Sub-Arachnoid Haemorrhage - Guideline for management of patients admitted to Critical Care

Salford Royal NHS
NHS Foundation Trust
University Teaching Trust

safe • clean • personal

Classification: Clinical Guideline
Lead Authors: Dr MJ Naisbitt, Dr P Ferris
Authors Division: Critical Care

Unique ID: TWCG9(12)
Issue number: 2
Expiry Date: November 2017

Contents

Who should read this document?

This document should be read by all members of staff who look after and deal with patients with subarachnoid haemorrhage in critical care.

Key Messages

1. Subarachnoid haemorrhage is a complicated multi-system disease that requires focused and appropriate treatment guided by the multi-disciplinary team at a senior level

2. Initially, management should focus on preventing re-bleeding by devising and implementing a plan for securing the aneurysm in a timely manner by the most appropriate route and by the most appropriate clinicians. Again a multi-disciplinary approach is key

3. Appropriate cardiovascular management should be instituted from admission. This is often complicated by instability and unusual fluid shifts. Cardiac output monitoring is often required even in previously fit individuals.

4. The aim should be to establish and maintain euvolaemia. This requires frequent clinical assessment and appropriate monitoring.

5. Delayed cerebral ischaemia (often called vasospasm) can be devastating. Appropriate cardiovascular management and a high index of suspicion can minimise the impact.

Background & Scope

Subarachnoid haemorrhage is seen commonly at Salford with approximately 200 admissions to the critical care unit each year. Unfortunately these patients have a high morbidity and mortality.
The disease process is a complicated multi-system disorder which if managed appropriately and aggressively can lead to improved outcomes.
This document outlines the key medical interventions required for these patients. It does not go into surgical and interventional radiology processes that may be required.

What is new in this version?

1. Risk stratification for delayed cerebral ischaemia

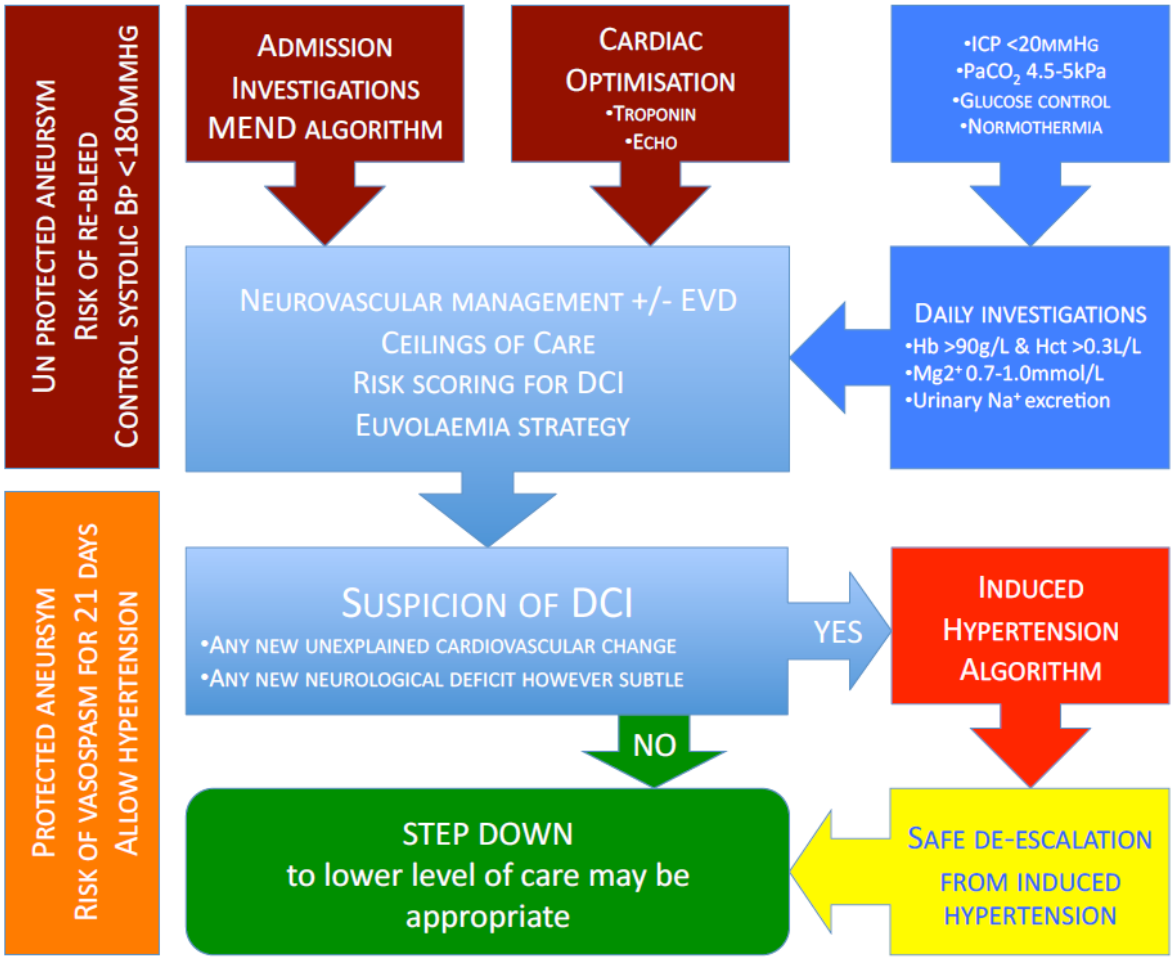
2. Guidance for ensuring euvolaemia in high risk patients with the use of cardiac output monitoring utilising transpulmonary thermodilution

3. Algorithms to guide treatment of delayed cerebral ischaemia utilising induced hypertension, including a de-escalation strategy

4. Guidance on diagnosing and managing abnormalities of sodium homeostasis associated with subarachnoid haemorrhage

5. Frequently asked questions section

Guideline Overview



Admission to Critical Care

All patients:

- Site ART line and aim MAP 80-90mmHg
- Consider CVC
- Maintain good oxygenation (PaO2>11kPa, SaO2 >95%)
- Stop antihypertensive medications
- Continue statin prescription if applicable

- Relatives discussion
- Multidisclipinary consultant review

Mechanically ventilated patients:
- Sedation to a level to attain targets and remain endotracheal tube tolerant
- Head up 30O
- PaCO2 4.5-5kPa
- ICP <20mmHg
- CPP> 60mmHg (if ICP is low then targets are MAP driven)

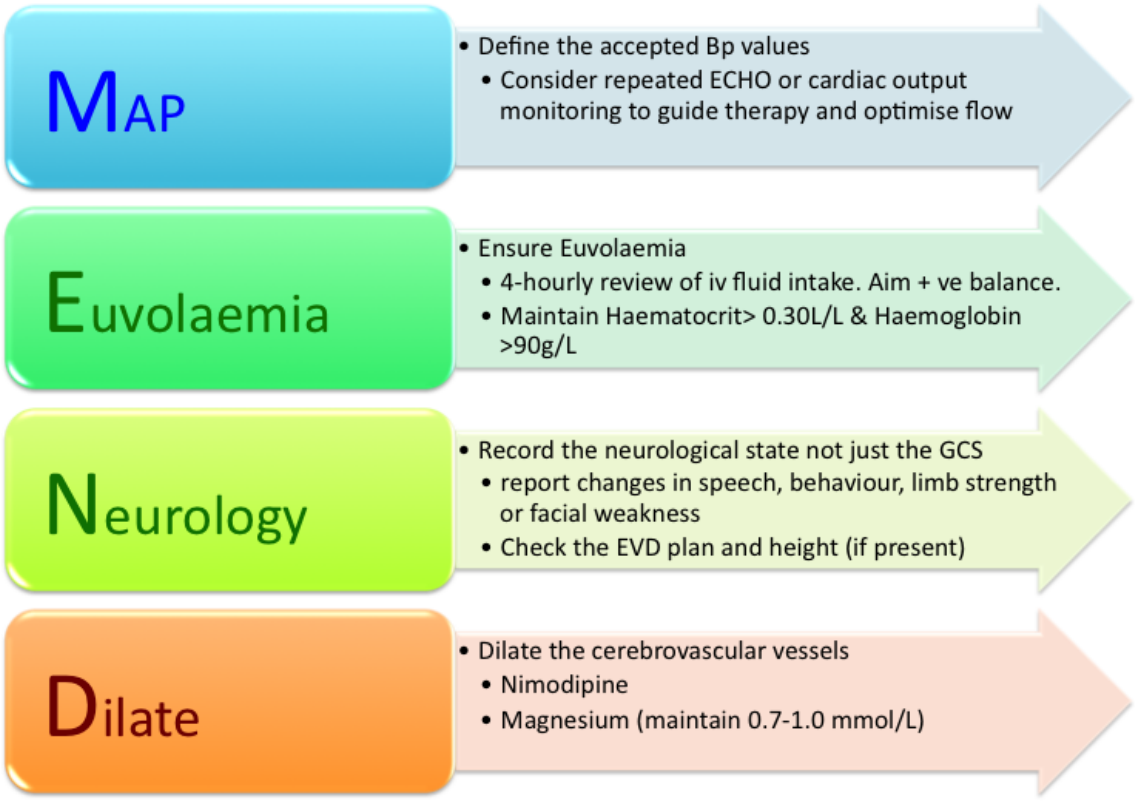
Documented review by:
- neurosurgical registrar within 2 hours
- critical care registrar within 2 hours
- critical care consultant within 12 hours
- neurosurgical consultant within 12 hours

Documented plan for aneurysm management should be made by the neurovascular team as soon as practicable (Coiling v Clipping v Conservative)

Investigations on admission:
- ECG
- Urea and Electrolytes
- Mg2+
- FBC
- G&S
- Troponin I
- Lactate
- Plasma glucose
- ABG
- CXR
- Consider ECHO

Hypovolaemia is common on admission and fluid resuscitation is often required to limit the degree of early brain injury and improve tissue perfusion. Serum lactate can be raised initially, often after the administration of mannitol or following seizure activity.

MEND Protocol



MEND sticker to all patients for first 14 days

MAP

- Define the mean arterial blood pressure target for the next 24 hours
    - Prior to Aneurysm protection Systolic Bp should be controlled <180mmHg using labetalol
    - After aneurysm protection cardiovascular targets should be agreed at each MDT ward round.
- Stop antihypertensive medications
- Do not treat hypertension after the aneurysm is protected unless there is current evidence of ongoing myocardial ischaemia, LV dysfunction or severe valvular disease. Assessment of the patient is required to rule out other causes of hypertension such as pain which should be treated appropriately.
- B-blockers may be continued but at a low dose

Euvolaemia

- Assessement of volaemic status is difficult.
- Patients with a good grade SAH require supplemental fluid therapy. For a 70kg man this would typically be 3 litres per 24 hours of crystalloid, usually plasmalyte. The amount of supplemental fluid may need to be modified depending on the patients age, weight and clinical circumstances.
- Sicker patients and those requiring mechanical ventilation are often more challenging. Aim for euvolaemia and to maintain serum Sodium 140mmol/l

- Aim slight positive fluid balance of up to 500ml daily with parenteral 50-150ml/hr Plasmalyte in addition to full enteral feed. Higher volumes of fluid may be required to maintain euvolaemia during hypertensive polyuric episodes. Invasive monitoring of cardiac output supplemented by the use of ECHO is useful.
- Sodium levels can fluctuate but hyponatraemia is problematic. Appendix 1 gives an outline of how to manage hyponatraemia
- Extra parenteral fluid may be required if insensible losses are high (pyrexia or diarrhoea)
- Daily weights are useful
- Haematocrit should be measured twice daily and maintained 0.30- 0.35l/l. Haemoglobin should be maintained >90g/l
- Report new unexplained cardiovascular disturbances (rise in MAP, HR)

Neurology

- A bedside neurological examination should be recorded every 2 hours by the nursing staff, **never just the GCS.** It is important the worst score is recorded (subtly different from traumatic brain inury neurology observations)
- Report agitation or changes in speech, behaviour, limb strength, facial weakness
- Report ANY subtle changes in neurology
- Report unexplained global decreases in GCS
- Ensure the EVD is zeroed at the ear and the prescribed plan and height are followed
- Report rises in ICP >20mmHg

Dilate

- Nimodipine 60mg 4-hourly enterally, (haemodynamically stable patients without a confirmed viable enteral route may be given IV nimodipine @10ml/hr- if vasoactive drugs are required to maintain MAP >90mmHg then consider stopping)
- Maintain serum Magnesium levels within normal limits (0.7-1.0mmol/L)
- Continue statins if prescribed pre-SAH

Euvolaemia: Rationale and Strategy

Patients who have suffered a spontaneous subarachnoid haemorrhage are in a vasoconstricted hypovolaemic low capacitance state on presentation due to high circulating levels of catecholamines and a generalised inflammatory response.

The primary brain injury caused by the haemorrhage, cerebral ischaemia and its subsequent inflammatory response causes a variable degree of early brain injury. The extent of this brain injury has been implicated in the subsequent risk of delayed cerebral ischaemia and the quality of survival.

Any brain injury can be exacerbated by the low oxygen delivery seen in a low cardiac output state. Hypovolaemia is thought to play a role in the etiology of delayed cerebral ischaemia and often temporally predates any clinical signs of DCI.

Historically hypervolaemic fluid strategies have been employed to manage SAH patients. These are associated with an increase in mortality secondary to an increase in the incidence of systemic physiology deteriorations e.g. Pulmonary oedema.

It is therefore important that we maintain tight euvolaemic control. This strategy is supported by international guidance on SAH management and forms a key part of the Salford MEND algorithm.

The clinical assessment of euvolaemia is difficult and wide variability can exist between different assessing clinicians or modalities of assessment.

Simple fluid balance guided strategies have been associated with a 54% incidence of severe hypovolaemia.

There is therefore a strong rationale to utilize cardiac output monitoring in patients whom are most at risk of delayed cerebral ischaemia, especially those who are sedated and mechanically ventilated. See Appendix 2

Delayed Cerebral Ischaemia

All patients who suffer a spontaneous subarachnoid haemorrhage are at risk of the development of delayed cerebral ischaemia. Patients with the highest degree of early brain injury are at the highest risk, i.e. the ventilated population.

The peak risk period is between 3-14 days post ictus.

Often when delayed cerebral ischaemia is clinically manifest it is referred to as vasospasm. 70% of patients have angiographic evidence of arterial narrowing after SAH but not all demonstrate an altered neurological state.

Specific risk factors for the development of DCI include:

- Poor grade (eg WFNS 4/5)
- Large blood load (Fisher scale III/IV)
- Current smoking
- Female
- Age <55 years
- Admission Glucose
- Hydrocephalus present on initial CT

The risk of DCI can be quantified by using the risk chart seen below:

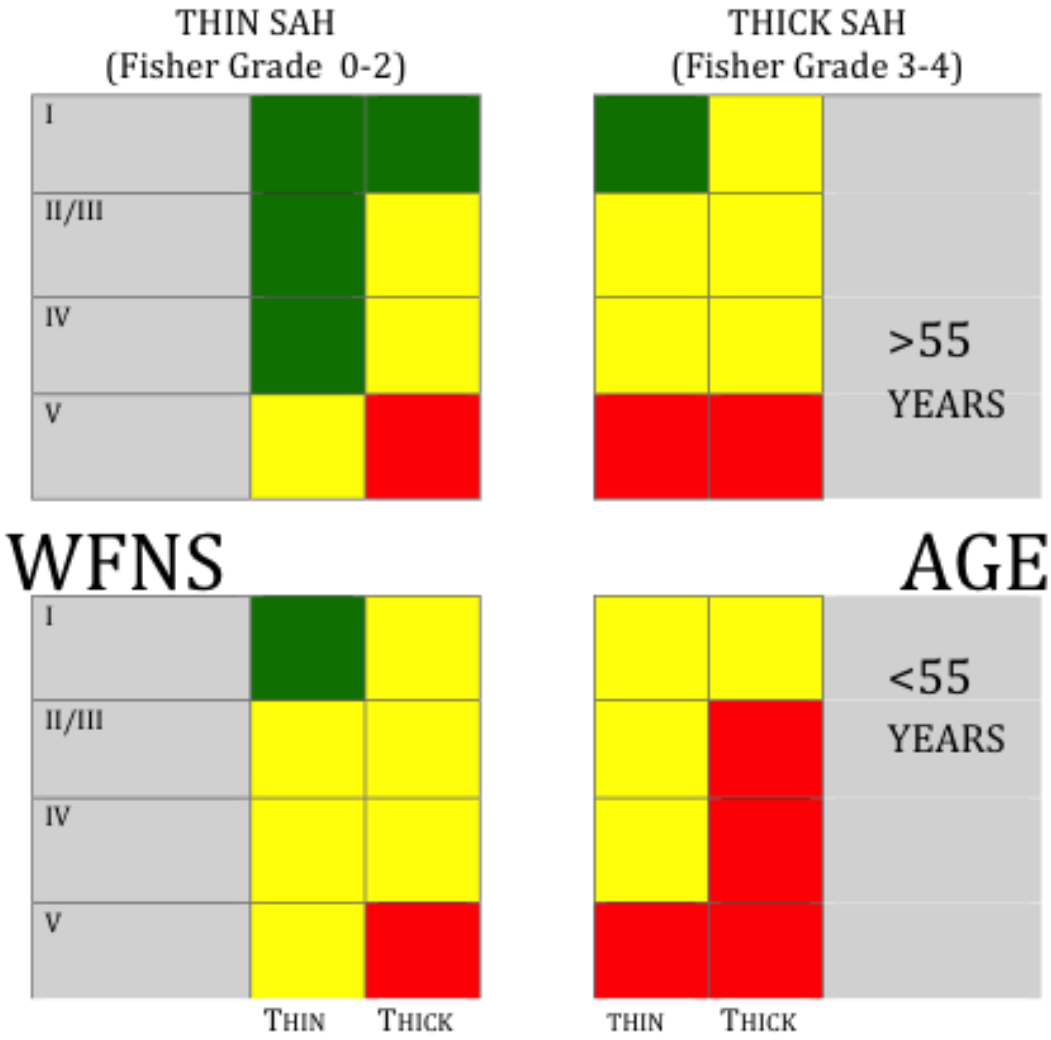
Low risk	<20% risk of clinically significant DCI
Medium risk	20-40% risk
High risk	>40% risk of clinically significant DCI

Low risk and most medium risk patients should be managed using a fluid balance based algorithm targeting a daily positive fluid balance.
In a **mechanically ventilated patient or patient scored as high risk** it is often difficult to assess volaemic status accurately. There should be consideration for cardiac output monitoring to guide fluid therapy and manipulation of the cardiovascular system. This may also apply to an active smoker in the medium risk group. See Appendix 2 for an example of an algorithm for managing volaemic status using invasive transpulmonary thermodiltuion cardiac output monitoring (Volumeview)

Risk of Delayed Cerebral Ischaemia in Aneurysmal Subarachnoid Haemorrhage

Low risk	Fluid balance based euvolaemia strategy
Medium Risk	Consider Invasive Euvolaemia Strategy
High Risk	Use Invasive Euvolaemia Strategy -Volumeview

Amount of cisternal blood on initial CT



Amount of intraventricular blood on CT

Thin = lateral ventricles show small degree of gravitational blood

Thick = more than 1 ventricle casted with blood or ≥3 show some sedimentation

Diagnosing and Treating Delayed Cerebral Ischaemia

Clinically significant delayed cerebral ischaemia following SAH can be a challenge to detect and manage following SAH. The following signs or symptoms should prompt urgent medical review.

Any new unexplained cardiovascular changes:

- Hypertension
- Tachycardia

Any abnormal spontaneous ventilatory pattern:

- Tachypnoea is associated with DCI and a poor outcome.

Any new neurological deficit however subtle:

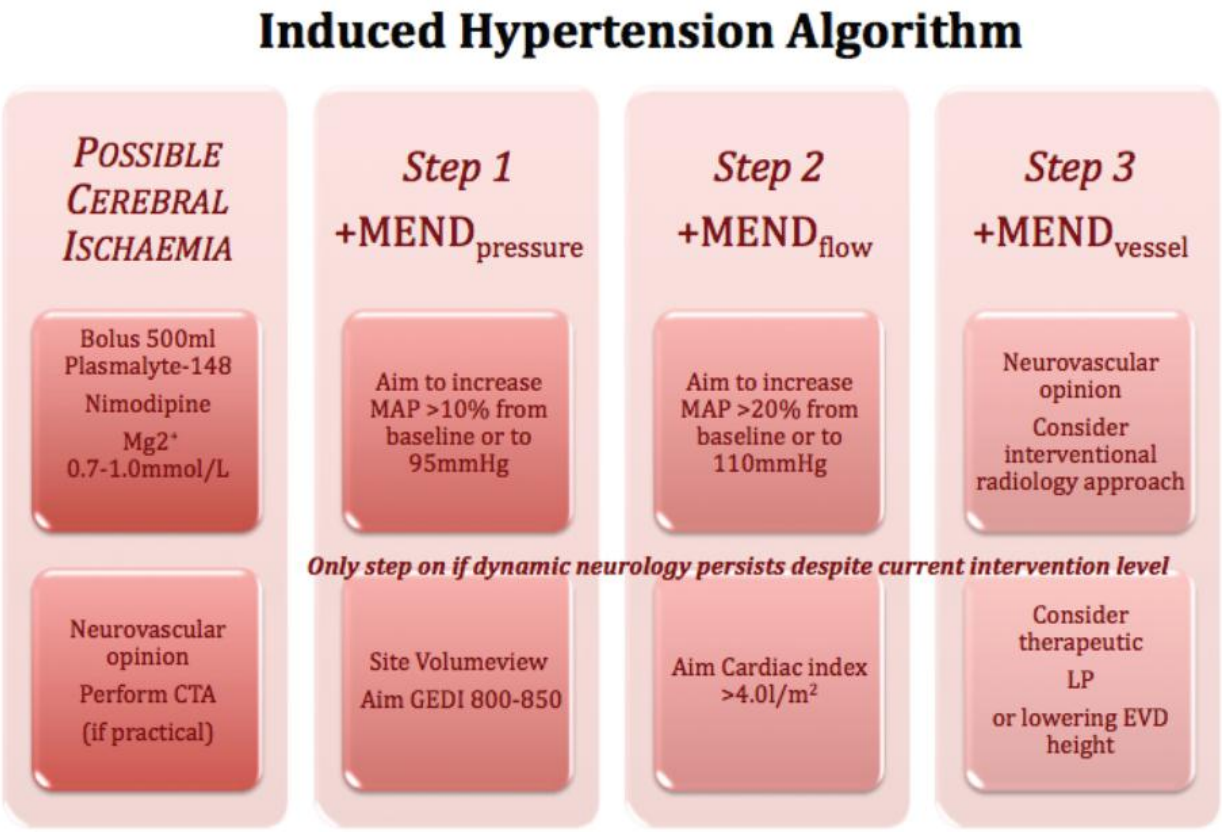
- Agitation or behavioural changes
- Drowsiness
- Reduced spontaneous interaction with people/carers
- Global reduction in GCS
- Increased effort to attain previous GCS
- Reduced time maintaining GCS after stimulation
- Changes in speech or new difficulty in comprehension
- Any new focal motor deficit including facial expression, strength in all 4 limbs
- The development of pronator drift

Always be suspicious of the possibility of delayed cerebral ischaemia. DO NOT TREAT unexplained hypertension in the at risk period.

Sedated ventilated patients are at high risk, particularly as our best monitor- their neurological state is lost.

Seek prompt MDT opinion and follow the induced hypertension algorithm below:

Slight hypercapnia to promote vasodilatation ($PaCO_2$ 5-6.5kPa) may be of benefit if an EVD is in-situ.



Inducing hypertension may be impossible or unsafe in patients with limited physiological reserve.

De-escalation from the Induced Hypertension algorithm

- Ongoing dynamic changes in neurology
- Reduction in global GCS
- Recent CTA evidence of spasm

- Stable neurology over last 24 hours
- >72 hours since diagnosis of clinically significant vasospasm

- No dynamic neurology present in last 48 hours
- >21 days since ictus

My patient has hypertension with a systolic blood pressure ≥180mmHg and the bedside monitor alarm is repeatedly sounding- What should I do?

Is the aneurysm protected/ coiled/ clipped?

NO — If it is unprotected and SBP ≥180mmHg there is a risk of a re-bleed from the unprotected weakness in a cerebral artery as a result of high blood pressure. The SBP should therefore be reduced to <180mmHg by an infusion of labetalol.

YES — Could the hypertension be a response to ongoing delayed cerebral ischaemia?
If yes follow the induced hypertension algorithm.
Is my patient in pain?
Could the hypertension be as a result of impending coning?
Hypertension should NOT be treated within the first 21 days following ictus unless there is a clear consultant critical care and neurovascular documented decision to do so.
Clear evidence of ongoing myocardial ischaemia, decompensated diastolic dysfunction or the presence of severe valve disease may warrant intervention to control BP acutely.

My patient has a new tachycardia with HR >110bpm - What is the cause?

Could the tachycardia be as a result of ongoing vasospasm?
Observational cohorts demonstrate that a high heart rate is associated with an increased incidence of delayed cerebral ischaemia.

Is my patient hypovolaemic?
Hypovolaemia is common especially on initial presentation. IT is also associated with poor outcome

Is my patient in pain?

Does my patient have other signs consistent with the development of an infection?
Be aware external ventricular drains have a 7% risk of ventriculitis.

My patient has a temperature of 39^{o}C –Why?

Marked pyrexia is often seen in patients with a subarachnoid haemorrhage and is associated with an increased incidence of delayed cerebral ischaemia. Commonly it is as a direct result of red cell breakdown within the subarachnoid space or within the

cerebrospinal fluid but the development of a new infection (VAP or CAUTI) should be ruled out. Pyrexia may also be secondary to any associated systemic inflammation.

Patients with pyrexia often have high insensible losses and may require high volumes of fluid to attain a positive fluid balance. A cold fluid bolus and Paracetamol is appropriate in most patients. active external cooling to target normothermia may be employed at consultant discretion.

My patient requires >10ml/hr 4mg:50ml Noradrenaline to achieve the MAP target- what is the next step?

Prior to this point aggresive management of the CVS system should have included the use of invasive cardiac output monitoring. If not already established it should be now urgently. (see Appendix 2)

In the presence of dynamic neurological changes within the last 24 hours or if myocardial function is impaired then re-calibrate cardiac output monitoring to ensure fluid status is optimised, aim for GEDI 800-850.

The decision to use >10ml/hr 4:50mg Noradrenaline should be made at a consultant level.

Very high doses of noradrenaline may result in profound arteriolar vasoconstriction and subsequently compromise cerebral blood flow to the point of increasing any ischaemic injury.

Could my patient have Vasospasm?

Are there any new unexplained cardiovascular changes:

- Hypertension
- Tachycardia

Are there any new neurological deficit however subtle:

- Agitation or behavioural changes
- Drowsiness
- Reduced spontaneous interaction with people/carers
- Global reduction in GCS
- Increased effort to attain previous GCS
- Reduced time maintaining GCS after stimulation
- Changes in speech or new difficulty in comprehension
- Any new focal motor deficit including facial expression, strength in all 4 limbs
- Pronator drift

If the answer is YES to any of the above, then assume that the patient is developing delayed cerebral ischaemia and inform a consultant member of the MDT.

Thromboprophylaxis

Mechanical methods alone until aneurysm protected.

Tinzaparin can be started immediately following successful endovascular coiling of the aneurysm.

Seek neurosurgical opinion on the when to start low molecular weight heparin following clipping or if there is a large unevacuated haematoma present.

Explanation of terms & Definitions

EVD – External ventricular drain

DCI – Delayed cerebral ischaemia
This is often referred to as 'vasospasm'. It is invariably caused by vasospasm, however not all vasospasm causes DCI.

WFNS – World Federation of Neurosurgeons scoring system
This is the scale used to grade severity of aneurysmal subarachnoid haemorrhage. It is based on the GCS and any neurogical deficit.
1 – GCS 15 and no deficit
2 – GCS 13-14 and no deficit
3 – GCS 13-14 with deficit
4 – GCS 7-12 with or without deficit
5 – GCS 3-6 with or without deficit

Fisher Scale
This scale is related to radiological findings on CT and correlates with risk of vasospasm
I – no blood
II – diffuse deposition of SAH without clots or layers of blood >1mm
III – localized clots and/or vertical layers of blood 1mm or > thickness
IV – diffuse or no subarachnoid blood but intracerebral or intraventricular clots

GEDI – Global End Diastolic Index
This is a derived value from transpulmonary thermodilution (Volumeview CO monitor) that is well validated to correlate with preload. It can be a useful indication of volaemic status when used in conjunction with clinical examination and other physiological parameters such as BP, HR and cardiac output.

Roles and responsibilities

The Critical Care Neurogovernance Group (CCNG) and particularly Dr Ferris and Dr Naisbitt shall be responsible for ensuring the appropriate dissemination of the guideline.
They will also be responsible for ensuring care of these patients is audited against the standards highlighted within the guideline, and developing strategies to ensure compliance is high.

References

1. Early Intensive Versus Minimally Invasive Approach to Postoperative Hemodynamic Management After Subarachnoid Hemorrhage. ***Stroke*. 2014;45:1280-1284**


Stroke aSAH GEDI.pdf

2. Critical Care Management of Patients Following Aneurysmal Subarachnoid Hemorrhage: Recommendations from the Neurocritical Care Society's Multidisciplinary Consensus Conference. ***Neurocrit Care* (2011) 15:211–240**


Critical Care Management of Patie

3. Delayed neurological deterioration after subarachnoid haemorrhage. **Macdonald, R. L. *Nat. Rev. Neurol.* 10, 44–58 (2014);**


natureDCI.pdf

Appendix 1: Pressure Natriuresis, Diuresis and the Place for Fludrocortisone

The hypertension associated with a subarachnoid haemorrhage often causes a physiological pressure natriuresis and diuresis.

It is paramount that any patient who has suffered aneurysmal subarachnoid haemorrhage does not become volume deplete and hyponatraemia is avoided. If a high fluid flux state has developed and delayed cerebral ischaemia is suspected then cranial diabetes insipidus must be ruled out.

Several mechanisms for pressure natriuresis and diuresis have been proposed:

- Systemic hypertension leads to an increase in capillary and vasa recta pressure increasing the movement of H_2O and Na^+ into the proximal convoluted tubule and the descending limb of the loop. A subsequent increase in renal medullary interstitial pressure counteracts the normal osmotic gradient, leading to reduced reabsorption of NaCl in the loop and consequently reduced H_2O reabsorption from the collecting duct.
- Noradrenaline causes increased local renal prostaglandin production. This directly inhibits NaCl reabsorption in the thick ascending limb of the loop of Henle.

Sodium levels often fluctuate in critically unwell patients and there needs to be consideration of trends and other medical conditions prior to aggressive therapy. **Fluid restriction is the wrong treatment for hyponatraemia in the context of subarachnoid haemorrhage during the risk period for DCI (up to 21 days post ictus)**

Early warning of the development of a clinically significant natriuresis can be measured by tracking the urinary sodium excretion daily:

Random urine Na^+ x 24hour urine output = Daily urinary Na^+ excretion
(mmol/L) (L) (mmol)

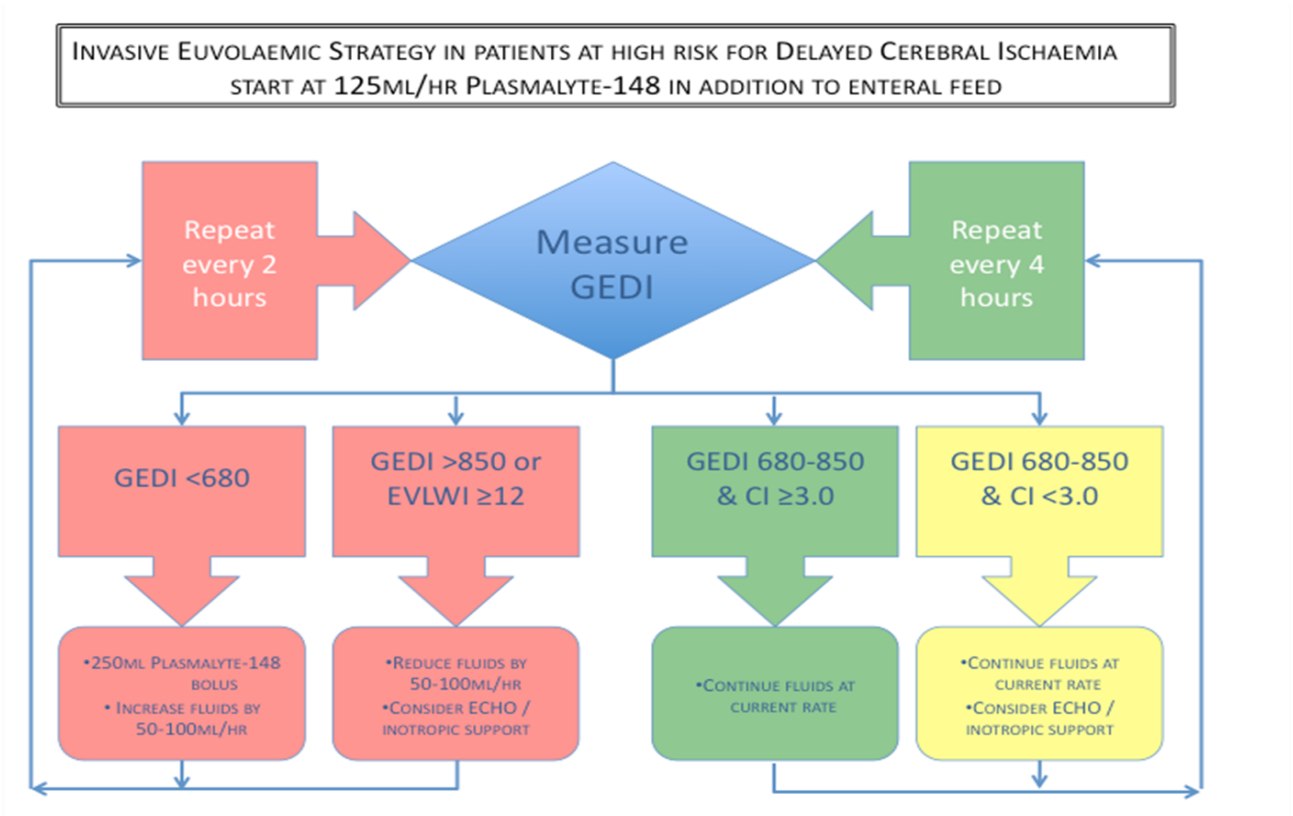
If urinary Na^+ excretion ≥300mmol/24hours AND the serum sodium is <135 or has fallen by 5 mmol over last 24 hours, prescribe 200 micrograms Fludrocortisone twice a day.

Once commenced Fludrocortisone should be continued for at least 7 days or until the urinary Na^+ excretion normalises ≤100mmol/24hours, which may be up to 14 days later.

Acute onset hyponatraemia resulting in plasma Na^+ ≤135mmol/L or a drop ≥5mmol/L should prompt medical review. Any diuretics should be stopped and consideration given to Na^+ supplementation. There is an enteral preparation available (5 mmol per milliliter) Discuss with pharmacy

Appendix 2 : Algorithm for Cardiovascular Management with the Volumeview Cardiac Output Monitor

Cardiovascular management of patients with subarachnoid haemorrhage is difficult. Patients have a variety of reasons for instability. They are often hypovolaemic on admission, they also often have a degree of myocardial dysfunction due to subendocardial ischaemia at the time of ictus. Also, the cardiovascular pathophysiology is fluid and liable to changes.
For these reasons it is imperative to ensure a thorough assessment is undertaken. This includes review of fluid balance taking in factors such as pyrexia, clinical examination and assessment of simple CVS parameters such as HR and BP, response to simple measures such as passive leg raise. Invasive cardiac output monitoring, as well as intermittent ECHO can be helpful in guiding this assessment, and in sedated patients is usually required.
Therapy should initially be aimed at ensuring euvolaemia, and then focused on optimising cardiac output as shown in the algorithm below utilising the Volumeview cardiac output monitor.



Volumeview cardiac output monitor is a transpulmonary thermodilution method of measuring cardiac output that is well validated. The line kit is expensive and difficult to insert and should be only undertaken by an experienced practitioner.