

Shock

Types, recognition and therapy

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SHOCK SYNDROME

- **Shock** is a condition in which the cardiovascular system fails to perfuse tissues adequately
- An impaired cardiac pump, circulatory system, and/or volume can lead to compromised blood flow to tissues
- Inadequate tissue perfusion can result in:
 - generalized cellular hypoxia (starvation)
 - widespread impairment of cellular metabolism
 - tissue damage → organ failure
 - death

PATHOPHYSIOLOGY

Cells switch from aerobic to anaerobic metabolism

lactic acid production



Cell function ceases & swells



membrane becomes more permeable



electrolytes & fluids seep in & out of cell



Na⁺/K⁺ pump impaired



mitochondria damage

cell death

COMPENSATORY MECHANISMS: Sympathetic Nervous System (SNS)-Adrenal Response

- SNS - Neurohormonal response
Stimulated by baroreceptors

- ▣ Increased heart rate

- ▣ Increased contractility

- ▣ Vasoconstriction (SVR-Afterload)

- ▣ Increased Preload

COMPENSATORY MECHANISMS: Sympathetic Nervous System (SNS)-Adrenal Response

- SNS - Hormonal: Renin-angiotension system
 - ☐ Decrease renal perfusion
 - ☐ Releases renin → angiotension I
 - ☐ angiotension II → potent vasoconstriction &
 - ☐ releases aldosterone adrenal cortex
 - ☐ sodium & water retention →

COMPENSATORY MECHANISMS: Sympathetic Nervous System (SNS)-Adrenal Response

- SNS - Hormonal: Antidiuretic Hormone
 - ☐ Osmoreceptors in hypothalamus stimulated
 - ☐ ADH released by Posterior pituitary gland
 - ☐ Vasopressor effect to increase BP
 - ☐ Acts on renal tubules to retain water

COMPENSATORY MECHANISMS: Sympathetic Nervous System (SNS)-Adrenal Response

- SNS - Hormonal: Adrenal Cortex
 - ☐ Anterior pituitary releases adrenocorticotrophic hormone (ACTH)
 - ☐ Stimulates adrenal Cx to release glucocorticoids
 - ☐ Blood sugar increases to meet increased metabolic needs

Failure of Compensatory Response

- **Decreased blood flow to the tissues causes cellular hypoxia**
- **Anaerobic metabolism begins**
- **Cell swelling, mitochondrial disruption, and eventual cell death**
- **If Low Perfusion States persists:**

IRREVERSIBLE → DEATH IMMINENT!!

Stages of Shock

- **Initial stage** - tissues are under perfused, decreased CO, increased anaerobic metabolism, lactic acid is building
- ✿ **Compensatory stage** - Reversible. SNS activated by low CO, attempting to compensate for the decrease tissue perfusion.
- ✿ **Progressive stage** - Failing compensatory mechanisms: profound vasoconstriction from the SNS → ISCHEMIA
Lactic acid production is high → metabolic acidosis
- ✿ **Irreversible or refractory stage** - Cellular necrosis and Multiple Organ Dysfunction Syndrome may occur

DEATH IS IMMINENT!!!!

Pathophysiology Systemic Level

- **NET RESULTS OF CELLULAR SHOCK:**
 - systemic lactic acidosis
 - decreased myocardial contractility
 - decreased vascular tone
 - decrease blood pressure, preload, and cardiac output

Shock Syndromes

- **Hypovolemic Shock**
 - blood **VOLUME** problem
- **Cardiogenic Shock**
 - blood **PUMP** problem
- **Distributive Shock**
[septic;anaphylactic;neurogenic]
 - blood **VESSEL** problem

Hypovolemic Shock

- **Loss of circulating volume “Empty tank”** → decrease tissue perfusion → general shock response
- **ETIOLOGY:**
 - Internal or External fluid loss
 - Intracellular and extracellular compartments
- **Most common causes:**
 - Hemorrhage
 - Dehydration

Hypovolemic Shock: External loss of fluid

- **Fluid loss: Dehydration**
 - Nausea & vomiting, diarrhea, massive diuresis, extensive burns
- **Blood loss:**
 - trauma: blunt and penetrating
 - **BLOOD YOU SEE**
 - **BLOOD YOU DON'T SEE**

Hypovolemic Shock: Internal fluid loss

- **Loss of Intravascular integrity**
- **Increased capillary membrane permeability**
- **Decreased Colloidal Osmotic Pressure**
(third spacing)

Pathophysiology of Hypovolemic Shock

- **Decreased intravascular volume leads to....**
 - ✉ Decreased venous return (Preload, RAP) leads to...
 - ✉ Decreased ventricular filling (Preload, PAWP) leads to....
 - ✉ Decreased stroke volume (HR, Preload, & Afterload) leads to
 - ✉ Decreased CO leads to...(Compensatory mechanisms)
 - ✉ **Inadequate tissue perfusion!!!!**

Clinical Presentation Hypovolemic Shock

- Tachycardia and tachypnea
- Weak, thready pulses
- Hypotension
- Skin cool & clammy
- Mental status changes
- Decreased urine output: dark & concentrated

Assessment & Management

S/S vary depending on severity of fluid loss:

- **15%[750ml]-** compensatory mechanism maintains CO
- **15-30% [750-1500ml]-** Hypoxemia, decreased BP & UOP
- **30-40% [1500-2000ml] -**Impaired compensation & profound shock along with severe acidosis
- **40-50% -** refractory stage:
loss of volume= death

Classification of shock

<u>% blood volume lost</u>	pulse	BP	UO / cons. level	therapy
0 – 15% 0-750	+	120/80	Normal/ agitated	nil
15 – 30% 750-1500	+++	110/90	agitated	i.v. fluids
30-40% 1500- 2000	+++++	90/75	agitated	Fluids + blood
➤40% ➤ > 2000	+++++++	palpable	obtund	Surgery + blood

Therapy of hypovolaemic shock

- Airway / breathing / C/spine control
- Stop all obvious haemorrhage
- Insert I.v. lines, take blood for X-match
- Give rapid bolus of fluid, then assess response
- Decide on need for surgery vs. decision to investigate

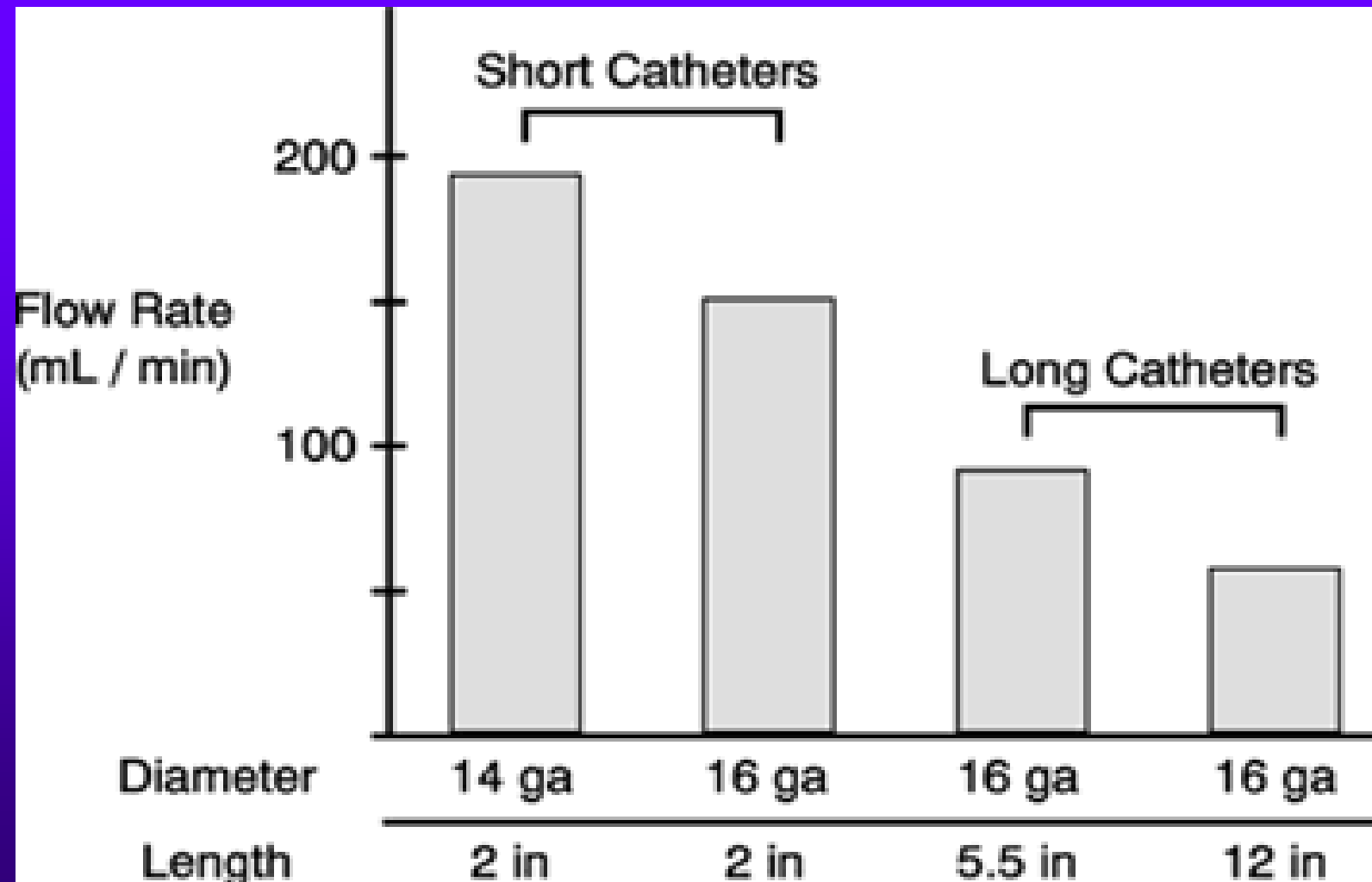
Colloid vs. crystalloid

- Large meta-analysis (BMJ, 1998) suggested that colloid associated with less pulmonary oedema, lower volumes but equal mortality
- Crystalloid produces smaller rise in BP, and more hypothermia
- NB gelatines and ACE inhibitors

Intravenous Access

- the rate of volume infusion is determined by the dimensions of the vascular catheter, not by the size of the vein
- cannulation of the large central veins requires catheters that are at least 5 inches in length, whereas cannulation of peripheral veins can be accomplished with catheters that are 2 inches in length

Intravenous Access



Intravenous Access

- central venous cannulation is reserved for monitoring cardiac filling pressures and venous O₂ saturation unless very-large-bore *introducer catheters* are used for volume resuscitation
- central venous catheters are 3 to 4 times longer than peripheral venous catheters, **the infusion rate through central catheters will be as much as 75% less than the infusion rate through peripheral catheters (of equal diameter)**

Resuscitation Endpoints

- The following are common endpoints of volume resuscitation:
 1. CVP = 15 mm Hg
 2. Wedge pressure = 10 to 12 mmHg
 3. Cardiac index > 3 L/min/m²
 4. Blood lactate < 4 mmol/L
 5. Base deficit -3 to +3 mmol/L

Fluid Composition

- Normal Saline
 - 154 meq sodium
 - 154 meq chloride
 - 308 mOsm
 - pH 5.6

Fluid Composition

- Lactated Ringer's
 - 130 meq sodium
 - 109 meq chloride
 - 4 meq potassium
 - 3 meq calcium
 - 28 meq lactate
 - 273 mOsm
 - pH 6.6
 - Can't use LR while infusing blood!

Cardiogenic shock

- Syndrome of inadequate tissue perfusion associated with normal circulating BV, and low cardiac output
- Symptoms: dyspnoea, poor exercise tolerance, confusion, sweating, PND
- Signs: tachycardia, cold skin, high JVP, added heart sounds, engorged liver, peripheral oedema

Cardiogenic Shock

- **Assess for:**
 - **Blunt trauma to the chest**
 - **Cardiac tamponade**
 - **Cardiac dysrhythmias**
 - **Myocardial infarction**
 - **Cardiac contusion**
 - **Tachycardia**
 - **Muffled heart sounds**
 - **Engorged neck veins with hypotension**
 - **Dyspnea**

Core Skills **Edema in feet and ankles** Treat for Shock

Cardiogenic Shock : Etiologies

- **Mechanical:**
complications of MI:
 - Papillary Muscle Rupture!!!!
 - Ventricular aneurysm
 - Ventricular septal rupture
- **Other causes:**
 - Cardiomyopathies
 - tamponade
 - tension pneumothorax
 - arrhythmias
 - valve disease

Cardiogenic Shock: Pathophysiology

- **Impaired pumping ability of LV leads to...**
 - ☐ Decreased stroke volume leads to.....
 - ☐ Decreased CO leads to
 - ☐ Decreased BP leads to.....
 - ☐ Compensatory mechanism which may lead to ...
 - ☐ **Decreased tissue perfusion !!!!**

Cardiogenic Shock: Pathophysiology

- **Impaired pumping ability of LV leads to...**
 - ➔ Inadequate systolic emptying leads to ...
 - ↑ Left ventricular filling pressures (preload) leads to...
 - ↑ Left atrial pressures leads to
 - ↑ Pulmonary capillary pressure leads to ...
 - ➔ **Pulmonary interstitial & intraalveolar edema**
!!!!

Clinical Presentation

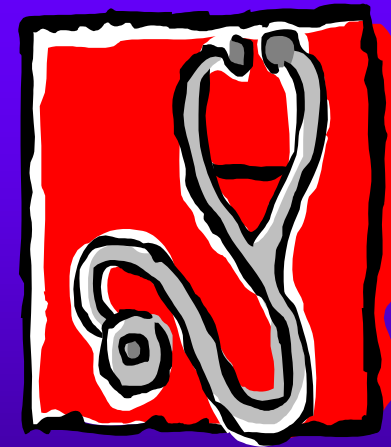
Cardiogenic Shock

- Similar catecholamine compensation changes in generalized shock & hypovolemic shock
- May not show typical tachycardic response if on Beta blockers, in heart block, or if bradycardic in response to nodal tissue ischemia
- Mean arterial pressure below 70 mmHg compromises coronary perfusion
 - $(MAP = SBP + (2) DBP/3)$

Cardiogenic Shock: Clinical Presentation

Abnormal heart sounds

- Murmurs
- Pathologic S3 (ventricular gallop)
- Pathologic S4 (atrial gallop)



Clinical Presentation

Cardiogenic Shock

- Pericardial tamponade
 - muffled heart tones, elevated neck veins
- Tension pneumothorax
 - JVD, tracheal deviation, decreased or absent unilateral breath sounds, and chest hyperresonance on affected side

COLLABORATIVE MANAGEMENT

- **Goal of management :**
- Treat Reversible Causes
- Protect ischemic myocardium
- Improve tissue perfusion
- **Treatment is aimed at :**
- Early assessment & treatment!!!
- Optimizing pump by:
 - Increasing myocardial O₂ delivery
 - Maximizing CO
 - Decreasing LV workload (Afterload)

COLLABORATIVE MANAGEMENT

Limiting/reducing myocardial damage during Myocardial Infarction:

- Increased pumping action & decrease workload of the heart
 - Inotropic agents
 - Vasoactive drugs
 - Intra-aortic balloon pump
 - Cautious administration of fluids
 - Transplantation
- Consider thrombolytics, angioplasty in specific cases

Management Cardiogenic Shock

OPTIMIZING PUMP FUNCTION:

- **Pulmonary artery monitoring is a necessity !!**
- Aggressive airway management: Mechanical Ventilation
- Judicious fluid management
- Vasoactive agents
 - **Dobutamine**
 - **Dopamine**

Management Cardiogenic Shock

OPTIMIZING PUMP FUNCTION (CONT.):

- Morphine as needed (Decreases preload, anxiety)
- Cautious use of diuretics in CHF
- Vasodilators as needed for afterload reduction
- Short acting beta blocker, esmolol, for refractory tachycardia

Hemodynamic Goals of Cardiogenic Shock

Optimized Cardiac function involves cautious use of combined fluids, diuretics, inotropes, vasopressors, and vasodilators to :

- Maintain adequate filling pressures (LVEDP 14 to 18 mmHg)
- Decrease Afterload (SVR 800-1400)
- Increase contractility
- Optimize CO/CI

Therapy of low SV

- Drugs to move point back down curve: e.g.. GTN, diuretics, head-up posture
- Drugs to improve contractility: I.v. inotropes, amrinone, ACE inhibitors
- Drugs to prevent fluid retention: ACE inhibitors, diuretics
- Other methods: IABP, LVAD, heart transplant

Dopamine

- Dopamine receptor activation at low doses-
”splanchnic dilation” (2-5 mcg/kg/min)
- Beta receptor activation-increase cardiac
output (5-10 mcg/kg/min)
- Alpha receptor activation-vasoconstriction
(>10 mcg/kg/min)

Dobutamine

- primarily a β_1 -receptor agonist (cardiac stimulation), but it also has mild β -2 effects (vasodilation)
- causes a dose-dependent increase in stroke volume
- decrease in cardiac filling pressures
- an alkaline pH inactivates catecholamines such as dobutamine
- Dose 2-20 mcg/kg/min

Dobutamine

- dobutamine is the preferred inotropic agent for the acute management of low output states due to systolic heart failure. Because dobutamine does not usually raise the arterial blood pressure, it is **not indicated as monotherapy in patients with cardiogenic shock**

Norepinephrine

- α -receptor agonist that promotes widespread vasoconstriction
- administration of any vasoconstrictor agent carries a risk of hypoperfusion and ischemia involving any tissue bed or vital organ
- Dose 8-12 mcg/min

Distributive Shock

- Inadequate perfusion of tissues through maldistribution of blood flow
- Intravascular volume is maldistributed because of alterations in blood vessels
- Cardiac pump & blood volume are normal but blood is not reaching the tissues

Vasogenic/Distributive Shock

- **ETIOLOGIES**

- **Septic Shock (Most Common)**
- **Anaphylactic Shock**
- **Neurogenic Shock**

Anaphylactic Shock

- A type of distributive shock that results from widespread systemic allergic reaction to an antigen
- This hypersensitive reaction is **LIFE THREATENING**

Pathophysiology Anaphylactic Shock

- Antigen exposure
- body stimulated to produce IgE antibodies specific to antigen
 - **drugs, bites, contrast, blood, foods, vaccines**
- Reexposure to antigen
 - **IgE binds to mast cells and basophils**
- Anaphylactic response

Anaphylactic Response

- Vasodilatation
- Increased vascular permeability
- Bronchoconstriction
- Increased mucus production
- Increased inflammatory mediators
recruitment to sites of antigen interaction

Clinical Presentation

Anaphylactic Shock

- Almost immediate response to inciting antigen
- Cutaneous manifestations
 - urticaria, erythema, pruritis, angioedema
- Respiratory compromise
 - stridor, wheezing, bronchorrhea, resp. distress
- Circulatory collapse
 - tachycardia, vasodilation, hypotension

Management Anaphylactic Shock

- **Early Recognition, treat aggressively**
- **AIRWAY SUPPORT**
- **IV EPINEPHRINE (open airways)**
- **Antihistamines, diphenhydramine 50 mg IV**
- **Corticosteroids**
- **IMMEDIATE WITHDRAWAL OF ANTIGEN IF POSSIBLE**
- **PREVENTION**

Management Anaphylactic Shock

- **Judicious crystalloid administration**
- **Vasopressors to maintain organ perfusion**
- **Positive inotropes**
- **Patient education**

NEUROGENIC SHOCK

- A type of distributive shock that results from the loss or suppression of sympathetic tone
- Causes massive vasodilatation in the venous vasculature, ↓ venous return to heart, ↓ cardiac output.
- Most common etiology: Spinal cord injury above T6
- Neurogenic is the rarest form of shock!

Pathophysiology of Neurogenic Shock

Distruption of sympathetic nervous system

Loss of sympathetic tone

Venous and arterial vasodilation

Decreased venous return

Decreased stroke volume

Decreased cardiac output

Decreased cellular oxygen supply

Impaired tissue perfusion

Impaired cellular metabolism

Assessment, Diagnosis and Management of Neurogenic Shock

PATIENT ASSESSMENT

- Hypotension
- Bradycardia
- Hypothermia
- Warm, dry skin
- RAP ↓
- PAWP ↓
- CO ↓
- Flaccid paralysis below level of the spinal lesion

MEDICAL MANAGEMENT

- Goals of Therapy are to treat or remove the cause & prevent cardiovascular instability, & promote optimal tissue perfusion

MANAGEMENT OF NEUROGENIC SHOCK

**Hypovolemia- tx with careful fluid replacement for
BP<90mmHg, UO<30cc/hr**

Changes in LOC

Observe closely for fluid overload

Vasopressors may be needed

Hypothermia- warming txs

-avoid large swings in pts body temperature

Treat Hypoxia

Maintain ventilatory support

MANAGEMENT OF NEUROGENIC SHOCK

- **OBSERVE FOR BRADYCARDIA-MAJOR DYSRHYTHMIA**
- Observe for DVT- venous pooling in extremities make patients high-risk>>P.E.
- Use prevention modalities [TEDS, ROM, Sequential stockings, anticoagulation]

NURSING DIAGNOSIS

- Fluid Volume Deficit r/t relative loss
- Decreased CO r/t sympathetic blockade
- Anxiety r/t biologic, psychologic or social integrity

Management Neurogenic Shock

- Alpha agonist to augment tone if perfusion still inadequate
 - **dopamine at alpha doses (> 10 mcg/kg per min)**
 - **ephedrine (12.5-25 mg IV every 3-4 hour)**
- Treat bradycardia with atropine 0.5-1 mg doses to maximum 3 mg
 - **may need transcutaneous or transvenous pacing temporarily**

Septic shock

- Syndrome of profound hypotension due to release of endotoxins / TNF / vasoactive peptides following bacterial destruction
- Usually associated with normal blood volume, high / low CO, and low SVR
- Re-distribution of blood to splanchnic vessels, with resultant poor skin perfusion

Pathophysiology of Septic shock

- Initiated by **gram-negative (most common) or gram positive bacteria**, fungi, or viruses
 - ➔ Cell walls of organisms contain Endotoxins
 - ➔ Endotoxins release inflammatory mediators (systemic inflammatory response) causes.....
 - ➔ Vasodilation & increase capillary permeability leads to
 - ➔ Shock due to alteration in peripheral circulation & massive dilation

Clinical Presentation Septic Shock

- **TWO PHASES:**

- “Warm” shock - early phase

- hyperdynamic response,
VASODILATION

- “Cold” shock - late phase

- hypodynamic response
- **DECOMPENSATED STATE**

Clinical Manifestations

- **EARLY---HYPERDYNAMIC STATE---COMPENSATION**

- **Massive vasodilation**
- **Pink, warm, flushed skin**
- **Increased Heart Rate**
Full bounding pulse
- **Tachypnea**
- **Decreased SVR**
- **Increased CO & CI**
- **SVO₂ will be abnormally high**
- **Crackles**

Clinical Manifestations

- **LATE--HYPODYNAMIC STATE--DECOMPENSATION**

- Vasoconstriction
- Skin is pale & cool
- Significant tachycardia
- Decreased BP
- Increase SVR
- Decreased CO
- Decreased UOP
- Metabolic & respiratory acidosis with hypoxemia

COLLABORATIVE MANAGEMENT

- **Prevention !!!**
- **Find and kill the source of the infection**
- Fluid Resuscitation
- Vasoconstrictors
- Inotropic drugs
- Maximize O2 delivery Support
- Nutritional Support
- Comfort & Emotional support

Initial management of septic shock

- Administer pure oxygen
- Start I.v. line, and take bloods for culture
- Give 20ml/kg boluses of colloid
- Observe rise in BP, CVP line if possible
- If $> 60\text{ml/kg}$ (4200mL) consider ICU referral
- Broad spectrum antibiotics urgently

ICU care of septic shock

- Adequate oxygenation and ventilation
- CVP and PA line
- Broad spectrum antibiotics
- Drive oxygen delivery towards $600\text{ml}/\text{min}/\text{m}^2$
- Attempt to identify source of sepsis