OD</td>
<td></td>
</tr>
<tr>
<td>Propranolol</td>
<td>0.01–0.1 mg/kg/dose IV q 6–8 hr</td>
<td>0.5–8 mg/kg/24 hr PO</td>
</tr>
<tr>
<td>Labetalol</td>
<td>0.2–1 mg/kg/dose IV 1–3 mg/kg/24 hr PO 6–12 hr</td>
<td>0.25–2 mg/kg/hr</td>
</tr>
<tr>
<td>Clonidine</td>
<td>0.005–0.025 mg/kg/24 hr PO q 6–12 hr</td>
<td></td>
</tr>
<tr>
<td>Sodium nitropru side</td>
<td></td>
<td>0.3–0.5 µg/kg/min; titrate dose to desired effect; rarely requires &gt; 6 µg/kg/min (probable max. 8 µg/kg/min)</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>0.1–0.4 mg/kg/dose IV q 4–6 hs</td>
<td>0.25–1 mg/kg/dose and max. 200 mg/24 hr PO q 6–12 hr</td>
</tr>
</tbody>
</table>

### Organophosphorus Poisoning

<table>
<thead>
<tr>
<th>Drug</th>
<th>Doses</th>
<th>Continuous infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine</td>
<td>0.02–0.05 mg/kg q 10–20 min until atropine effect (tachycardia, mydriasis, fever), then q 1–4 hr for at least 24 hr</td>
<td></td>
</tr>
<tr>
<td>Pralidoxime (PAM)</td>
<td>Children: 20–50 mg/kg/dose repeated in 1–2 hr if muscle weakness has not been relieved; when desired effect obtained, dose q 12 hr</td>
<td></td>
</tr>
</tbody>
</table>

### Thrombolytic

<table>
<thead>
<tr>
<th>Drug</th>
<th>Doses</th>
<th>Continuous infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heparin</td>
<td>50 U/kg/dose</td>
<td>15–35 U/kg/hr</td>
</tr>
<tr>
<td>Warfarin</td>
<td>0.2 mg/kg/dose PO, then 0.1 mg/kg/day OD</td>
<td></td>
</tr>
<tr>
<td><strong>Antibiotics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----------------</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td><strong>Drug</strong></td>
<td><strong>Doses</strong></td>
<td><strong>Continuous infusion</strong></td>
</tr>
<tr>
<td>Aminoglycoside</td>
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<tr>
<td>Amikacin</td>
<td>15–25 mg/kg/24 hr divided q 8–12 hr</td>
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<tr>
<td>Gentamicin</td>
<td>2.5 mg/kg/24 hr divided q 8–12 hr or 5–7.5 mg/kg/24 hr IV once daily</td>
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<tr>
<td>Netilmicin</td>
<td>4 mg/kg OD</td>
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<tr>
<td>Streptomycin</td>
<td>15 mg/kg OD</td>
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<tr>
<td>Tobramycin</td>
<td>2.5 mg/kg/24 hr divided q 8–12 hr or 5–7.5 mg/kg/24 hr IV</td>
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<td>Cephalosporin</td>
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<td>Ceftriaxone</td>
<td>75–100 mg/kg/24 hr divided q 6–8 hr IV or IM</td>
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<tr>
<td>Ceftazidime</td>
<td>90–150 mg/kg/24 hr divided q 8 hr IV or IM</td>
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<tr>
<td>Cefotaxime</td>
<td>100–200 mg/kg/24 hr divided q 6–8 hr IV or IM (meningitis: 200 mg/kg/24 hr divided q 6–8 hr IV)</td>
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<tr>
<td>Cefoperazone</td>
<td>100–150 mg/kg/24 hr divided q 8–12 hr IV or IM</td>
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<tr>
<td>Lincosamide</td>
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<tr>
<td>Clindamycin</td>
<td>10–40 mg/kg/24 hr divided q 6–8 hr IV, IM or PO</td>
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<tr>
<td>Penicillin</td>
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<tr>
<td>Amoxicillin</td>
<td>20–50 mg/kg/24 hr divided q 8–12 hr PO 80–90 mg/kg 24 hr, PO for otitis media</td>
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<tr>
<td>Amoxicillin + Clavulnic acid</td>
<td>20–45 mg/kg 24 hr divided q 8–12 hr PO 80–90 mg/kg/24 hr, PO for otitis media</td>
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<tr>
<td>Cloxacillin</td>
<td>50–100 mg/kg/24 hr divided q 6 hr PO</td>
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<tr>
<td>Piperacillin + Tazobactam</td>
<td>300–400 mg/kg/24 hr divided q 6–8 hr IV or IM</td>
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<tr>
<td>Penicillin G</td>
<td>100,000–250,000 units/kg/24 hr divided q 4–6 hr IV or IM</td>
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<tr>
<td>Ticarcillin</td>
<td>200–400 mg/kg/24 hr divided q 4–6 hr IV; cystic fibrosis: 400–600 mg/kg/24 hr IV</td>
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<tr>
<td>Miscellaneous</td>
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<td>Fluconazole</td>
<td>6 mg/kg/day OD</td>
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<tr>
<td>Amphotericin B</td>
<td>0.25–1.5 mg/kg/day OD (water soluble) 5 mg/kg/day OD (liposomal)</td>
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<tr>
<td>Ciprofloxacin</td>
<td>15–30 mg/kg/24 hr divided q 12 hr PO or IV; cystic fibrosis: 20–40 mg/kg/24 hr divided q 8–12 hr PO or IV</td>
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<tr>
<td>Gatifloxacine</td>
<td>10 mg/kg/day OD</td>
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<tr>
<td>Ofloxacin</td>
<td>15 mg/kg/day q 12 hr</td>
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<tr>
<td>Levofoxcin</td>
<td>10 mg/kg/day q 12 hr &gt; 5 years, OD &lt; 5 years</td>
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<th>Drug</th>
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<tr>
<td>Imipenem +</td>
<td>60–100 mg/kg/24 hr divided q 6–8 hr IV or IM</td>
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<td>Cilastatin</td>
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<tr>
<td>Meropenem</td>
<td>60 mg/kg/24 hr divided q 8 hr IV, meningitis: 120 mg/kg/24 hr</td>
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<tr>
<td>Metronidazole</td>
<td>30 mg/kg/24 hr divided q 6–8 hr PO or IV</td>
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<tr>
<td>Vancomycin</td>
<td>45–60 mg/kg/24 hr divided q 8–12 hr IV</td>
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<tr>
<td>Colistin</td>
<td>50,000 IU/Kg/day divided in three doses</td>
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<tr>
<td>Polymyxin B</td>
<td>&lt; 2 years: 15000–45,000 U/kg/day q 12 hr</td>
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</tr>
<tr>
<td></td>
<td>&gt; 2 years: 15,000–25,000 U/kg/day q 12 hr</td>
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