休克之處理

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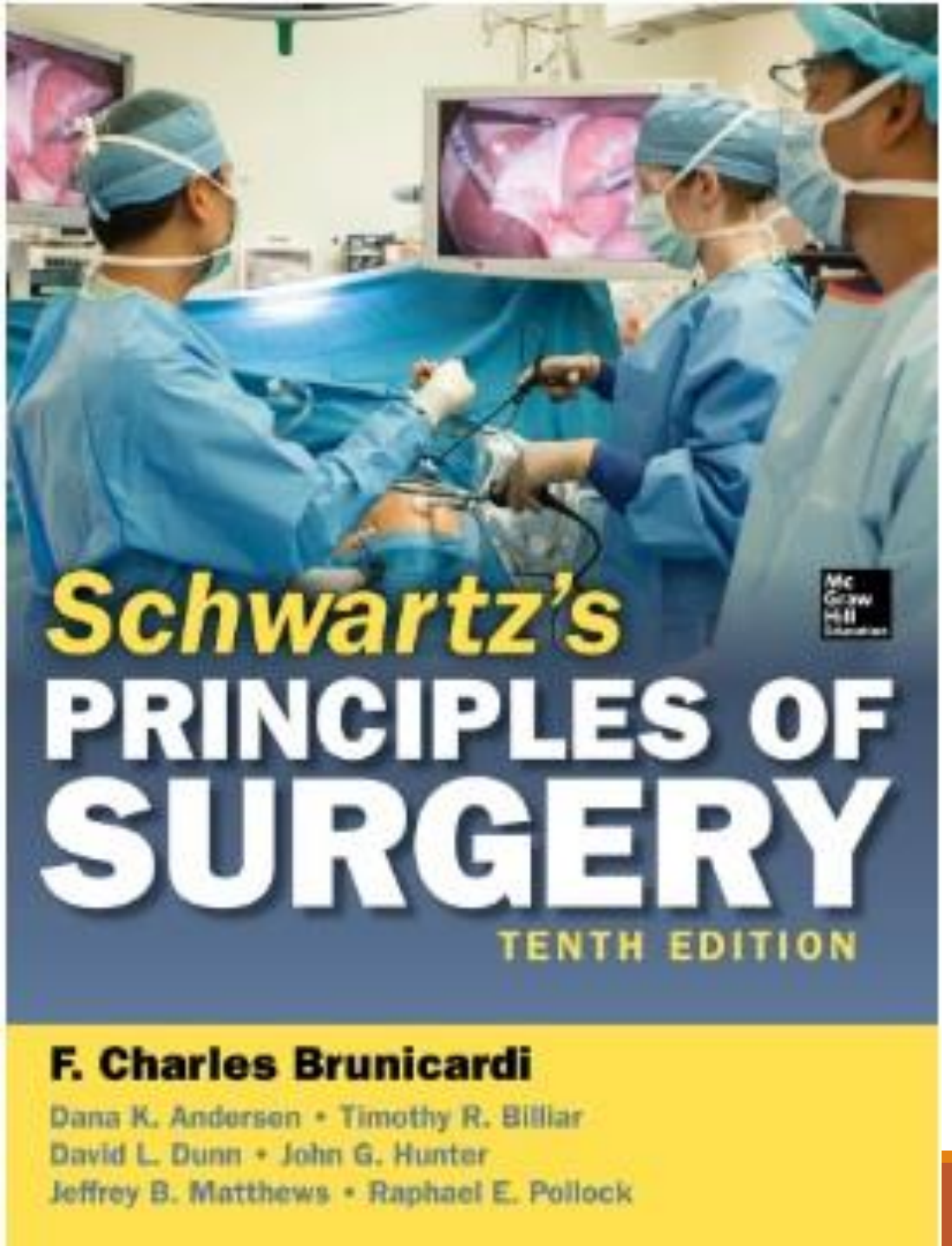
參考書目 ：



FORMS OF SHOCK

Hypovolemic/Hemorrhagic

The most common cause of shock in the surgical or trauma patient is loss of circulating volume from hemorrhage.

Shock in a trauma patient or postoperative patient should be presumed to be due to hemorrhage until proven otherwise

Diagnosis

A secure airway must be confirmed or established and volume infusion initiated while the search for the cause of the hypotension is pursued.

The clinical signs of shock may be evidenced by agitation, cool clammy extremities, tachycardia, weak or absent peripheral pulses, and hypotension. Such apparent clinical shock results from at least 25% to 30% loss of the blood volume.

Classification of hemorrhage

	CLASS			
PARAMETER	**I**	**II**	**III**	**IV**
Blood loss (mL)	<750	750–1500	1500–2000	>2000
Blood loss (%)	<15	15–30	30–40	>40
Heart rate (bpm)	<100	>100	>120	>140
Blood pressure	Normal	Orthostatic	Hypotension	Severe hypotension
CNS symptoms	Normal	Anxious	Confused	Obtunded

bpm = beats per minute; CNS = central nervous system.

Serum lactate and base deficit are measurements that are helpful to both estimate and monitor the extent of bleeding and shock.

The amount of lactate that is produced by anaerobic respiration is an indirect marker of tissue hypoperfusion, cellular O_2 debt, and the severity of hemorrhagic shock.

Base deficit values derived from arterial blood gas analysis provide clinicians with an indirect estimation of tissue acidosis from hypoperfusion.

Treatment

Control of ongoing hemorrhage - diagnostic evaluation to identify a source

Fail to respond to initial resuscitative efforts should be assumed to have ongoing active hemorrhage from large vessels and require prompt operative intervention.

The appropriate priorities in these patients are :

(a) secure the airway,

(b) control the source of blood loss, and

(c) intravenous (IV) volume resuscitation.

Damage control resuscitation

Initial resuscitation is limited to keep SBP around 80 to 90 mmHg. This prevents renewed bleeding from recently clotted vessels.

Resuscitation and intravascular volume resuscitation are accomplished with blood products and limited crystalloids.

Fluid resuscitation is a major adjunct to physically controlling hemorrhage in patients with shock.

Crystalloids continue to be the mainstay of fluid choice.

In patients with severe hemorrhage, restoration of intravascular volume should be achieved with blood products.

Hypertonic saline solutions :

Resulting in decreased reperfusion-mediated injury with decreased O_2 radical formation, less impairment of immune function compared to standard crystalloid solution, and less brain swelling in the multi-injured patient.

May contribute to a decrease in the incidence of ARDS and multiple organ failure

Transfusion of packed red blood cells and other blood products is essential in the treatment of patients in hemorrhagic shock.

Current recommendations in stable ICU patients aim for a target hemoglobin of 7 to 9 g/dL.

Additional resuscitative adjuncts in patients with hemorrhagic shock include minimization of heat loss and maintaining normothermia - associated with acidosis, hypotension, and coagulopathy.

Septic Shock (Vasodilatory Shock)

In the peripheral circulation, profound vasoconstriction is the typical physiologic response to the decreased arterial pressure and tissue perfusion with hemorrhage, hypovolemia, or acute heart failure.

Vasodilatory shock is the result of dysfunction of the endothelium and vasculature secondary to circulating inflammatory mediators and cells or as a response to prolonged and severe hypoperfusion.

The most frequently encountered form of vasodilatory shock is septic shock.

Causes of septic and vasodilatory shock

- Systemic response to infection
- Noninfectious systemic inflammation
  - Pancreatitis
  - Burns
- Anaphylaxis
- Acute adrenal insufficiency
- Prolonged, severe hypotension
  - Hemorrhagic shock
  - Cardiogenic shock
  - Cardiopulmonary bypass
- Metabolic
  - Hypoxic lactic acidosis
  - Carbon monoxide poisoning

Patients with sepsis have evidence of an infection, as well as systemic signs of inflammation (e.g., fever, leukocytosis, and tachycardia).

Hypoperfusion with signs of organ dysfunction is termed severe sepsis.

Septic shock requires the presence of the above, associated with more significant evidence of tissue hypoperfusion and systemic hypotension.

Signs of hypoperfusion such as confusion, malaise, oliguria, or hypotension may be present.

These should prompt an aggressive search for infection, including a thorough physical examination, inspection of all wounds, evaluation of intravascular catheters or other foreign bodies, obtaining appropriate cultures, and adjunctive imaging studies, as needed.

Treatment

Begins with an assessment of the adequacy of their airway and ventilation - intubation and ventilation to prevent respiratory collapse.

Because vasodilation and decrease in total peripheral resistance may produce hypotension, fluid resuscitation and restoration of circulatory volume with balanced salt solutions is essential.

This resuscitation should be at least 30 m*L*/kg within the first 4 *to* 6 hours.

Empiric antibiotics must be chosen carefully based on the most likely pathogens (gram-negative rods, gram-positive cocci, and anaerobes).

IV antibiotics will be insufficient to adequately treat the infectious episode in the settings of infected fluid collections, infected foreign bodies, and devitalized tissue.

These situations require source control and involve percutaneous drainage and operative management to target a focus of infection.

After first-line therapy of the septic patient with antibiotics, IV fluids, and intubation if necessary, vasopressors may be necessary to treat patients with septic shock.

Catecholamines are the vasopressors used most often, with norepinephrine being the first-line agent followed by epinephrine.

Occasionally, patients with septic shock will develop arterial resistance to catecholamines. Arginine vasopressin, a potent vasoconstrictor, is often efficacious in this setting and is often added to norepinephrine.

The majority of septic patients have hyperdynamic physiology with supranormal cardiac output and low systemic vascular resistance.

Dobutamine therapy is recommended for patients with cardiac dysfunction as evidenced by high filling pressures and low cardiac output or clinical signs of hypoperfusion after achievement of restoration of blood pressure following fluid resuscitation.

Rivers and colleagues reported that goal-directed therapy of septic shock and severe sepsis initiated in the emergency department and continued for 6 hours significantly improved outcome.

Surviving Sepsis Campaign Bundles

To be Completed Within 3 Hours:

1) Measure lactate level
2) Obtain blood cultures prior to administration of antibiotics
3) Administer broad spectrum antibiotics
4) Administer 30 mL/kg crystalloid for hypotension or lactate ≥ 4 mmol/L

To be Completed Within 6 Hours:

5) Apply vasopressors (for hypotension that does not respond to initial fluid resuscitation) to maintain a mean arterial pressure (MAP) ≥ 65 mm Hg
6) In the event of persistent arterial hypotension despite volume resuscitation (septic shock) or initial lactate ≥ 4 mmol/L (36 mg/dL):
    - Measure central venous pressure (CVP)*
    - Measure central venous oxygen saturation ($Scvo_2$)*
7) Remeasure lactate if initial lactate was elevated*

*Targets for quantitative resuscitation included in the guidelines are CVP of ≥ 8 mm Hg, $Scvo_2$ of ≥ 70%, and normalization of lactate.

Cardiogenic Shock

Circulatory pump failure leading to diminished forward flow and subsequent tissue hypoxia, in the setting of adequate intravascular volume.

Hemodynamic criteria include sustained hypotension (i.e., SBP <90 mmHg for at least 30 minutes), reduced cardiac index (<2.2 L/min per square meter), and elevated pulmonary artery wedge pressure (>15 mmHg).

Acute, extensive MI is the most common cause of cardiogenic shock.

Seventy-five percent of patients who have cardiogenic shock complicating acute MIs develop signs of cardiogenic shock within 24 hours after onset of infarction (average 7 hours).

In evaluation of possible cardiogenic shock, other causes of hypotension must be excluded, including hemorrhage, sepsis, pulmonary embolism, and aortic dissection.

Signs of circulatory shock include hypotension, cool and mottled skin, depressed mental status, tachycardia, and diminished pulses.

Confirmation of a cardiac source for the shock requires electrocardiogram and urgent echocardiography.

Other useful diagnostic tests include chest radiograph, arterial blood gases, electrolytes, complete blood count, and cardiac enzymes.

Treatment

After ensuring that an adequate airway is present and ventilation is sufficient, attention should be focused on support of the circulation.

Rapidly excluding hypovolemia and establishing the presence of cardiac dysfunction are essential.

Treatment of cardiac dysfunction includes maintenance of adequate oxygenation to ensure adequate myocardial O_2 delivery and judicious fluid administration to avoid fluid overload and development of cardiogenic pulmonary edema.

Electrolyte abnormalities, commonly hypokalemia and hypomagnesemia, should be corrected.

Pain is treated with IV morphine sulfate or fentanyl.

Significant dysrhythmias and heart block must be treated with antiarrhythmic drugs, pacing, or cardioversion, if necessary.

Early consultation with cardiology is essential in current management of cardiogenic shock, particularly in the setting of acute MI.

When profound cardiac dysfunction exists, inotropic support may be indicated to improve cardiac contractility and cardiac output.

Dobutamine primarily stimulates cardiac $\beta_1$receptors to increase cardiac output but may also vasodilate peripheral vascular beds, lower total peripheral resistance, and lower systemic blood pressure through effects on $\beta_2$receptors.

Dopamine stimulates receptors (vasoconstriction), β_1 receptors (cardiac stimulation), and β_2 receptors (vasodilation), with its effects on β receptors predominating at lower doses.

Dopamine may be preferable to dobutamine in treatment of cardiac dysfunction in hypotensive patients.

Serial assessment of tissue perfusion using indices such as capillary refill, character of peripheral pulses, adequacy of urine output, or improvement in laboratory parameters of resuscitation such as pH, base deficit, and lactate.

Invasive monitoring generally is necessary in these unstable patients.

Patients whose cardiac dysfunction is refractory to cardiotonics may require mechanical circulatory support with an intra-aortic balloon pump.

Intra-aortic balloon pumping increases cardiac output and improves coronary blood flow by reduction of systolic afterload and augmentation of diastolic perfusion pressure.

Anticoagulation and aspirin are given for acute MI.

Although thrombolytic therapy reduces mortality in patients with acute MI, its role in cardiogenic shock is less clear.

Additional pharmacologic tools may include the use of β-blockers to control heart rate and myocardial O_2consumption.

Nitrates to promote coronary blood flow through vasodilation.

ACE inhibitors to reduce ACE-mediated vasoconstrictive effects that increase myocardial workload and myocardial O_2 consumption.

Obstructive Shock

Result in mechanical obstruction of venous return.

In trauma patients, this is most commonly due to the presence of tension pneumothorax.

Causes of obstructive shock

- Pericardial tamponade
- Pulmonary embolus
- Tension pneumothorax
- IVC obstruction
  - Deep venous thrombosis
  - Gravid uterus on IVC
  - Neoplasm
- Increased intrathoracic pressure
  - Excess positive end-expiratory pressure
  - Neoplasm

IVC = inferior vena cava.

Diagnosis and Treatment

The diagnosis of tension pneumothorax should be made on clinical examination.

The classic findings include respiratory distress (in an awake patient), hypotension, diminished breath sounds over one hemithorax, hyperresonance to percussion, jugular venous distention, and shift of mediastinal structures to the unaffected side with tracheal deviation.

Empiric treatment with pleural decompression is indicated rather than delaying to wait for radiographic confirmation.

When a chest tube cannot be immediately inserted, such as in the prehospital setting, the pleural space can be decompressed with a large-caliber needle.

Cardiac tamponade results from the accumulation of blood within the pericardial sac, usually from penetrating trauma or chronic medical conditions such as heart failure or uremia.

Patients who present with circulatory arrest from cardiac tamponade require emergency pericardial decompression, usually through a left thoracotomy

Diagnostic pericardial window represents the most direct method to determine the presence of blood within the pericardium.

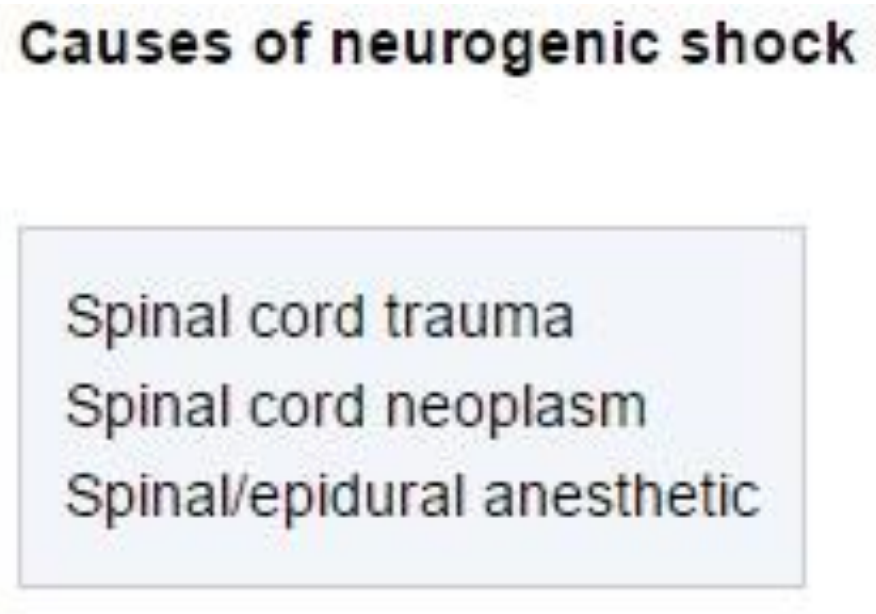
The procedure is best performed in the operating room under general anesthesia.

Neurogenic Shock

Diminished tissue perfusion as a result of loss of vasomotor tone to peripheral arterial beds.

Loss of vasoconstrictor impulses results in increased vascular capacitance, decreased venous return, and decreased cardiac output.

Usually secondary to spinal cord injuries from vertebral body fractures of the cervical or high thoracic region that disrupt sympathetic regulation of peripheral vascular tone.



Sympathetic input to the heart, which normally increases heart rate and cardiac contractility, and input to the adrenal medulla, which increases catecholamine release, may also be disrupted, preventing the typical reflex tachycardia that occurs with hypovolemia.

Management of acute spinal cord injury with attention to blood pressure control, oxygenation, and hemodynamics, essentially optimizing perfusion of an already ischemic spinal cord, seems to result in improved neurologic outcome.

Patients with hypotension from spinal cord injury are best monitored in an ICU and carefully followed for evidence of cardiac or respiratory dysfunction.

Acute spinal cord injury may result in bradycardia, hypotension, cardiac dysrhythmias, reduced cardiac output, and decreased peripheral vascular resistance.

Treatment

After the airway is secured and ventilation is adequate, fluid resuscitation and restoration of intravascular volume often will improve perfusion in neurogenic shock.

Most patients with neurogenic shock will respond to restoration of intravascular volume alone, with satisfactory improvement in perfusion and resolution of hypotension.

Administration of vasoconstrictors will improve peripheral vascular tone, decrease vascular capacitance, and increase venous return.

But should only be considered once hypovolemia is excluded as the cause of the hypotension and the diagnosis of neurogenic shock established.

If the patient's blood pressure has not responded to what is felt to be adequate volume resuscitation, dopamine may be used first.

Appropriate rapid restoration of blood pressure and circulatory perfusion may improve perfusion to the spinal cord, prevent progressive spinal cord ischemia, and minimize secondary cord injury.

ENDPOINTS IN RESUSCITATION

Resuscitation is complete when O_2 debt is repaid, tissue acidosis is corrected, and aerobic metabolism is restored.

Clinical confirmation of this endpoint remains a challenge.

Endpoints in resuscitation can be divided into *systemic* or *global par ameters, tissue-specific parameters,* and *cellular parameters*.

Endpoints in resuscitation

- Systemic/global
  - Lactate
  - Base deficit
  - Cardiac output
  - Oxygen delivery and consumption
- Tissue specific
  - Gastric tonometry
  - Tissue pH, oxygen, carbon dioxide levels
  - Near infrared spectroscopy
- Cellular
  - Membrane potential
  - Adenosine triphosphate

Lactate and base deficit indicate global tissue acidosis.

Gastric tonometry has been used to assess perfusion of the GI tract.

Gastric intramucosal pH (pHi) is calculated by applying the Henderson-Hasselbalch equation. pHi should be greater than 7.3; pHi will be lower in the setting of decreased O_2 delivery to the tissues

Near infrared (NIR) spectroscopy can measure tissue oxygenation and redox state of cytochrome a,a_3 on a continuous, noninvasive basis.

Tissue probes with optical sensors have been used to measure tissue pH and partial pressure of O_2 and CO_2 in subcutaneous sites, muscle, and the bladder.

外科專師考古題

1. 下列何者是「過敏性休克」的首選升壓或強心藥物？

(A) epinephrine (B) dopamine (C) vasopressin (D) norepinephrine

治療方面的原則是儘早送醫，使用腎上腺素（epinephrine），可避免致命的悲劇。

在大腿肌肉注射腎上腺素比手臂肌肉注射的吸收效果來得快，目前國外已有腎上腺素注射筆可供病患在緊急時自行使用，以減少延遲治療的遺憾。

除此之外，抗組織胺、氧氣及其他支持性療法可改善病況，而類固醇則可減少晚期的反應。

由於高達五分之一的病患會在發作八至十二小時後再次發作，且無法由第一次發作的嚴重度來預測，因此在病患發作後須觀察至少二十四小時，才可返家。

2. 有關dopamine 投予劑量與作用的敘述，下列何者正確？

(A) < 5 mcg/kg/min 時，作用於peripheral α-receptors，使血壓上升

(B) 5-10 mcg/kg/min 時，作用於 cardiac β1-receptors，使心收縮力上升

(C) 6-14 mcg/kg/min 時，為vasodilator，可增加腎血流量

(D) > 15 mcg/kg/min 時，作用於 cardiac β2-receptors，使心輸出量上升

At low rates of infusion (0.5-2 mcg/kg/min) dopamine causes vasodilation that is presumed to be due to a specific agonist action on dopamine receptors (distinct from alpha- and beta- adrenoceptors) in the renal, mesenteric, coronary, and intracerebral vascular beds.

At intermediate rates of infusion (2-10 mcg/kg/min) dopamine acts to stimulate the beta1-adrenoceptors, resulting in improved myocardial contractility, increased SA rate and enhanced impulse conduction in the heart. There is little, if any, stimulation of the b2-adrenoceptors (peripheral vasodilation).

At higher rates of infusion (10-20 mcg/kg/min) there is some effect on alpha-adrenoceptors, with consequent vasoconstrictor effects and a rise in blood pressure. The vasoconstrictor effects are first seen in the skeletal muscle vascular beds, but with increasing doses, they are also evident in the renal and mesenteric vessels.

At very high rates of infusion (above 20 mcg/kg/min), stimulation of alpha-adrenoceptors predominates and vasoconstriction may compromise the circulation of the limbs and override the dopaminergic effects of dopamine, reversing renal dilation and naturesis.

3.關於嚴重敗血症的early goal-directed therapy (EGDT)，下列敘述何者正確？

(A) 若液體復甦無反應，vasopressin 為首選藥

(B) 在六小時內開始執行EGDT 能降低死亡率

(C) 膠質溶液優於晶質溶液

(D) 應維持ScvO2 > 60%、MBP > 65 mmHg

4.下列何者為敗血性休克的初期血流動力學變化？

(A) 肺微血管楔壓↑、心輸出量↓、全身血管阻力↓

(B) 肺微血管楔壓↓、心輸出量↑、全身血管阻力↓

(C) 肺微血管楔壓↓、心輸出量↓、全身血管阻力↑

(D) 肺微血管楔壓↓、心輸出量↓、全身血管阻力↓

5. 敗血性休克(septic shock) 為下列哪一類休克？

(A) cardiogenic shock

(B) distributive shock

(C) obstructive shock

(D) hypovolemic shock

病人術後覺得口乾、少尿，並有心跳加速(120次/分)、血壓降低(80 / 60mmHg)之現象，應該補充何種輸液最為適合？

(A) 0.9%食鹽水　(B) 0.45%食鹽水

(C) 5%葡萄糖水　(D) 台大五號(TAITA No. 5)

根據 2016 年敗血性休克治療指引，除非有心肌缺氧或出血，敗血性休克病人建議血色素至少維持在多少(g/dL) 以上？

(A) 7　　(B) 9　　(C) 10　　(D) 12

根據 2016 年敗血性休克治療指引，除儘量避免低血糖發生外，建議敗血性休克病人血糖值不要高於多少(mg/dL) ？

(A) 110　(B) 140　(C) 180　(D) 250

有關心因性休克 (cardiogenic shock) 的描述，下列何者最不可能？

(A) 收縮壓(SBP)<90mmHg

(B) 心臟指數(cardiac index ,CI)>4L/min /m2

(C) 皮膚出現蛇紋(mottled skin)

(D) 脈搏壓(pulse pressure) 變窄

依據早期目標導向治療(early goal-directed therapy)，下列敘述何者不適合用來處置敗血症？

(A) 維持平均動脈壓(MAP) ≥ 65mmHg

(B) 儘快放置肺動脈導管

(C) 維持排尿量>0.5mL/Kg/ hr

(D) 維持中心靜脈血氧飽和濃度(ScvO2) ≥70%

敗血症治療指引2016

A.一開始的緊急處置

1. **不再建議要在6小時內積極給予輸液以達到特定目標(即所謂的early-goal-directed therapy)**，改建議在最初3小時內至少給予每公斤30mL的晶體溶液。

2. 建議頻繁的檢視並評估病人的血行動力學狀況，以決定後續的輸液要給多少。評估時應將病人的各種生理指標，包括心跳、血壓、動脈血氧飽和度、呼吸速率、體溫、尿量等等一起做整體性的評估。

3. 如需使用升壓劑，建議的治療目標是將**平均動脈壓維持在65mmHg**以上。

4. 當病人有乳酸升高的情形時，可以將”讓乳酸下降到正常值”當作輸液治療的目標。

B.及早發現有嚴重敗血症之病人:

每個醫院應該訂定一套方法來早期發現有嚴重敗血症之病人。

C. Diagnosis:

建議在給予抗生素前就完成細菌培養

D. Antimicrobial therapy

1. 盡量在1小時內就把IV注射的抗生素打入病人體內
2. 一開始選用廣效抗生素，之後要每天評估病人狀況，及早降階，一旦有培養的結果，也應及早降階
3. 對於已經敗血性休克之病人，一開始可以針對最有可能的致病菌，考慮併用抗生素。
4. 不建議使用在沒有敗血性休克之病人常規給予併用抗生素，也不再建議針對Neutropenia的病人一律常規給予併用抗生素。

D. Antimicrobial therapy

5. 抗生素療程一般為7-10天，下列狀況須延長療程: i. 臨床狀況進步緩慢；ii. 有無法引流之感染源；iii. 金黃葡萄球菌(Staphylococcus aureus)之菌血症；iv. Fungal or viral infection；v. 病人有Immunologic deficiency
6. 對於臨床上對引流治療反應很好的腹內感染以尿路感染，縮短療程是適當的。
7. 建議可以依據procalcitonin (PCT) level來及早停用抗生素

E. Source control (感染源的控制)
1. 應盡早確定感染所在位置，確定後及早予以引流或清創
2. 當中心靜脈導管(CVC)或其他放置於血管內之導管為可能之感染源時，應在放置好新的導管後立刻拔除舊的導管

F. 輸液治療
1. 一開始給予輸液時，**首選是晶體溶液(crystalloids)**
2. 建議不要使用hydroxyethyl starches
3. 當已經給予大量crystalloids時，可使用白蛋白
4. 晶體溶液(crystalloids)與gelatin相比，比較建議使用crystalloids

G. 昇壓劑

1. 目標是MAP≧65 mmHg
2. **首選藥物是norepinephrine (Levophed)**
3. 當已經用了norepinephrine (Levophed)，可是效果不理想時，可再加上epinephrine (Bosmin)或vasopressin (up to 0.03units/min)
4. 只有在心跳慢的病人，才考慮用dopamine取代norepinephrine，也不建議使用低劑量dopamine來保護腎臟功能
5. 當已給予充足的輸液治療以及昇壓劑，但是組織灌流不足之表現仍持續時，可嘗試給予dobutamine
6. 所有使用升壓劑之病人應該要使用動脈導管來監測血壓

H. 類固醇

1. 如果輸液治療及昇壓劑可以改善血循，不用給予類固醇；如果不行，建議給hydrocortisone 200mg/day

I. 輸血

1. 除非有心肌缺氧，acute hemorrhage，或嚴重低血氧，不然Hb維持在**7.0g/dL**即可，Hb小於7.0 g/dL才需考慮給予PRBC
2. 不建議使用erythropoietin (EPO)來治療敗血症病人的貧血
3. 除非有持續出血或者要進行侵入性治療，否則不建議預防性的以FFP來矯正凝血功能的異常
4. 沒有出血危險時，血小板小於10,000才考慮輸血小板；有出血之危險時，血小板小於20,000時考慮輸血小板；如有active bleeding或要進行手術/侵入性處置，則輸到血小板大於50,000

M. Mechanical ventilation in sepsis-induced ARDS
1. Tidal volume: 6mL/kg predicted body weight
2. Plateau pressure 小於30 cmH2O
3. 建議使用高一點的PEEP
4. 當**PaO2/FiO2小於150**時 (2012年是100)，考慮趴睡 (prone positioning)
5. 不建議使用高頻振盪呼吸器來治療sepsis-induced ARDS
6. 建議使用recruitment maneuver來治療sepsis-induced ARDS
7. 當PaO2/FiO2<150 p="">8. 治療sepsis-induced ARDS時，若病人已經沒有tissue hypoperfusion時，IV fluid不要給太多
9. 不建議使用beta2-agonist (如Bricanyl)治療ARDS
10. 不建議常規使用動脈導管
11. 所有使用呼吸器的敗血症病人，一律建議床頭提高至30-45度以預防呼吸器相關肺炎(VAP)
12. 當病人已準備好可以weaning時，建議要進行spontaneous breathing trial
13. 建議使用weaning protocol

N. 鎮靜、止痛
1. 盡量減少鎮靜劑之使用，並訂定使用之目標

O. 血糖控制
1. 建議訂定protocol來控制血糖，當血糖值連續兩次超過**180mg/dL**時開始使用，控制目標仍建議定為180mg/dL，而非110mg/dL
2. 使用RI infusion時，先q1-2h檢測血糖值，穩定後再改為q4h測血糖
3. 當使用微血管的血液以point-of-care testing來檢測血糖時，應注意其結果可能會不夠準確