SRB’s Manual of Surgery

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Forewords
Prakash Rao
Thangam Verghese Joshua
God could not be everywhere all the time, so, He created Mothers

This book is in the memory of my mother

Late Mrs Devaki Krishna Bhat

who continues to inspire and guide me
A true surgeon is never fearless; he fears for his patients, he fears for his shortcomings, his own mistakes, but never fears for himself or his professional reputation.

—Samuel J Mixter

The manual is written for the students in this firing line for their MBBS degree. Common conditions seen on a day-to-day basis have been dealt with in a little more detail. The uncommon and rare conditions have been dealt with briefly. The mode of clinical presentation and the physical signs are mentioned vividly so as to register in the mind of the reader. There is a brief mention of necessary investigations in each of the surgical condition. The accepted mode of recent advances in investigation and treatment has been included. This manual is meant for Final MBBS degree examination and not a consolidated textbook. Certain chapters are included at the end of the book for the more enthusiastic students and for those interested in participating in quiz programmes.

The clinical photographs are of very good quality, which are self-explanatory of the conditions. The clinching physical signs have been highlighted in box forms with different shades of colour. This mode of presentation emphasises the point to be conveyed to the students which they can recollect when required either while arriving at a diagnosis in their examination or while in practice after obtaining the degree. This book gives a good foundation in surgery for those who wish to pursue surgery as a career.

What does the surgeon ought to be?

The conditions necessary for the surgeon are four—first, he should be learned; second, he should be an expert; third, he must be ingenious; and the fourth, he should be able to adapt himself.

Therefore, as briefly mentioned above, the first and foremost, it is required that the surgeon should know not only the principles of surgery, but also those of medicine in theory and practice; second, he should have seen others operate; third, he should be ingenious, of good judgement and memory to recognise conditions; and the fourth, he should be adaptable and able to accommodate himself to circumstances.

Let the surgeon be bold in all sure things, and fearful in dangerous things, let him avoid all faulty treatments and practices. He ought to be gracious to the sick, considerate to his associates, cautious in his prognostications. Let him be modest, dignified, gentle, pitiful, merciful; neither covetous nor an extortionist of money: but rather let his reward be according to his work, to the means of the patient, to the quality of the issue, and to his own dignity.

—Ars Chirurgica

I thank Sriram Bhat M for giving me this privilege of writing a foreword to his excellent manual.

Prakash Rao MS
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SRB’s Manual of Surgery has helped innumerable young doctors in the making, to start their voyage on a sea of surgical knowledge, well equipped with a sense of direction. This book is in its fifth edition, which speaks volumes about its popularity among the medical students. The first edition came out in 2004. It soon gained nationwide recognition as a comprehensible source of information. The text and the style of writing is easy to understand and to remember, the accompanying diagrams and photographs are perceivable and unforgettable—a combination which is so very important to a student facing theory and practical examinations.

With each new edition, this manual has been updated and upgraded with a lot of dedication by the author. The newer concepts of treatment and staging of malignancies have been documented. Surgical anatomy has been included wherever the author deemed it essential. The book contains all the important chapters of general surgery, the common conditions have been dealt with in detail. The reader is also introduced to the various surgical specialties and is imparted with a basic yet-practical knowledge, which is an essential armament to both—the student of surgery as well as to the young practicing surgeon.

I consider it a great privilege to be given the honour of writing a foreword to this manual, which has been authored by Dr Sriram Bhat M, a favourite student of mine. He is a great teacher, a skilful surgeon, renowned author of several surgical books, but above all a good human being. In spite of all the fame that rests lightly on his shoulders, he remains humble; in spite of being knowledgeable, he remains eager to learn, and in spite of being busy, he gives quality time to his family and friends.

I wish Dr Sriram Bhat M, the best in his endeavours of inspiring generations of competent surgeons. I also wish the students who read this book to be greatly benefited, and perhaps one-day surpass their teachers.

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It is my pleasure to release the fifth edition, three years after the release of fourth edition of *SRB’s Manual of Surgery*. As my earlier editions were well accepted by the students, I intend to bring out the fifth edition in the intention of updating the treatment strategies of the needed surgical conditions. Extensive corrections, elaboration of a few topics, and updating of staging and grading of malignant conditions as per new standards have been done in this edition. I have referred many books, journals and took the help of my colleagues in surgery and other departments. I have retained all chapters with some rearrangements of a few chapters and topics.

For better understanding of the students, three sizes of fonts are used namely—10pt, 9pt, and 8.5pt. Important subjects are kept in font size 10pt; other subjects are in 9pt; and notes, remember, boxes and tables are in 8.5pt. This will be useful especially for undergraduates to understand which topics have got priorities.

I sincerely express my thanks to everybody who have helped me and also to the publishers who are the backbone of this upgraded edition. I hope this edition will be well accepted by the teachers, surgeons, undergraduates and postgraduates of surgery department. I sincerely welcome all criticisms.

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This book is born out of a desire to provide a complete, authoritative, current textbook on surgery. For the past fifteen years of my teaching profession, watching students hurriedly jotting down notes during classes, an urge was born within me to put my thoughts in print. I am fortunate to have been guided by my teachers and supported by my students towards this goal.

An attempt has been made to make this book simple and reader-friendly while not compromising on the aspect of providing students with necessary information and a better clinical understanding of surgery. Recent advances in surgery till date have been included to the best of my knowledge. Text has been presented in a simple and lucid language so as to help students understand and recapitulate the subject better. Since a picture is worth a thousand words, over 500 photographs, X-rays and illustrations have been incorporated in this work to make surgery more interesting and understandable. Inspirational quotes have been interspersed to motivate students to go the extra mile.

Discussion on Instruments and Operative Procedures has been dealt with in necessary detail separately. A chapter containing a list of interesting new developments in surgery and uniquely new topics like Fascinating Signs, Misnomers, Triads and other interesting chapters have been included.

While this book has been compiled with undergraduates in mind, I am confident that it will serve as a useful reference for postgraduates and practitioners.

In compiling this book I have consulted many authoritative books and publications and I sincerely express my appreciation and gratitude to all of them.

Suggestions and constructive criticisms towards improving this book in subsequent editions are always welcome.

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I am happy to bring out one more edition (fifth edition) of the book *SRB's Manual of Surgery*. This is due to constant help and support of many.

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Shock

Shock is a state of poor perfusion with impaired cellular metabolism manifesting with severe pathophysiological abnormalities. It is due to circulatory collapse and tissue hypoxia. Normal aerobic metabolism is not maintained due to hypoperfusion. Shock is meant by ‘inadequate perfusion’ to maintain normal organ function.

At cellular level hypoxia causes change of normal aerobic to anaerobic metabolism causing lactic acidosis. Intracellular potassium is released into circulation. Lysosomes from cells get released into blood causing cell lysis. Hypoxia and acidosis through complements release free oxygen radicals and cytokines which damage capillary endothelium. Eventually cardiovascular, respiratory, renal, endocrine and GIT will be affected presenting as systemic features.

Causes of shock

1. **Hypovolaemic shock**—due to reduction in total blood volume. It may be due to:
   a. Haemorrhage
      i. External from wounds, open fractures
      ii. Internal from injury to spleen, liver, mesentery or pelvis
   b. Severe burns, which results in loss of plasma
   c. Peritonitis, intestinal obstruction
   d. Vomiting and diarrhoea of any cause
2. **Cardiac causes**
   a. Acute myocardial infarction, acute carditis
   b. Acute pulmonary embolism wherein embolus blocks the pulmonary artery at bifurcation or one of the major branches
   c. Drug induced
   d. Toxaemia of any causes
   e. Cardiac surgical conditions like valvular diseases, congenital heart diseases
   f. Cardiac compression causes
      i. Cardiac tamponade due to collection of blood, pus, fluid in the pericardial space which prevents the heart to expand leading to shock
      ii. Trauma to heart
3. **Septic shock**—is due to bacterial infections which release toxins leading to shock

Shock may be hypovolaemic, cardiogenic, obstructive, distributive or of endocrine variety.

**Pathophysiology of Shock**

Any cause of shock

↓

**Low cardiac output**

Vasoconstriction occurs as a compensation to perfuse vital organs like brain, heart, kidneys, liver

Because of vasoconstriction and tachycardia

Dynamic circulation increases

Tachypnoea occurs to increase the oxygen saturation

Peripheral veins (capacitance vessels) constrict diverting blood from splanchnic system towards essential vital organs

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Decreased renal blood flow reduces the GFR and thereby the urine output.

Renin angiotensin mechanism gets activated causing further vasoconstriction and aldosterone release.

Causes salt and water retention.

ADH is released.

Further concentration of urine occurs.

When shock persists cardiac output falls further.

Hypotension and tachycardia occurs leading to poor perfusion of coronaries.

Hypoxia—metabolic acidosis.

Release of cardiac depressants.

Cardiac (pump) failure.

Hypoxia.

Anaerobic metabolism.

Lactic acidosis.

Cell wall damage.

Sodium and calcium enter the cell.

Potassium leaks out of the cell.

Causes hyperkalaemia, hyponatraemia and hypocalcaemia.

Intracellular lysosomes break down releasing powerful enzymes which destroy own cell.

SICK CELL SYNDROME.

Platelets are activated forming small clots in many places.

Disseminated intravascular coagulation (DIC) (Consumption coagulopathy).

Further bleeding.

Hypoxia and its effects.

SIRS (Systemic inflammatory response syndrome) is due to vasodilatation, increased endothelial permeability, thrombosis, leucocyte migration and activation.

All these lead to altered cytokines level, abnormal NO (nitric oxide) synthesis, abnormal arachidonic acid metabolism, neutrophil activation, free radical production, altered complement activation, failure to have a localisation of inflammation. It is severe type of reversible shock.

Which will lead to established microvascular occlusion, cellular dysfunction, sick cell syndrome, DIC and PUMP failure.

MODS (Multiorgan dysfunction syndrome) (Irreversible shock)—of lungs, kidneys, liver, clotting system and brain.

### Stages of shock

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
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<tbody>
<tr>
<td>Stage 1</td>
<td>Stage of compensatory shock by neuroendocrine response to maintain the perfusion of the vital organs like brain, heart, kidney, liver.</td>
</tr>
<tr>
<td>Stage 2</td>
<td>Stage of decompensatory shock where there is progressive shock causing persistent shock with severe hypotension (with mean arterial pressure &lt; 65 mmHg); oliguria, tachycardia.</td>
</tr>
<tr>
<td>Stage 3</td>
<td>Stage of irreversible shock with severe hypoxia and MODS.</td>
</tr>
</tbody>
</table>

**Note:**

- **Distributive shock** is one in which there is vasodilatation, decreased vascular resistance, hypotension, altered microvascular perfusion with arteriovenous shunting, altered cellular oxygen metabolism. It is seen in septic shock, spinal trauma, adrenal crisis and anaphylaxis.

- **Obstructive shock** occurs due to mechanical impediment of circulation due to pulmonary embolism, tension pneumothorax or cardiac tamponade.

### EFFECTS OF SHOCK

**Heart**: Low perfusion → low venous return → decreased cardiac output → hypotension → tachycardia. Persistent shock causes hypoxia and release of myocardial depressants leading to further cardiac damage.

**Lung**: Interstitial oedema → decreased gaseous exchange → pulmonary arteriovenous shunting → tachypnoea → Adult/Acute respiratory distress syndrome (ARDS) and pulmonary oedema.

**Metabolic**: Shock leads to hypoxia, which activates anaerobic metabolism leading to lactic acidosis. Antidiuretic hormone (ADH) is released which increases the reabsorption of water from renal tubules. Other hormones released are ACTH, prostaglandins, histamine, bradykinin, and serotonin to compensate the effects of shock to increase the perfusion of vital organs like heart, brain and lungs.

**Cellular changes** occur in persistent shock due to release of lysosomal enzymes, which alters the cell membrane permeability causing cell death—sick cell syndrome.

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*Ideas have a short shelf life—that’s why we must act before the expiry date.*
Sympathetic overactivity alters the microcirculation leading to capillary dysfunction.

Brain perfusion, when decreases the patient becomes drowsy. Brain is the last organ to get underperfused in shock.

Kidneys: GFR decreases and tubular reabsorption of salt and water increases for compensatory response. But in severe cases tubular necrosis sets in leading into irreversible damage.

Blood: Alteration in cellular components including platelets leads to Disseminated intravascular coagulation (DIC). It causes bleeding from all organs.

Gastrointestinal tract: Mucosal ischaemia develops causing bleeding from GIT with haematemesis and malaena. It is aggravated by DIC. Hepatic ischaemia leads into increased enzyme levels.

Types of Shock

1. Vasovagal Shock
   It is sudden dilatation of peripheral and splanchnic vessels causing reduced cardiac output and shock. Often it may be life-threatening due to hypoxia.

2. Neurogenic Shock
   - It is usually due to spinal cord injury, which causes dilatation of splanchnic vessels.
     - This type can safely be treated with vasoconstrictor drugs to bring up the blood pressure. There will be bradycardia, hypotension, arrhythmias, and decreased cardiac output. Blood pressure control, oxygen delivery, maintenance of haemodynamics, airway, fluid therapy, intravenous methylprednisolone therapy should be done. Dopamine and or phenylephrine (α agonist) can be used.

3. Hypovolaemic Shock—Most Common Type
   - Haemorrhage, may be due to injury to the liver, spleen, bone fractures, haemothorax, vascular injury, severe bleeding on table during surgeries of thyroid, liver, portal vein or major vessels.
   - Vomiting, diarrhoea due to any cause.
   - Burns.

<table>
<thead>
<tr>
<th>Types of Hypovolaemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Covert compensated hypovolaemia (Mild &lt;15%): When blood volume is reduced by 10-15%, there will not be significant change in heart rate, cardiac output and splanchnic blood compensates for the same.</td>
</tr>
<tr>
<td>b. Overt compensated hypovolaemia (Moderate 15–40%): Here patient has cold periphery, tachycardia, a wide arterial pressure, tachypnoea, confusion, hyponatremia, metabolic acidosis, but systolic pressure is well-maintained but postural hypotension.</td>
</tr>
<tr>
<td>c. Decompensated hypovolaemia (Severe &gt;40%): Here all features of hypovolaemia are present like hypotension, tachycardia, sweating, tachypnoea, oliguria, drowsiness, eventually features of SIRS is seen and often if not treated on time leads to MODS, i.e. irreversible shock.</td>
</tr>
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</table>

4. Cardiogenic Shock
   - Cardiogenic shock is defined as circulatory failure causing diminished forward flow leading into tissue hypoxia in the setting of adequate intravascular volume with systolic blood pressure <90 mmHg for 30 minutes; cardiac index <2.2 L/minute/sq metre; raised PCWP (pulmonary capillary wedge pressure) >15 mmHg. It is commonly seen in acute MI with a mortality >50%.
   - Cardiogenic shock develops within 24 hours of MI. It occurs when 50% of left ventricular wall is damaged by infarction.
   - It leads to pulmonary oedema and severe hypoxia. Ischaemic necrosis of left ventricular wall causes failure of pump thereby decreasing stroke volume.

Diagnosis is established by ECG, echocardiography, arterial blood gas analysis, cardiac enzymes, PCWP and electrolyte estimation (hypokalaemia and hypomagnesaemia are common) are the essential investigations.

Management

- Proper oxygenation with intubation, ventilator support, cardioversion, pacing, antiarrhythmic drugs, correction of electrolytes, avoiding fluid overload, prevention of pulmonary oedema as immediate measures.
- Dobutamine (β1 receptor agonist) is used to raise cardiac output provided there is adequate preload and intravascular volume (it is peripheral vasodilator and reduces BP). Dopamine is preferred in patients with hypotension. But it may increase peripheral resistance and heart rate worsening cardiac ischaemia. Often both dopamine and dobutamine combination may be required.
- Careful judicious use of epinephrine, norepinephrine, phosphodiesterase inhibitors (amrinone, milrinone) are often needed. Anticoagulants and aspirin are given. Thrombolytics can be used. β blockers, nitrates (nitroglycerine causes coronary arterial dilatation), ACE inhibitors are also used. Intra-aortic balloon pump (IABP) may need to be introduced transfemorally as a mechanical circulatory support to raise cardiac output and coronary blood flow.
- Relief of pain, preserving of remaining myocardium and its function, maintaining adequate preload, oxygenation, minimizing sympathetic stimulation, correction of electrolytes should be the priorities.
- Percutaneous transluminal coronary angioplasty (PTCA) and coronary artery bypass graft (CABG) are the final choices.

5. Cardiac Compression Shock
   - It is probably due to pericardial tamponade of any cause or kinking of great vessels, massive pulmonary embolism, tension pneumothorax, air embolism causes obstructive shock with reduced preload to heart.
   - Acute massive pulmonary embolism from a thrombus or an air embolism (50 ml of air), obstructing more than 50% of pulmonary vasculature leads to severe shock and sudden death.
   - Tachycardia, hypotension, pulmonary oedema, raised JVP, gallop rhythm are the features.

6. Septic Shock
   - Septic shock may be due to gram-positive organisms, gram negative organisms, fungi, viruses or protozoal origin.
   - Gram-negative septicaemia/gram-negative septic shock is called as endotoxic shock. It occurs due to gram-negative bacterial infections, commonly seen in strangulated intestines, peritonitis, gastrointestinal fistulas, biliary and urinary infections, pancreatitis, major surgical wounds, diabetic wounds and crush injuries.
**Gram-positive septic shock**
- Due to exotoxin by gram +ve bacteriaemia like *Clostridium tetani/welchii*, *staphylococci*, *streptococci pneumoniae*
- Fluid loss, hypotension is common; with normal cardiac output

**Gram-negative septic shock**
- Gram negative bacteria cause endotoxaemia and its effects.
- Urinary/gastrointestinal/biliary and respiratory foci are common

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Pathophysiology of septic shock

Toxins/endotoxins from organisms like *E. Coli*, *Klebsiella*, *Pseudomonas*, and *Proteus*.

- Inflammation, cellular activation of macrophages, neutrophils, monocytes
- Release of cytokines, free radicals
- Chemotaxis of cells, endothelial injury, altered coagulation cascade—SIRS

Reversible hyperdynamic warm stage of septic shock with fever, tachycardia, tachypnoea

- Severe circulatory failure with MODS (failure of lungs, kidneys, liver, heart) with DIC

Hypodynamic, irreversible cold stage of septic shock.

- Septic shock is typically a vasodilatory shock wherein there is peripheral vasodilation causing hypotension which is resistant to vasopressors. This is due to toxin-induced release of isofrom of nitric oxide synthetase from the vessel wall which causes sustained prolonged release of high levels of nitric oxide.

- Magnitude of infection is quantified as:
  1. *Sepsis* which shows fever, tachycardia, leucocytosis.
  2. *Severe sepsis* which shows low tissue perfusion with organ dysfunction (lactic acidosis, dysfunction of liver, kidney, lungs).
  3. *Septic shock* with systemic hypotension (BP < 90 mmHg in spite adequate fluid therapy), severe organ dysfunction (acute lung, kidney, liver, heart), maldistribution of blood flow, shunting in microcirculation.

Stages of septic shock

a. **Hyperdynamic (warm) shock**:
This stage is reversible stage. Patient is still having inflammatory response and so presents with fever, tachycardia, and tachypnoea. Pyrogenic response is still intact. Patient should be treated properly at this stage. Based on blood culture, urine culture (depending on the focus of infection), higher antibiotics like third generation cephalosporins, aminoglycosides, metronidazole are started. The underlying cause is treated like draining the pus, laparotomy for peritonitis, etc.

b. **Hypodynamic hyperpoloaenic septic shock (cold septic shock)**:
Here pyrogenic response is lost. Patient is in decompenated shock. It is an irreversible stage along with MODS (Multi-organ dysfunction syndrome) with anuria, respiratory failure (cyanosis), jaundice (liver failure), cardiac depression, pulmonary oedema, hypoxia, drowsiness, eventually coma and death occurs (Irreversible stage).

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**Treatment of septic shock**

- Correction of fluid and electrolyte by crystalloids, blood transfusion. **Perfusion** is very/most important.
- Appropriate antibiotics—third generation cephalosporins/aminoglycosides.
- Treat the cause or focus—drainage of an abscess; laparotomy for peritonitis; resection of gangrenous bowel; wound excision.
- Pus/urine/discharge/bile/blood culture and sensitivity for antibiotics.
- Critical care, oxygen, ventilator support, dobutamine/dopamine/noradrernine to maintain blood pressure and urine output.
- Activated C protein prevents the release of inflammatory mediators and blocks the effects of these mediators on the cellular function.
- Monitoring the patient by pulse oximetry, cardiac status, urine output, arterial blood gas analysis.

**Septic shock**

- Common causes are biliary, urinary, GIT sepsis (peritonitis, strangulation), respiratory (pneumonia)
- Common bacteria are *E. coli*, *Klebsiella*, *Pseudomonas*
- Common pathophysiologicals are release of toxins, neutrophil activation, cytokine release, and sick cell syndrome, SIRS, MODS
- Clinical stages are hyperdynamic amd hypodynamic
- Find out the source of the infection by US, CT scan
- Do pus/blood/urine culture
- Start antibiotics of high generations like cefazidime, amikacin, cefoperazone
- Dopamine/dobutamine infusion (slow)
- Monitoring by pulse, BP, respiration, urine output, level of consciousness
- Ventilator support, ICU management
- Treat the causes like peritonitis, abscess

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**Anaphylactic Shock**

Injections—penicillins, anaesthetics, stings, venom, shellfish may be having antigens which will combine with IgE of mast cells and basophils, releasing histamine and large amount of SRS-A (Slow releasing substance of anaphylaxis). They cause bronchospasm, laryngeal oedema, respiratory distress, hypotension and shock. Mortality is 10%. Rashes all over the body are commonly observed.

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**Tears shed for self are tears of weakness, but tears shed for others is a sign of strength.**
Anaphylactic shock
- Sudden onset
- Distributive shock
- Bronchospassm, laryngeal oedema
- Generalised rashes and oedema
- Hypotension, feeble pulse
- Mortality 10%
- To start adrenaline 100 ug IV, steroids, IV fluids, oxygen with foot end elevation
- Ventilator in severe cases
- Cardiac massage, defibrillation

CLINICAL FEATURES OF SHOCK (HYPOVOLAEMIC SHOCK)
- In early stage—tachycardia, sweating, cold periphery, hypotension, restlessness, air hunger, tachypnoea, oliguria, collapsed veins.
- In late stage—cyanosis, anuria, jaundice, drowsiness.

Clinically shock may be:
- Compensated with mild tachycardia, normal blood pressure, urine output, normal respiration and mild lactic acidosis.
- Mild shock with mild lactic acidosis, tachycardia, tachypnoea and anxioinss.
- Moderate shock with significant lactic acidosis, decreased urine, tachycardia, tachypnoea, drowsiness, and mild hypotension.
- Severe shock with severe lactic acidosis, anuria, tachypnoea with gasping, severe tachycardia, profound hypotension and unconsciousness.

Note:
- Shock index is ratio of pulse rate to blood pressure; normal shock index is < 1. In shock it reverses.
- Tachycardia, tachypnoea, oliguria, hypotension are typical features in shock.

Complications of shock
- Acute respiratory distress syndrome and respiratory failure
- Acute renal failure
- Hypoxia, metabolic acidosis
- Stress ulcers, ileus, liver failure
- Disseminated intravascular coagulation (DIC) and thrombocytopenia
- Systemic inflammatory response syndrome and multiorgan dysfunction syndrome (MODS)

ASSESSMENT, INVESTIGATIONS AND MONITORING
- Regular monitoring with blood pressure, pulse, heart rate, respiratory rate, urine output measurement (hourly) should be done. Urine output should be more than 0.5 ml/kg/hour. Pulse oximetry should be used.
- Central venous pressure (CVP)—only have gross assessment), pulmonary capillary wedge pressure (PCWP—an accurate assessment of left ventricular function) monitoring should be done. ICU care is needed during monitor period. But both CVP and PCWP are not accurate method of assessing tissue perfusion.
- Complete blood count, ESR, pH assessment, serum electrolyte estimation, chest X-ray (to rule out ARDS/pulmonary problems).
- Pus/urine/blood/bile/sputum cultures depending on the focus and need in sepsis.
- Serum lactate estimation is an important prognostic factor. Level >2 mEq/L suggest tissue ischaemia.
- USG of part, CT/MRI of the location of the pathology of the septic focus should be done; often may require repetition of these imaging to assess progress.
- Blood urea, serum creatinine, liver function tests, prothrombin time (PT), activated partial thromboplastin time (APTT), ECG monitoring are also should be done.
- All these tests including platelet count and arterial blood gas analysis (ABG) should be repeated at regular intervals.

Treatment of Shock

Guidelines
- To treat the cause
- To improve cardiac function
- To improve tissue perfusion

Treatment of shock
- First stabilize the patient with initial resuscitation
- Next evaluate the patient for cause and severity
- Lastly treat the specific cause to achieve cure
- Treat the cause, e.g. arrest haemorrhage, drain pus.

Fluid replacement:
- Plasma, normal saline, Ringer’s lactate, plasma expander (haemaccel) (maximum 1 litre can be given in 24 hours).
- Initially crystalloids then colloids are given. Blood transfusion is done whenever required.
- Fluid therapy is ideally done with crystalloids like normal saline, Ringer’s lactate, Hartmann’s solution. Blood loss should be corrected by blood transfusion only. Crystalloids and colloids do not have O2 carrying capacity. Hypotonic solutions like dextrose are poor volume expanders and so should not be used in shock.
- Dynamic fluid response is studied by infusing 500 ml of fluid rapidly in 10 minutes. Responders show improvement; transient responders show improvement temporarily but revert back to original status probably due to still existing fluid/blood loss or still existing fluid shift from intravascular space; nonresponders will not respond as fluid loss is severe and persistently ongoing.
- Inotropic agents: Dopamine, dobutamine, adrenaline infusions—mainly in distributive shock like septic shock.

Note:
- Correction of acid-base balance: Acidosis is corrected by using 8.4% sodium bicarbonate intravenously.
- Steroid is often life-saving. 500–1000 mg of hydrocortisone can be given. It improves the perfusion, reduces the capillary...
leakage and systemic inflammatory effects.

- **Antibiotics** in patients with sepsis; proper control of blood sugar and ketosis in diabetic patients.
- **Catheterisation** to measure urine output (30–50 ml/hour or >0.5 ml/kg/hour should be maintained).
- **Nasal oxygen** to improve oxygenation or ventilator support with intensive care unit monitoring has to be done.
- **CVP line** to perfuse adequately and to monitor fluid balance. TPN is given when required.
- **PCWP** to monitor very critical patient.
- **Haemodialysis** may be necessary when kidneys are not functioning.
- **Control pain** using morphine (4 mg IV).
- **Ventilator and ICU/critical care management**.
- **Injection ranitidine IV or omeprazole IV or pantoprazole IV**.
- **Activated C protein** even though costly is beneficial as it prevents the release and action of inflammatory response.
- **MAST** (military antishock trouser): Provides circumferential external pressure of 40 mmHg. It is wrapped around lower limbs and abdomen, and inflated with required pressure. It redistributes the existing blood and fluid towards centre. It should be deflated carefully and gradually.

**Remember**

- Response to dynamic fluid therapy can be checked in patient in shock by perfusing 500 ml of fluid rapidly in 10 minutes and patient is checked as nonresponder/transient only initial responder or proper responder.
- **Vasopressor** like dobutamine is used only in distributive shock like due to sepsis (not in hypovolaemic, haemorrhagic shock where there is low preload).
- **Intubation and ventilator** may be needed in shock.
- **The patient is monitored with ECG, pulse oximetry, blood pressure/invasive blood pressure, CVP/PCWP, urine output, papillary reaction (dilated or not), serum electrolytes, arterial PO2 and PCO2 analysis.**

**CENTRAL VENOUS PRESSURE (CVP)**

It is a method to measure the right atrial pressure by placing a venous catheter (20 cm) into the SVC (superior vena cava). Commonly for CVP monitoring, a venous catheter is passed through internal jugular vein or infraclavicular subclavian vein to the SVC (used for TPN purpose). Occasionally a long catheter (60 cm) can be passed through basilic vein (not commonly done). Under radiological guidance, initially a needle is passed 3 cm above the medial end of the clavicle, in the hollow between the two heads of sternomastoid muscles, directing towards the suprasternal notch into the right internal jugular vein. Then through a guide wire, a venous catheter is passed into the SVC through right internal jugular vein, which can also be confirmed by changes in flow during inspiration and expiration.

Catheter is connected to saline manometer, taking manubriosternal angle (angle of Louis) as zero point. Normal value is 2–10 cm of saline.

If less than 2 cm, more fluid is infused. If more than 10 cm, fluid infusion should be restricted.

**Complications of CVP**

- Pneumothorax
- Haemothorax
- Injury to brachial plexus and vessels
- Bleeding
- Sepsis
- Catheter displacement

Excuses are the nails used to build a house of failure.
PULMONARY CAPILLARY WEDGE PRESSURE (PCWP)

It is a better indicator of circulating blood volume and left ventricular function.

Catheter used is Swan Ganz triple channel pulmonary artery balloon catheter.

**It is used to:**
- Differentiate right and left ventricular failure, pulmonary embolus, septic shock
- To measure and monitor cardiac output during the use of inotropic agents, vasodilators and fluid therapy

**Procedure**

Under strict aseptic precaution, using cannula and guide wire, catheter is passed through internal jugular vein, into the right atrium. Balloon is inflated by 1.5 ml of air and then negotiated into pulmonary artery, until it reaches a small branch and wedges it. Pressure at this point is called as pulmonary capillary wedge pressure.

PCWP normally is 8–12 mmHg, considering mid axillary point as zero reference point.

After that, balloon is deflated to get pulmonary artery pressure which is normally 25 mmHg systolic and 10 mmHg diastolic.

PCWP catheter can be kept in situ only for 72 hours.

**Complications**
- Arrhythmias
- Pulmonary artery rupture
- Balloon rupture
- Pulmonary infarction
- Pneumothorax
- Haemothorax
- Bleeding, sepsis, thrombosis

**Differences between CVP and PCWP**

<table>
<thead>
<tr>
<th>CVP</th>
<th>PCWP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Technically easier</td>
<td>1. Requires skilled experts</td>
</tr>
<tr>
<td>2. Normal pressure is 2–10 cm of saline</td>
<td>2. 8–12 mmHg</td>
</tr>
<tr>
<td>4. Left ventricular function is not assessed</td>
<td>4. Left ventricular function is very well-assessed</td>
</tr>
<tr>
<td>5. Not used to differentiate between right and left ventricular function</td>
<td>5. Very well-differentiated</td>
</tr>
<tr>
<td>6. Can be kept in situ as long as desired</td>
<td>6. Cannot be kept in situ for more than 72 hours</td>
</tr>
<tr>
<td>7. Catheter tip is in SVC</td>
<td>7. Catheter tip is in pulmonary capillary with wedging</td>
</tr>
<tr>
<td>8. Plain tip catheter</td>
<td>8. 1.5 ml air filled balloon tip</td>
</tr>
<tr>
<td>9. Can be used for TPN, fluid infusion, etc.</td>
<td>9. Can not be used for TPN, or fluid infusion</td>
</tr>
<tr>
<td>10. Complications are easy to tackle</td>
<td>10. Often difficult to tackle</td>
</tr>
<tr>
<td>11. Not as sensitive and specific as PCWP</td>
<td>11. Sensitive and specific</td>
</tr>
</tbody>
</table>

**SYSTEMIC INFLAMMATORY RESPONSE SYNDROME (SIRS)**

- SIRS is systemic manifestations of inflammation due to variety of causes like infection, pancreatitis, polytrauma, burns, transfusion reaction, and malignancy. So it is often categorised as infectious cause SIRS or noninfectious cause SIRS. It causes either hyperthermia (>38°C) or hypothermia (<36°C); tachycardia (pulse >90/minute); tachypnoea (>20/minute); total white cell count >12,000/cu mm, or count <4000/cu mm.
- It is final common pathway in shock due to any cause (trauma, sepsis, endotoxaemia, burns) where there is failure of inflammatory localisation with vasodilatation, increased endothelial permeability with damage, thrombosis, leucocyte migration and activation.
- It is associated with release of free radicals, abnormal arachidonic acid release, cytokine release, neutrophil sequestration, abnormal NO synthesis, complement activation, DIC.

**Fig. 1.210**: Pulmonary capillary wedge pressure. Note the wedged balloon in the tip of the venous catheter in the pulmonary arteriole.
It is a part of severely decompensated reversible shock which eventually leads to MODS (Multiorgan dysfunction syndrome), a state of irreversible shock wherein patient is anuric, drowsy, cold and terminally ill.

SIRS carries poor prognosis.

**MULTIPLE ORGAN DYSFUNCTION SYNDROME (MODS)**

- It is progressively becoming irreversible injury of all tissues like kidney, lungs, liver, GIT. Lungs and liver are commonly involved (70%). Next organs to be involved are kidney and GIT. Order of involvement of organs in MODS is—lungs → right ventricular failure → liver → kidney. But mortality is higher if kidney and lungs are involved.
- It occurs in critically ill patient after severe trauma, burns, acute pancreatitis, bleeding and sepsis.
- It is more common in elderly, diabetic, smokers, alcoholics, cirrhosis, malnutrition, patients under steroids and cytotoxic drugs, and uraemia.
- Multiple features related to multiple organ dysfunctions are typical. Oliguria, jaundice, hypotension, drowsiness, respiratory distress are common.
- Platelet microaggregation, acute pulmonary hypertension, ARDS, DIC, circulatory failure with reduced total oxygen utilization in spite of adequate oxygen supply, impaired defense mechanism are the pathogenetic features.
- Respiratory, renal, hepatic, circulatory, coagulative and cardiac failure occurs as an end stage MODS.
- Primary MODS is due to a well defined cause like pulmonary contusion, rhabdomyolysis, multiple transfusions.
- Secondary MODS occurs as a result of host response in SIRS.
- Management of MODS is critical care in ICU with ventilator support, haemodialysis, transfusions, antibiotics, proper nutrition in the form of TPN or enteral. MODS stage has got high mortality.

**OXYGEN THERAPY**

**Indications**

2. Gas gangrene with toxic haemolysis.
3. Coal gas poisoning.
4. Over morphinisation.
5. Pulmonary embolism and fat embolism.
7. Cardiogenic shock and acute bronchitis.
   - 27% oxygen is delivered through ventimask (disposable polythene mask) at a rate of 4–6 litres per minute. Oxygen is also given along with positive pressure ventilation.

**HYPERBARIC OXYGEN**

It is administration of oxygen 1 or 2 atmospheres above the atmospheric pressure in a compression chamber. It increases the arterial oxygen saturation so that oxygen perfusion of tissues will be increased.

**Indications**

1. Carbon monoxide poisoning.
2. Tetanus, gas gangrene infections.
4. Drenching in paralytic ileus to reduce the nitrogen gas in distended bowel.
5. As a radiosensitizer in the treatment of cancer.

**Complications**

- Cerebral gas embolism.
- Rupture of tympanic membrane.
- Visual defects.
- O₂ toxicity.
- CO₂ narcosis.
- Respiratory depression.

**Contraindications**

- Asthma/emphysema.
- High fever.
- Chronic sinusitis.
- Viral infection.
- Pregnancy.

**TOPICAL O₂ THERAPY**

H₂O₂ to release nascent oxygen in ulcers and abscess.

**CARDIAC ARREST**

*It is the cessation of the heart. Heart stops contracting.*

**Causes:** All causes for shock.

**Features of cardiac arrest**

- No palpable pulse
- Heart sounds not heard
- Cessation of respiration—cyanosis occurs
- Development of unconsciousness
- Pupils start dilating

**Critical Period**

Once heart and lungs stop, *brain death occurs in 3 minutes.*

**Immediate measures**

- Airway
- Breathing
- Cardiac compression
- Drugs and Defibrillator
- ECG, Endotracheal tube and Monitor

Hope puts a smile on our face when the heart cannot manage.
b. Another person at the same time should give mouth to mouth breathing at a rate of 20 to 30 per minute after clearing the airway by removing froth and dentures. A bag with mask can be used to ventilate using air or oxygen.

c. Endotracheal intubation and ventilator support.

d. Injection of 1:10,000 adrenaline and 10% calcium chloride intravenously.

e. Sodium bicarbonate 8.4% injection, hydrocortisone injection.

f. Defibrillator, if there is ventricular fibrillation.

g. Analysis of blood gas (PCO₂ and PO₂), and serum electrolytes assessment at repeated intervals.

h. Urinary catheterization, Ryle’s tube insertion.

i. Monitoring the patient with BP, pulse, respiration, and temperature chart.

Once patient recovers, the cause and sequelae has to be managed properly.

**Sequelae are due to hypoxia and circulatory collapse**

- Cerebral oedema and permanent brain damage
- ARDS (Adult respiratory distress syndrome)
- Renal failure

### Internal Open Cardiac Massage

This method is used when cardiac arrest occurs in the operation theatre during surgery, acute tamponade, and acute bilateral pneumothorax.

Left side thorax is opened through a lengthy incision along 4th or 5th intercostal space. Initially heart with intact pericardium is rhythmically compressed and relaxed using left hand against sternum. Meanwhile costal cartilages above and below are cut with a knife to have a better exposure. Pericardium is opened in front of the phrenic nerve. Direct cardiac massage is undertaken until heart regains its function and later shifted to ventilatory support and critical care.

**Fig. 1.213:** Left thoracotomy for open internal cardiac massage.

### Defibrillation Technique (Cardioversion)

Apply gelly to the site of electrodes. One electrode at the base of heart to the right of the sternum other over the estimated area of the apex of the heart. Ensure that nobody is in contact with the patient. Activate the defibrillator. Resume ventilation and ECG monitor immediately. After that, monitor continuously, correct the acidosis, catheterise and observe urine output. Arrange for ICU care.

**Fig. 1.214:** Defibrillator used in case of cardiac arrest.