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*Grand Rounds*

# Critical Care Neurology for Junior Doctors; Four Key Management Strategies

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## INTRODUCTION:

As with all acutely unwell patients, the management of those with neurological insults should focus on promoting adequate oxygenation and perfusion to the compromised organ system. Brain injuries can be classified as either primary or secondary and can be ischaemic, traumatic, metabolic, inflammatory or multifactorial in aetiology. Prior to implementing targeted treatment based on a specific cause however, it is important to know how to provide general neurological protection. This protection involves improving oxygenation and perfusion of brain tissue and relies on four main management strategies: 1) Maintaining mean arterial pressure, 2) Reducing intracranial pressure if raised, 3) Optimising oxygen delivery and 4) Reducing oxygen demand. This article will adopt a case study and explore underlying physiology to illustrate how intensive care principles for neurological protection can be used in a ward-based environment by any junior doctor.

## CASE SCENARIO:

A 27-year-old male presents to the Emergency Department with lethargy, vomiting and a widespread non-blanching maculopapular rash. He is diagnosed with bacterial meningitis and is treated with appropriate antibiotics and steroids. He later complains of headache and has one tonic-clonic seizure. On examination he is drowsy, confused and his left pupil is dilated and non-reactive to light. His observations are as follows; blood pressure 86/45mmHg, heart rate 110bpm, temperature 38.7°C and oxygen saturations 86% on room air. How should a clinician approach the management of this critically unwell man? Using the steps outlined below one can protect against further neurological damage by using simple measures to promote adequate blood and oxygen supply to the brain.

## 1. MAINTAINING MEAN ARTERIAL PRESSURE:

Considering that perfusion to the brain relies on the cerebral perfusion pressure (CPP=MAP-ICP), it is usually reasonable to assume that optimising the mean arterial pressure should lead to an increase in cerebral perfusion.[1] Cerebral perfusion pressure should be maintained between 60mmHg-100mmHg, but as it is not feasible to calculate this exactly in a ward environment it is important to understand and apply the main principles of the equation. As mean arterial pressure is calculated using the systolic and diastolic blood pressures (MAP=SBP+2DBP/3), it is also safe to assume that by manipulating blood pressure you can increase the mean arterial pressure, to a target of over 65mmHg.[2] Therefore the key to management of this critically unwell patient is to monitor the blood pressure and treat any identifiable cause of hypotension, such as hypovolaemic shock (e.g. gastrointenstinal blood losses), distributive shock (e.g. sepsis, neurogenic shock), obstructive shock (e.g. tamponade, pulmonary embolus) and cardiogenic shock (e.g. myocardial infarction, cardiac failure). By administering intravenous fluids, packed red cells, antibiotics or by treating a tamponade or myocardial infarction you can increase the mean arterial pressure, thereby increasing perfusion to the brain and providing critical neurological support.

## 2. REDUCING INTRACRANIAL PRESSURE:

An equally important component of cerebral perfusion regulation is an appropriate intracranial pressure. The Monro-Kellie doctrine states that the skull is a rigid compartment with three main components of blood, cerebrospinal fluid and brain tissue, each of which can increase or decrease in volume to a certain degree without causing significant rises in intracranial pressure.[3] However, when such compensatory mechanisms are overwhelmed, intracranial pressure can rise, causing compression of arterioles and consequently a reduction in cerebral perfusion. Intracranial pressure should ideally be less than 15mmHg, but on a ward having this specifically monitored is not practicable. It is therefore crucial to recognise symptoms and signs of rising intracranial pressure (Figure 1) which include but are not limited to; headache, vomiting, visual disturbance, absence of pupillary reaction to light and perhaps most importantly, altered mental status. If one recalls the equation for cerebral perfusion pressure, treatment of increased intracranial pressure can salvage the brain tissue from the effects of hypoperfusion. Temporary reduction in intracranial pressure can be achieved

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by using hypertonic saline or mannitol but ultimately treatment must address the underlying cause such as surgical debulking of cerebral metastases or treatment of meningitis with antibiotics. Knowledge and application of the principles of the cerebral perfusion pressure equation can allow for general protective measures to be instigated whilst definitive therapy is awaited.

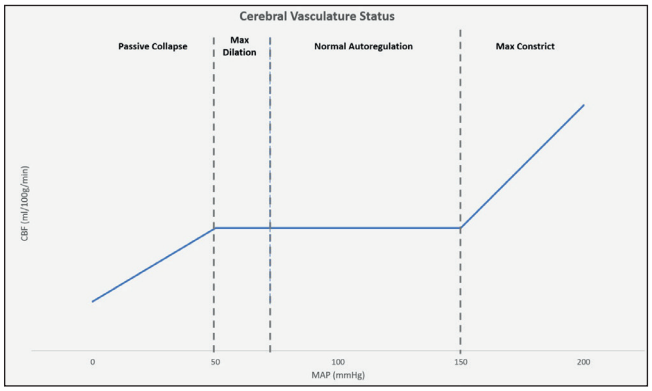


*Figure 1: Graph displaying relationship between mean arterial pressure, cerebral blood flow and autoregulation of cerebral vasculature.*

## 3. INCREASING OXYGEN DELIVERY:

In addition to perfusion, oxygenation is another essential component of critical neurological support. As above, using a simple equation can demonstrate how manipulation of various physiological processes can equip junior doctors with the key principles to maintain oxygen supply to the brain. The oxygen delivery equation can guide our management here as it lists some key components that can be manipulated on the ward;[4]

$DO2=CO \times (1.34 \times Hb \times SaO2 + (PaO2 + 0.003))$

DO2 is oxygen delivery

CO is cardiac output

1.34 is the oxygen binding capacity of haemoglobin

Hb is haemoglobin in g/l

SaO2 is haemoglobin oxygen saturation

PaO2 is partial pressure of oxygen

0.003 is the amount of dissolved oxygen in blood.

It is not essential to remember the specifics of the equation, however the understanding that cardiac output, haemoglobin level and oxygen saturation contribute to oxygen delivery allows the junior doctor to take ward-based actions that can improve oxygenation of vulnerable brain tissue. Cardiac output relies on heart rate and stroke volume, therefore using vasopressors or increasing preload with volume expansion can increase this. Transfusion of packed red cells to increase haemoglobin if anaemic or in the setting of acute blood loss as well as providing supplemental oxygen if saturations are low are other ways of increasing oxygenation. These are simple measures that can be taken by applying the principles of the oxygen delivery equation.

## 4. REDUCING OXYGEN DEMAND:

To ensure appropriate oxygenation to the brain one must also consider any increased oxygen demands that are present at the time of insult. These include fever, seizures, anxiety, agitation, pain, shivering and excess stimulation. A sensible approach reducing oxygen demand is to reduce stimuli, particularly in the first 24-48 hours of the injury. Antipyretics can be given and targeted temperature management (maintaining body temperature between 32 and 36 degrees Celsius) may be employed in situations such as in post-cardiac arrest.[5] Anticonvulsants may be required if seizures are present, including the use of benzodiazepines to terminate status epilepticus. Patients often require sedation if distressed or agitated and investigations must be undertaken to establish the cause, for example inadequate control of pain. In summary one should be mindful about any bodily processes that are energy-requiring and potentially superfluous and bring about their minimisation where possible, to reduce their effect of the oxygen demands of the brain.

## 5. WORKED CASE SCENARIO:

If we return to the above case of bacterial meningitis, we can see that each of the four principles can be applied here in order to maximise perfusion and oxygenation of brain tissue. The patient has a reduced mean arterial pressure, the underlying cause likely being vasodilatation from sepsis-induced bradykinin and prostaglandin release. He is also exhibiting signs of increased intracranial pressure, this likely arising from vasogenic oedema caused by inflammation of the meninges. He has also dropped his oxygen saturations due to an imbalance of delivery and demand. We can therefore increase perfusion to his brain by giving intravenous crystalloids to expand his intravascular volume. Prescription of mannitol might also be considered upon consultation with senior colleagues in order to reduce his intracranial pressure whilst being wary of dropping his blood pressure further. We can increase his oxygenation by giving supplemental oxygen through a non-rebreather mask and can reduce his oxygen demands by controlling his pyrexia with paracetamol and his seizures with anti-epileptic medication. These measures can be implemented on the ward whilst awaiting senior support and can be crucial in preventing irreversible neurological damage and potentially life-altering consequences.

## CONCLUSION

Using the above principles and treatment strategies, junior doctors can feel empowered to employ basic measures to improve oxygenation and perfusion to the brain, thereby protecting it whilst waiting for senior advice on definitive management. To improve perfusion to the brain we must consider mean arterial pressure and intracranial pressure and to improve oxygenation we must increase delivery and reduce demands. These four key strategies can be utilised on the ward, by any junior doctor and in doing so can protect

this vital organ in the critically unwell patient whilst awaiting further support.

**REFERENCES:**

1. Walters FJ. Intracranial pressure and cerebral blood flow. *Physiology*. 1998;8:1-4.
2. Nosek TM. Essentials of human physiology. Section 3/3ch7/s3ch7_4. Tampa, Fla: Gold Standard Multimedia; 1998
3. Mokri B. The Monro-Kellie hypothesis: applications in CSF volume depletion. *Neurology*. 2001;56(12): 1746–8.
4. McLellan SA, Walsh TS. Oxygen delivery and haemoglobin. *Contin Educ Anaesth Crit Care Pain*. 2004; 4: 123–6
5. Peberdy,MA;Callaway,CW;Neumar,RW;Geocadin,RG;Zimmerman, JL; Donnino, M; Gabrielli, A; Silvers, SM; Zaritsky, AL; Merchant, R; Vanden Hoek, TL; Kronick, SL; American Heart, Association (2 November 2010). "Part 9: post-cardiac arrest care: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care". Circulation. 122 (18 Suppl 3): S768–86. doi:10.1161/CIRCULATIONAHA.110.971002. PMID 20956225.