

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

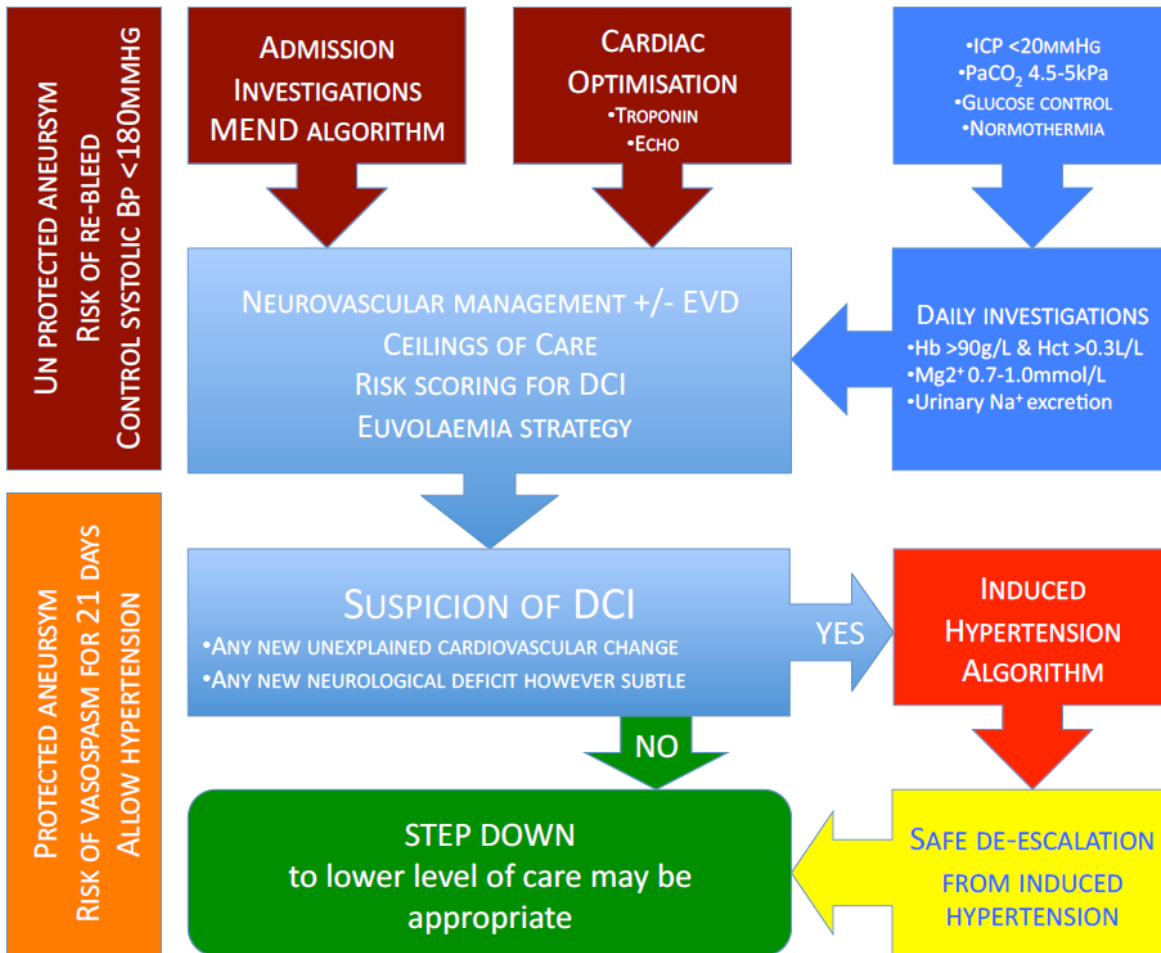
Lead Author:	Dr Jay Naisbitt, Consultant in Critical Care Medicine
Additional author(s)	Dr Paul Ferris, Consultant in Critical Care Medicine Dr Andrew Claxton, ST6 in Critical Care Medicine Emma Parkin, ACCP in Critical Care Medicine Dr Jack Hodd, ST8 in Critical Care Medicine
Division/ Department::	Critical Care Medicine
Applies to: (Please delete)	Salford Royal Care Organisation
Approving Committee	Critical Care Governance
Date approved:	26/11/2019
Expiry date:	November 2022

### Contents

Section	Page
<a href="#">Document summary sheet</a>	3
1 <a href="#">Overview</a>	4
2 <a href="#">Scope &amp; Associated Documents</a>	4
3 <a href="#">Background</a>	4
4 <a href="#">What is new in this version?</a>	4
5 <a href="#">Guideline</a>	5
5.1 <a href="#">Admission to Critical Care</a>	5
5.2 <a href="#">MEND Protocol</a>	6
5.3 <a href="#">Delayed Cerebral Ischaemia</a>	11
5.4 <a href="#">Diagnosing and Treating Delayed Cerebral Ischaemia</a>	12
5.5 <a href="#">De-escalation of therapy in SAH</a>	14
5.6 <a href="#">Trouble-Shooting Guide in Subarachnoid Haemorrhage</a>	15
5.7 <a href="#">Thromboprophylaxis</a>	22
6 <a href="#">Roles and responsibilities</a>	22
7 <a href="#">Monitoring document effectiveness</a>	23
8 <a href="#">Abbreviations and definitions</a>	23

9	<a href="#">References</a>	24
10	<a href="#">Appendices</a>	25
	<a href="#">Appendix 1 Pressure Natriuresis, Diuresis and the Place for Fludrocortisone</a>	25
	<a href="#">Appendix 2 Algorithm for Cardiovascular Management with the Volumeview Cardiac Output Monitor</a>	27
	<a href="#">Appendix 3 National Institute for Health Stroke Score (NIHSS)</a>	28
11	<a href="#">Document Control Information</a>	29
12	<a href="#">Equality Impact Assessment (EqIA) screening tool</a>	30

**Document Summary Sheet**  
**[Critical Care Management of Sub-arachnoid Haemorrhage]**



## 1. Overview (What is this guideline about?)

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.

## 2. Scope (Where will this document be used?)

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

### Associated Documents

- N/A

## 3. Background (Why is this document important?)

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.

## 4. What is new in this version?

1. Descalation of fluid therapy section.
2. Updated guidance on use of inotropes, vasopressors and cardiac output monitoring.
3. Updated guidance on management on ICU admission
4. Updated algorithm for detection and management of vasospasm/delayed cerebral ischaemia.

5. Updated guidance on clinical examination of the patient with SAH.
6. Guidance on management of raised ICP in subarachnoid haemorrhage.

## 5. Guideline

### 5.1 Admission to Critical Care

All patients:

- Site arterial line and aim for a Mean Arterial Pressure 80-90mmHg
- Consider CVC (peripheral metaraminol to achieve MAP targets is a short-term option – a CVC and noradrenaline should be commenced as soon as is practicably possible).
- Maintain good oxygenation (PaO<sub>2</sub>>11kPa, SaO<sub>2</sub> >95%)
- Stop antihypertensive medications
- Continue statin prescription if applicable
- Relatives discussion
- Multi-disciplinary consultant review

Mechanically ventilated patients:

- Sedate to attain targets and remain endotracheal tube tolerant. Aim RASS -2 to -3.
- Head up 30°
- PaCO<sub>2</sub> 4.5-5kPa
- ICP <20mmHg
- CPP> 60mmHg (if ICP is low then targets are MAP driven).
- If aneurysm UNPROTECTED, ensure systolic BP <180mmHg.

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)

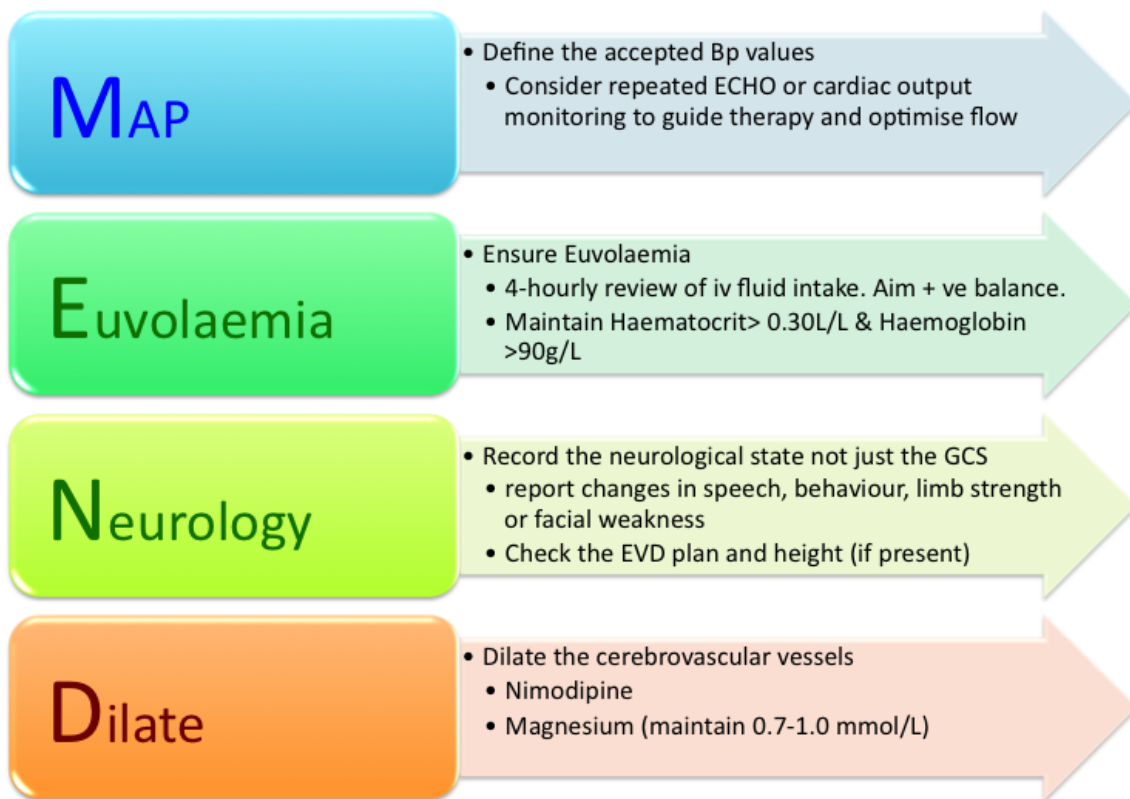
Investigations on admission:

- ECG

- Urea and Electrolytes
- Mg<sup>2+</sup>
- FBC
- G&S
- Troponin I
- Lactate
- Plasma glucose
- ABG
- CXR
- ECHO – initially FICE echo where possible, if ongoing cardiac instability then consider formal 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.**

## 5.2 MEND Protocol



MEND sticker to all patients for first 14 days

The objective of the MEND protocol in patients with subarachnoid haemorrhage is to optimise cerebral blood flow and thus oxygenation to compromised cerebral tissue, as well as reducing the risk of vasospasm and delayed cerebral ischaemia.

## MAP

By ensuring adequate MAP, we ensure adequate cerebral perfusion pressure (CPP) as  $CPP = MAP - ICP$ . Cerebral blood flow (CBF) is autoregulated to remain consistent between a CPP of approximately 60-150mmHg.

The considerations for the MAP are:

- 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.
- Beta blockers may be continued but at a low dose
- Where blood pressure augmentation is required to achieve MAP/CPP targets there should be careful consideration of which agent(s) to use. Clinical assessment and cardiac output monitoring are mandated in the high risk patient (see section 5.3), as this will help guide further fluid resuscitation. Echo (FICE or formal) may help uncover left ventricular systolic dysfunction (common following SAH). A clinical picture of the cause of relative hypotension should be constructed. Appendix X outlines some potential strategies for this.

## Euvolaemia

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, however these strategies 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, and 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 (see section 5.3), especially those who are sedated and mechanically ventilated. See [Appendix 2](#) for guidance on cardiac output monitoring (VolumeView).

The considerations for Euvolaemia are:

- Assessment of volaemic status is difficult.
- In good grade SAH patients:
  - 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.
  - 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. It may be useful to the next hour's fluid rate to the previous hours urine output (i.e. a previous hour urine output of 150ml, should mean 150mL/hr crystalloid for the next hour).
- In poor grade SAH patients and those at high risk of vasospasm/delayed cerebral ischaemia:
  - Aim for euvolaemia and to maintain serum Sodium 140mmol/L. These patients should have cardiac output monitoring sited as soon as is practicably possible. The VolumeView system should be used (see appendix 2 for a guide to it's use).
  - Echo (FICE or formal) can be used to help guide fluid status, and give an indication of cardiac function. This can be used to guide the use of vasopressors (noradrenaline/metaraminol) vs. inotropes (dobutamine/enoximone).
- In awake patients the assessment of euvolaemia can be guided by assessing patient thirst, moistness of mucous membranes, skin turgor, capillary refill time, urine output and fluid balance charts. No single measure will be totally representative of the volaemic status however together these indices can build a picture.
- All SAH patients:
  - Urine output may increase, driven by an augmented MAP to achieve an acceptable CPP, as well as by increase intravascular volume. Hyponatraemia in these patients is common. Appendix 1 gives an outline of how to manage hyponatraemia in these patients.
  - 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. Both these values can be found on ABGs.
  - Report new unexplained cardiovascular disturbances (rise in MAP, HR, RR)

## Neurology

**THE BEST TOOL FOR ASSESSING NEUROLOGY IS AN AWAKE PATIENT. AIM TO WAKE AND EXTUBATE PATIENTS AS SOON AS IS SAFE/FEASIBLE.**

**A bedside neurological examination should be recorded every 2 hours by the nursing staff, never just the GCS.**

The worst score should always be recorded - which is different to our TBI population.

Initial weakness that occurs after the bleed will generally be related to the vessel/territory involved.

Sub-Arachnoid Haemorrhage - Guideline for management of patients admitted to Critical Care			
Reference Number TWCG9(12)	Version 3	Issue Date: 09/12/2019	Page 8 of 30



*For example:*

*Anterior vessel bleeds may be result in foot or leg weakness*

*Middle cerebral vessel bleeds may result in arm or speech deficits*

*Posterior vessel bleeds may result in bulbar function deficits*

**However when a patient develops vasospasm this can occur anywhere within the circle of Willis and therefore any subtle or obvious changes to neurology should be charted and an ACCP/Doctor should be informed.**

### **Scoring or motor function**

**Obeying commands** – The patient should be able to obey commands with limbs and obey a number of commands , e.g, squeeze hand and let go, bend leg, move toes.

**Localising to pain** – the patient should react for your hand, arm to at least clavicle

**Flexion to pain** – Normal pattern of movement towards a stimulus but not high enough to reach the stimulus

**Abnormal flexion** – Flexion of the limb but not in coordinated way towards the stimulus

<b>MRC Muscle Power Assessment</b>	
<b>Score</b>	<b>Description</b>
0	No contraction
1	Flicker or trace of contraction
2	Active movement, with gravity eliminated
3	Active movement against gravity
4	Active movement against gravity and resistance
5	Normal power

**Extension** – Extension of limb

### **Power grading**

Power of limbs should be assessed using the MRC power scale to minimize subjectivity. Each limb should be assessed separately and documented.

### **Pronator Drift**

Pronator drift is an easy way to pick up subtle upper motor neuron issues.

This should be assessed as part of the patients regular neurological assessment .

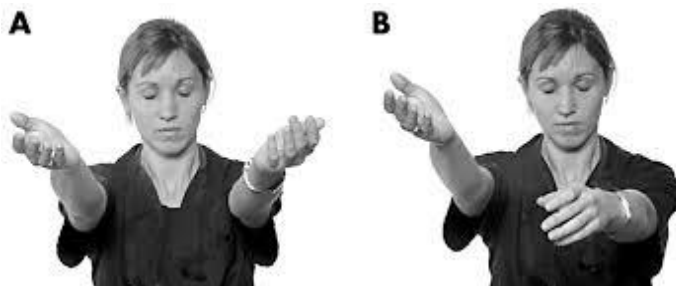


Figure A – Normal no evidence of drift

Figure B – Left sided drift, this could indicate right sided cerebral lesion/issue

## Speech

Is the patient now having trouble finding words or expressing themselves?

### Dysphasia Vs Aphasia

Aphasia is more severe and involves a complete loss of speech or comprehension. Dysphasia involves moderate language impairment.

Expressive aphasia	The patient knows what they want to say but has difficulty communicating
Receptive aphasia	The patient can hear voice or read print, but will not be able to understand the meaning of the message
Anomic aphasia	The person has word finding difficulty
Global aphasia	The person has difficulty understanding words and communicating.

## Behavioral changes

Is the patient alert, lethargic or confused, is this new?  
Are they orientated to time, person, place and or situation?  
Can they concentrate?  
Can they name the prime minister?  
Can they re-call words after a time delay?

**All patients should have neurology assessed. This includes lightly sedated patients whose neurology can be assessed when patients are exposed to stimulus such as suction or being turned.**

## Assessing neurology in patients under sedation

- What is the patient doing to stimulus such as suction or being turned?
- Make note which limbs are moving and how powerful they are
- Has this changed despite sedation being the same or being reduced?
- Make sure that any changes are highlighted to the medical team.

Each day the patient should have a National Institution for Health Stroke Score (NIHSS) completed by the medical team and repeated if there is deterioration. This should be documented on EPR and on the NIHSS flowchart.

See appendix 4 for detailed NIHSS document

## Dilate

The objective for this section of the protocol is to mitigate the risk of vasospasm by keeping cerebral blood vessels dilated. Nimodipine is a calcium channel blocker which has a degree of selectivity for cerebral blood vessels. Nimodipine acts on the calcium channels of the vascular

smooth muscle, preventing constriction. The role for magnesium as a vasodilator is poorly understood, and research regarding its use is conflicting. However as a physiological antagonist to calcium, it is rational to keep it within normal range.

Considerations for dilation:

- 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). If BP regularly drops after enteral nimodipine administration of 60mg 4-hourly, consider administering 30mg 2-hourly enterally, or converting to IV nimodipine. If ongoing CVS instability – consider stopping nimodipine. This MUST be a consultant/senior medical MDT decision.
- Maintain serum Magnesium levels within normal limits (0.7-1.0mmol/L)
- Continue statins if prescribed pre-SAH

### 5.3 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 (green)	<20% risk of clinically significant DCI
Medium risk (yellow)	20-40% risk
High risk (red)	>40% risk of clinically significant DCI

Low risk and most medium risk patients should be managed as per the MEND protocol, ensuring a mild positive fluid balance of approximately 500mL per day, and aiming for an Na<sup>+</sup> concentration of 140mmol/L.

In a **mechanically ventilated patient or patient scored as high risk** it is often difficult to assess volaemic status accurately. Cardiac output monitoring should be considered to help 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 cardiac output monitoring (VolumeView).

VASOGRADE	WFNS	Modified Fisher scale
Green	1-2	1-2
Yellow	1-3	3-4
Red	4-5	Any

#### 5.4 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:

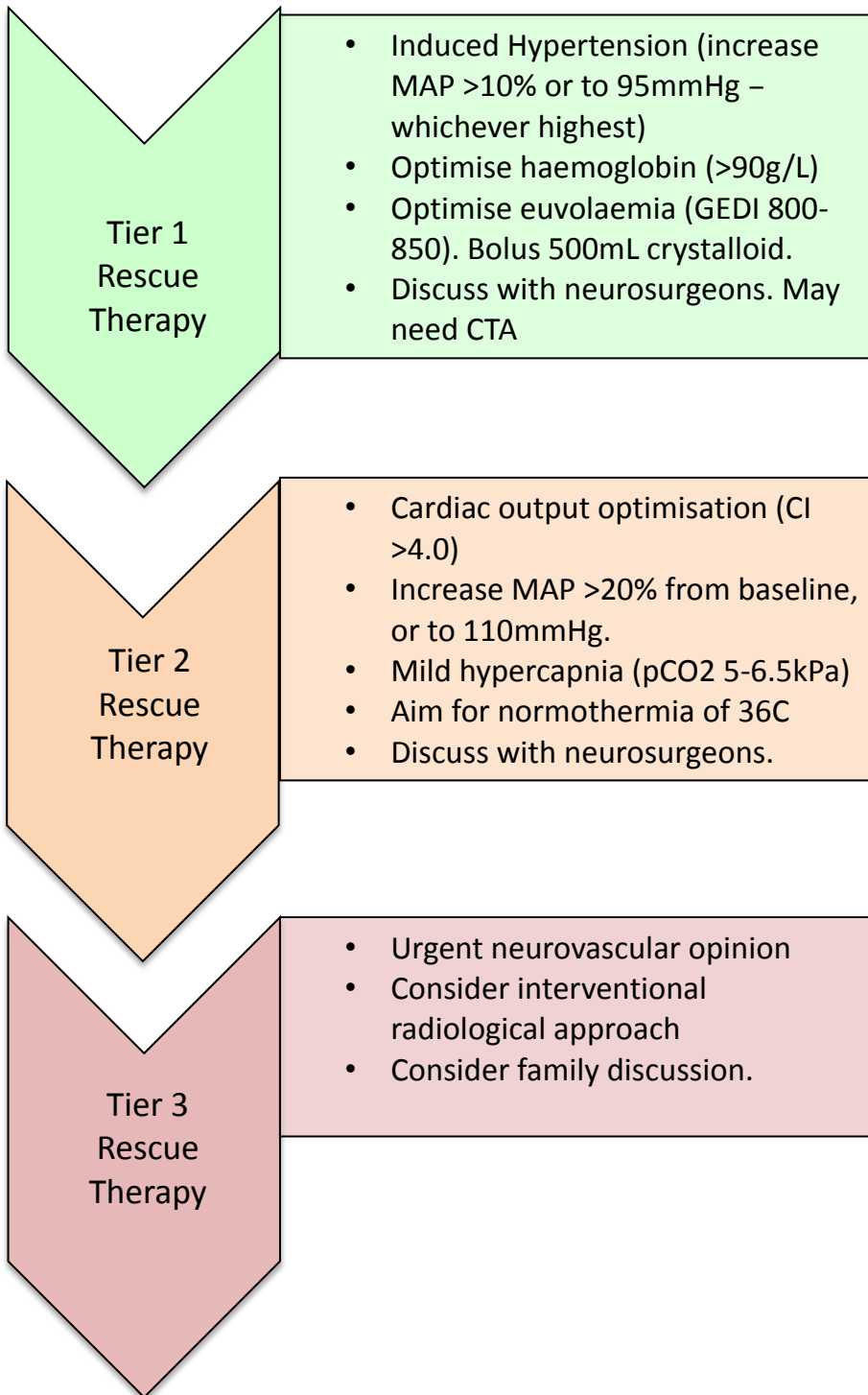
*Agitation or behavioural changes*  
*Drowsiness*  
*Reduced spontaneous interaction with people/carers*  
*Global reduction in GCS*  
*Increased effort to attain previous GCS*  
*Reduced time maintaining GCS after stimulation*  
*Changes in speech or new difficulty in comprehension*  
*Any new focal 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<sub>2</sub> 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

DO NOT  
De-  
escalate

- ONGOING DYNAMIC CHANGES IN NEUROLOGY
- REDUCTION IN GLOBAL GCS
- RECENT CTA EVIDENCE OF SPASM

Consider  
Trial 10%  
reduction  
in MAP

- STABLE NEUROLOGY OVER LAST 24 HOURS
- >72 HOURS SINCE DIAGNOSIS OF CLINICALLY SIGNIFICANT VASOSPASM

De-  
escalate

- NO DYNAMIC NEUROLOGY PRESENT IN LAST 48 HOURS
- >21 DAYS SINCE ICTUS

### 5.5 De-escalation of therapy in SAH

Patients who have suffered a subarachnoid haemorrhage are at risk from deterioration for weeks after the event. The principles of the MEND protocol should continue for 21 days at least, however some of these targets may become increasingly hard to achieve. For example patients may begin to diurese excessively due to the large volumes of IV fluid they have received, which results in further IV fluid to achieve euvolaemia, leading to a higher urine output etc etc... With the peak risk period for vasospasm/DCI occurring between days 3-14, in poor grade SAH patients, de-escalation of therapy should not be considered until after this period.

De-escalation of therapy should be an MDT-lead decision made in conjunction between intensive care and neurosurgeons. When de-escalating therapy in SAH patients, there needs to be extreme attention paid to the risk of vasospasm, with regular neurological examination.

There is no strong evidence base to suggest a strategy for de-escalating therapy; it should take a personalised approach based upon clinical judgment. Stepwise reductions in MAP target towards a baseline of 65mmHg (higher if patient is normally hypertensive) maybe readily achievable, however if ICP remains in the higher range of normal, targeting a MAP of 65mmHg will result in a lower CPP and therefore potentially lower cerebral blood flow, increasing the risk of DCI.

Within the highly monitored confines of critical care, fluid therapy should be rigorously monitored, aiming for a positive daily fluid balance of 500mL.

## 5.6 Trouble-Shooting Guide in Subarachnoid Haemorrhage

1. My patient has hypertension with a systolic blood pressure  $\geq 180$ mmHg 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  $\geq 180$ mmHg 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  $< 180$ mmHg 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.*

2. My patient has a new tachycardia with HR  $> 110$ bpm  
- 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.*

3. My patient has a temperature of  $39^{\circ}\text{C}$  –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.*

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

*Prior to this point aggressive 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 use of echo can help to identify if cardiac dysfunction is the cause of hypotension. In this setting, inotropy may be more beneficial than escalating vasopressors such as noradrenaline.*

*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.*

There are four main reasons why the need for blood pressure augmentation may increase in an SAH patient:

1. Hypovolaemia. This may persist despite initial fluid resuscitation, and especially if mannitol has been given. Lactate, urine output, echo, and cardiac output monitoring can be used to assess the need for further fluid.
2. Cardiac stunning. The catecholamine surge associated with subarachnoid haemorrhages can result in cardiac dysfunction and poor left ventricular contractility. Troponin, echo and cardiac output monitoring can be used to assess cardiac function. If LV dysfunction is present, then escalating noradrenaline can worsen this as it increased afterload. Consider dobutamine or enoximone.
3. Vasoplegia. Although there is an endogenous release of catecholamines following SAH, patients can develop a pro-inflammatory response, which results in vasoplegia. Use of VolumeView may help assess this. If there is good evidence that this is the cause (i.e. low SVR) consider adding a second vasoactive agent such as argipressin and administering endogenous corticosteroids.
4. High ICP. In order to achieve a CPP > 60mmHg, a higher MAP may be required if ICP is high (CPP = MAP – ICP). If this is the case, ensure good secondary neuroprotective measures are in place, and discuss with neurosurgeons. Is the EVD still draining? Has there been a rebleed? If EVD is still draining, then AFTER DISCUSSION WITH NEUROSURGEONS ONLY, the EVD height *may* be lowered to facilitate CSF drainage. See troubleshooting point 6 for further information in managing high ICP in SAH patients.



## 5. 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.

## 6. My patient has a high ICP

When waking patients ICP can transiently rise as the patient becomes more stimulated. SAH patients should aim to have a sedation hold with view to extubating as soon as is practical/safe. A rise in ICP is often symptomatic of the waking process, and spikes can occur from coughing on the endotracheal tube or other stimuli. If a high ICP is not related to the waking process nor does it settle within an appropriate time frame, then the cause of the high ICP should be investigated and appropriate therapies instigated as such (guidance below).

Sustained high ICP in aneurysmal SAH (aSAH) is associated with worse prognosis, particularly >20mmHg.<sup>1</sup> Raised ICP occurs on average in up to 50% of aSAH patients, with a higher incidence in high grade aSAH.<sup>2</sup> The causes of raised ICP in SAH can be due to oedema, haematoma, hydrocephalus, intraventricular haemorrhage, aneurysm re-rupture, aneurysm treatment or delayed cerebral ischaemia (DCI).<sup>1</sup> High ICP can occur early (<24hrs), subacutely (up to 7-10 days) or with a delay (>10 days).

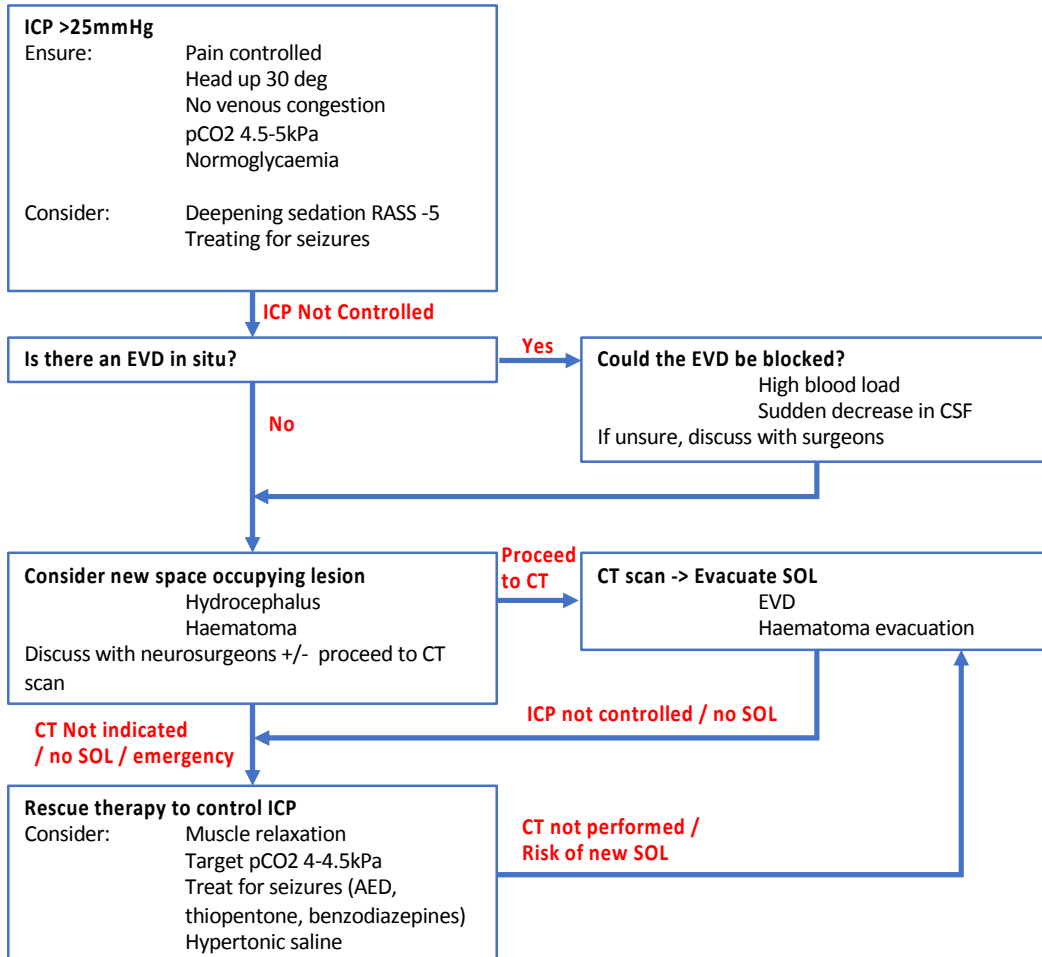
Control of high ICP in patients with SAH is complex, with a very limited evidence base. The information below is a guide and senior input should be sought as soon as possible.

Some specific points to consider:

1. Is there an EVD and is it draining? Hydrocephalus can result in high ICP. Call neurosurgeons if EVD is in situ and not draining. If no EVD, then CT brainlab is likely needed prior to placement.
2. Has there been a re-bleed? There may be frank blood coming through the EVD, or there may not be. Discuss with neurosurgeons, a CT/CTA is likely needed to evaluate this.

- Is there vasospasm? Are there clinical signs of this – i.e. increased HR, or RR, a change in neurology? If in doubt, follow induced hypertension algorithm and seek neurovascular opinion.
- It is important to consider the family/next of kin and where possible discussions to update them regarding changes in prognosis should occur as soon as is practicable.

## Algorithm for Raised ICP in aSAH



## Elements of care that are unlikely to be harmful and should be implemented

Little evidence has been found to support the following interventions, but they are unlikely to harm, may benefit ICP management and should be implemented.

Note that prophylactic administration of antiepileptic medications has not so far been found to be of benefit in aSAH. Of particular note, PHENYTOIN IS ASSOCIATED WITH WORSENING OUTCOMES IN aSAH AND SHOULD BE AVOIDED.

Head up 30 degrees	Unlikely to harm. Recommended
Avoid venous congestion	Unlikely to harm. Recommended
Give antiepileptics IF SEIZURES SUSPECTED of raising ICP	Unlikely to harm. Recommended
Neuromuscular blockade	Unlikely to harm. Recommended

## Hydrocephalus

The most common cause of raised ICP in patients with aSAH, occurring in 50%. Can be communicating or obstructive in nature. Early detection and control of hydrocephalus through the diversion of CSF is lifesaving.

Be aware that a rising ICP in a patient with an EVD may signify a blocked EVD, particularly in patients with high ventricular blood load.

Diversion of CSF in hydrocephalus (eg EVD)	Beneficial.
--	-------------

## Haematoma evacuation

Haematoma formation occurs in 30% of aSAH patients. Subdural haematoma occurs in 5% of patients.

If present, evacuation of haematoma should be considered and discussed with the neurosurgeons.

Haematoma evacuation	May be beneficial. Can be considered.
----------------------	---------------------------------------

## Manipulation of pCO<sub>2</sub>

There is evidence in murine models that cerebral microvasculature exposed to subarachnoid blood during the immediate phase of simulated SAH loses CO<sub>2</sub> reactivity.<sup>3</sup> This effect is also found in patients with poor grade aSAH.<sup>4</sup> Therefore the manipulation of pCO<sub>2</sub> in early (0-3 days) aSAH may have little effect in CBF in areas of the brain affected by subarachnoid blood.

Retrospective cohort analysis of patients with aSAH showed an association between abnormal pCO<sub>2</sub> (i.e. not between 35-45mmHg or 4.6-6kPa) during the first 14 days and adverse outcome. The signal for adverse outcome was greater with a pCO<sub>2</sub> below this range than above. Other studies have shown increased adverse outcomes below pCO<sub>2</sub> of 4kPa.<sup>5</sup>

However, higher levels of pCO<sub>2</sub> have been shown to at least transiently increase cerebral blood flow in patients with poor grade subarachnoid haemorrhage between days 4-10. However, this rise in CBF is associated with increased intracranial pressure.<sup>6</sup>

The evidence therefore is limited to guide the management of pCO<sub>2</sub> in patients with aSAH. It seems that aiming for a low normal 4.5-5kPa is safe, particularly in the first 3 days post-ictus, however after this time point, a low-normal pCO<sub>2</sub> may exacerbate the development of DCI. After day 3 a pCO<sub>2</sub> of 5-6kPa is optimal, with scope for an increase in pCO<sub>2</sub> up to 6.5kPa in the setting of suspected DCI.

Lowering pCO <sub>2</sub> below 4kPa to control ICP	<b>NOT recommended</b>
Targeting pCO <sub>2</sub> 4-4.5kPa to control ICP	Effect Unknown. Caution as may exacerbate DCI (especially after day 3)
Targeting pCO <sub>2</sub> 4.5-6kPa	Thought Safe. Note higher pCO <sub>2</sub> might raise ICP.

## Global oedema

Found in 8% of patients early, rising to 10-12% later.

### Hyperosmolar agents

Hyperosmolar agents lead to a change in blood fluid dynamics and decrease brain water content and thus volume in the presence of an intact blood brain barrier.

Mannitol is associated with a marked diuresis resulting in hypovolaemia and hypotension. These are at odds with the therapies used for vasospasm in aSAH.<sup>1</sup>

Hypertonic saline improves cerebral blood flow and exerts a positive inotropic effect in addition to haemodilution and hyperosmolality. The Brain Trauma Foundation therefore suggests that hypertonic saline is probably better than mannitol, though with no good evidence. Hypertonic saline has been shown to decrease ICP in aSAH. No complications have been found in retrospective cohort studies using hypertonic saline infusions, and there is an association with improved perfusion to ischaemic areas.

There is no difference in the efficacy of hypertonic saline and mannitol to decrease ICP in aSAH. The drop in ICP associated with hypertonic saline is reported as significant, but only 3-9mmHg.<sup>2</sup> Most centres target Na<sup>+</sup> of 155-160 and osmolality of 320mEq.

There are suggestions from pilot studies that hyperoncotic 20% human albumin solution may be of benefit in aSAH. However a phase III trial is ongoing. Note that administration of 4.5% HAS was found to increase mortality in brain injured patients in the SAFE trial.

### Recommendation

<b>Mannitol</b>	<b>Not Recommended In Raised ICP in aSAH</b>
<b>Hypertonic saline</b>	May be beneficial. Can be considered.
<b>20% HAS bolus</b>	May be beneficial. Can be considered.

## Induced hypothermia

Intraoperative hypothermia of 33-35C during coiling of aneurysms has **not** been found on systemic review to be associated with improving outcomes in the IHAAT trial.

There is very limited evidence looking at the ongoing management of refractory ICP elevation in patients with aSAH. Mild therapeutic hypothermia was used in a cohort of 100 patients with refractory raised ICP for 7 days with 36.5% achieving a good outcome.

Adverse rates are as high as 93%. In particular, hypothermia induced reduction in CO<sub>2</sub> production may induce cerebral vasoconstriction.

Where patients are pyrexia, cooling to normothermia is feasible, safe, and likely beneficial in reducing ICP.

### Recommendation

Mild therapeutic hypothermia	Feasible but Not Recommended in raised ICP in aSAH
------------------------------	--

## Barbiturate Coma

Barbiturates act by potentiating GABA effects at the GABA<sub>A</sub> receptor resulting in decreased metabolic rate. This decreases CBF and oxygen consumption.

Early case series suggested that patients with refractory vasospasm complicating aSAH had good recovery compared to controls. However, there are significant side effects associated with barbiturate coma. Hypotension is observed in 25% of patients with barbiturate coma, which is antagonistic to the decrease in ICP, also possibly worsening vasospasm and DCI.<sup>1</sup>

Barbiturate coma impairs observation of neurological status to target therapies for DCI, inhibiting appropriate evidence-based responses to vasospasm.

### Recommendation

Barbiturate Coma (infusion)	Not Recommended in Raised ICP in aSAH
Barbiturate bolus to terminate seizures	May be beneficial. Can be considered. No evidence found.

## Decompressive Craniectomy

Meta-analysis shows that decompressive craniectomy in aSAH for either refractory high ICP or mass effect associated with infarction is associated with poor outcomes in 60% and death in 29%. This is not dissimilar to outcomes without decompression. However the quality of evidence is low.

Decompressive Craniectomy	Not Recommended in Raised ICP in aSAH
---------------------------	---------------------------------------

## 5.7 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.

## 6. Roles & 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.

## 7. Monitoring document effectiveness

There is ongoing review of all patients admitted with subarachnoid haemorrhage by both the critical care team and the neuro-vascular team.

The management of these patients is regularly scrutinised and any mortality and morbidity is discussed at the neuro-critical care governance meetings and the critical care mortality and morbidity meetings.

On occasion certain aspects of the care are audited but this is not routine.

## 8. Abbreviations and 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 neurological 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

## 9. References

1. Early Intensive Versus Minimally Invasive Approach to Postoperative Hemodynamic Management After Subarachnoid Hemorrhage. *Stroke*. 2014;45:1280-1284
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
3. Delayed neurological deterioration after subarachnoid haemorrhage. Macdonald, R. L. *Nat. Rev. Neurol.* 10, 44–58 (2014);



## 10. Appendices

### 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<sub>2</sub>O and Na<sup>+</sup> 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<sub>2</sub>O 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<sup>+</sup> x 24hour urine output = Daily urinary Na<sup>+</sup> excretion  
(mmol/L) (L) (mmol)

**If urinary Na<sup>+</sup> 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<sup>+</sup> excretion normalises ≤100mmol/24hours, which may be up to 14 days later.

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

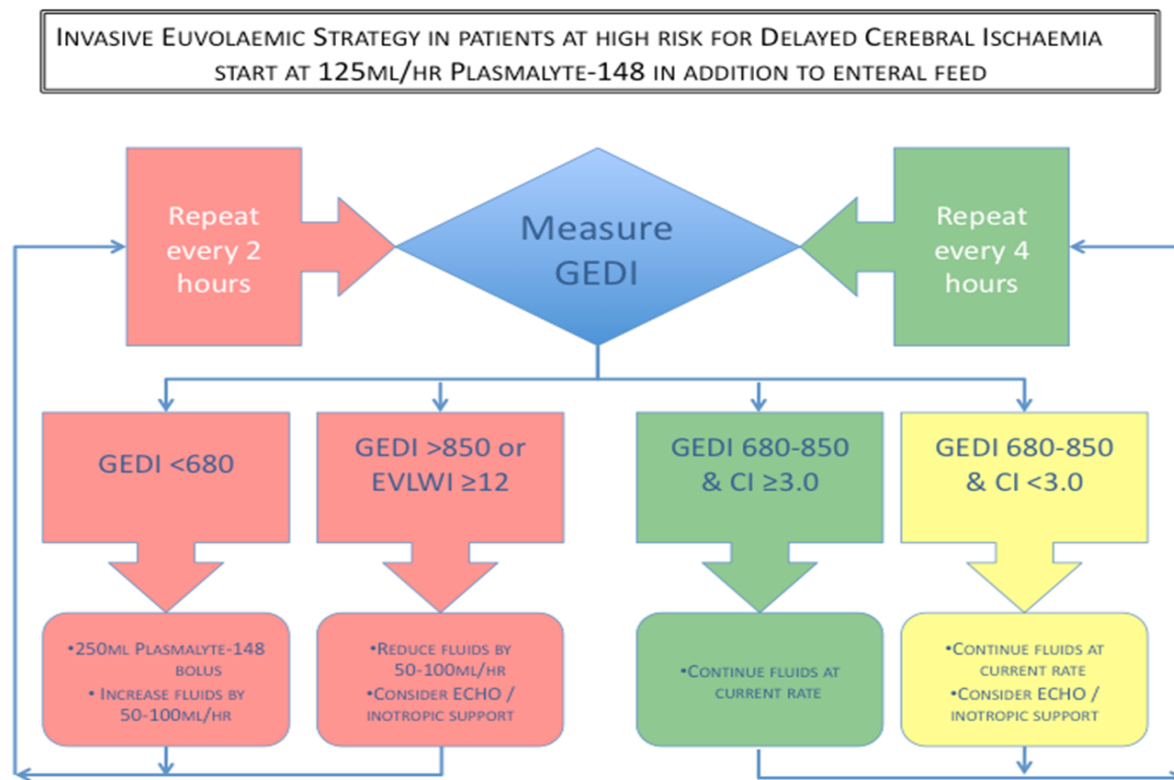
Cranial diabetes insipidus (DI) is most often caused by an injury or tumor of the pituitary gland. In subarachnoid haemorrhage, the cause of polyuria is highly **unlikely** to be DI, **therefore the**

patient should **NOT** be given DDAVP without discussion with a consultant/advanced trainee. High urine output within this cohort is most commonly due to high fluid input and pressure naturesis due to supra-normal MAP targets. Hypovolaemia should be avoided, and thus in high urine output states, the hourly fluid input can be titrated against the previous hour's urine output. Cardiac output monitoring (Oesophageal Doppler or VolumeView) may be used to help guide fluid management (appendix 2).

## 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.

## Appendix 3

### National Institute for Health Stroke Score (NIHSS)

1a—Level of consciousness	0 = Alert; keenly responsive 1 = Not alert, but arousable by minor stimulation 2 = Not alert; requires repeated stimulation 3 = Unresponsive or responds only with reflex
1b—Level of consciousness questions: What is your age? What is the month?	0 = Answers two questions correctly 1 = Answers one question correctly 2 = Answers neither questions correctly
1c—Level of consciousness commands: Open and close your eyes Grip and release your hand	0 = Performs both tasks correctly 1 = Performs one task correctly 2 = Performs neither task correctly
2—Best gaze	0 = Normal 1 = Partial gaze palsy 2 = Forced deviation
3—Visual	0 = No visual lost 1 = Partial hemianopia 2 = Complete hemianopia 3 = Bilateral hemianopia
4—Facial palsy	0 = Normal symmetric movements 1 = Minor paralysis 2 = Partial paralysis 3 = Complete paralysis of one or both sides
5—Motor arm Left arm Right arm	0 = No drift 1 = Drift 2 = Some effort against gravity 3 = No effort against gravity 4 = No movement
6—Motor leg Left leg Right leg	0 = No drift 1 = Drift 2 = Some effort against gravity 3 = No effort against gravity 4 = No movement
7—Limb ataxia	0 = Absent 1 = Present in one limb 2 = Present in two limbs
8—Sensory	0 = Normal; no sensory loss 1 = Mild-to-moderate sensory loss 2 = Severe-to-total sensory loss
9—Best language	0 = No aphasia; normal 1 = Mild-to-moderate aphasia 2 = Severe aphasia 3 = Mute; global aphasia
10—Dysarthria	0 = Normal 1 = Mild-to-moderate dysarthria 2 = Severe dysarthria
11—Extinction and inattention	0 = No abnormality 1 = Visual, tactile, auditory, spatial, or personal inattention 2 = Profound hemi-inattention or extinction
Score = 0–42	

## 11. Document Control Information

All sections must be completed by the author prior to submission for approval

<b>Lead Author:</b>	Dr Paul Ferris/Dr Jay Naisbitt		
<b>Lead author contact details:</b>	60955 Paul.ferris@srft.nhs.uk		
<b>Consultation</b> List the persons or groups who have contributed to this guideline. (please state which Care Organisation)	<b>Name of person or group</b>	<b>Role / Department / Committee (Care Org)</b>	<b>Date</b>
	Critical care neuro-governance	SRFT –critical care/neurosurgery	19/11/19
	Critical care governance	Critical care	26/11/19
<b>Endorsement</b> List the persons or groups who have seen given their support to this guideline. (please state which Care Organisation)	<b>Name of person or group</b>	<b>Role / Department / Committee (Care Org)</b>	<b>Date</b>
	Critical care neuro-governance	SRFT –critical care/neurosurgery	19/11/19
	Critical care governance	Critical care	26/11/19
<b>Keywords / phrases:</b>	Subarachnoid haemorrhage SAH MEND Brain haemorrhage stroke		
<b>Communication plan:</b>	Via governance structure Communication with key stake-holders Induction of critical care staff		
<b>Document review arrangements:</b>	This document will be reviewed by the author, or a nominated person, at least once every three years or earlier should a change in legislation, best practice or other change in circumstance dictate.		

This section will be completed following committee approval

<b>Guideline Approval:</b>	Name of Approving Committee: SRFT critical care governance	
	Chairperson: Dr Haslett	
	Approval date: 26/11/2019	
	<b>Formal Committee decision - Approved</b>	<b>Chairperson's approval - Approved</b>

## 12. Equality Impact Assessment (EqIA) screening tool

Legislation requires that our documents consider the potential to affect groups differently, and eliminate or minimise this where possible. This process helps to reduce health inequalities by identifying where steps can be taken to ensure the same access, experience and outcomes are achieved across all groups of people. This may require you to do things differently for some groups to reduce any potential differences.

<b>1a) Have you undertaken any consultation/ involvement with service users, staff or other groups in relation to this document?</b> If yes, specify what.	<i>Yes, as highlighted above</i>		
<b>1b) Have any amendments been made as a result?</b> If yes, specify what.	<i>Yes – numerous small alterations as advised by expert opinion</i>		
<b>2) Does this guideline have the potential to affect any of the groups below differently?</b> <i>Place an X in the appropriate box: Yes, No or Unsure</i> This may be linked to access, how the process/procedure is experienced, and/or intended outcomes. Prompts for consideration are provided, but are not an exhaustive list.			
Protected Group	Yes	No	Unsure
<b>Age</b> (e.g. are specific age groups excluded? Would the same process affect age groups in different ways?)		X	
<b>Sex</b> (e.g. is gender neutral language used in the way the guideline or information leaflet is written?)		X	
<b>Race</b> (e.g. any specific needs identified for certain groups such as dress, diet, individual care needs? Are interpretation and translation services required and do staff know how to book these?)		X	
<b>Religion &amp; Belief</b> (e.g. Jehovah Witness stance on blood transfusions; dietary needs that may conflict with medication offered.)		X	
<b>Sexual orientation</b> (e.g. is inclusive language used? Are there different access/prevalence rates?)		X	
<b>Pregnancy &amp; Maternity</b> (e.g. are procedures suitable for pregnant and/or breastfeeding patients?)	X		
<b>Marital status/civil partnership</b> (e.g. would there be any difference because the individual is/is not married/in a civil partnership?)		X	
<b>Gender Reassignment</b> (e.g. are there particular tests related to gender? Is confidentiality of the patient or staff member maintained?)		X	
<b>Human Rights</b> (e.g. does it uphold the principles of Fairness, Respect, Equality, Dignity and Autonomy?)		X	
<b>Carers</b> (e.g. is sufficient notice built in so can take time off work to attend appointment?)	X		
<b>Socio/economic</b> (e.g. would there be any requirement or expectation that may not be able to be met by those on low or limited income, such as costs incurred?)		X	
<b>Disability</b> (e.g. are information/questionnaires/consent forms available in different formats upon request? Are waiting areas suitable?) Includes hearing and/or visual impairments, physical disability, neurodevelopmental impairments e.g. autism, mental health conditions, and long term conditions e.g. cancer.		X	

<p><b>Are there any adjustments that need to be made to ensure that people with disabilities have the same access to and outcomes from the service or employment activities as those without disabilities?</b> (e.g. allow extra time for appointments, allow advocates to be present in the room, having access to visual aids, removing requirement to wait in unsuitable environments, etc.)</p>		x	
<p><b>3) Where you have identified that there are potential differences, what steps have you taken to mitigate these?</b></p> <p>This is an updated SRFT guideline and areas of best practice from previous guidelines have been carried over. Updates are stated in section 4 of the guidelines.</p> <p>Subarachnoid haemorrhage is seen commonly at Salford with approximately 200 admissions to the critical care unit each year. The disease process is a complicated multi-system disorder which if managed appropriately and aggressively can lead to improved outcomes, but an individual's protected characteristic has no bearing on the guidelines apart from anyone who is pregnant.</p> <p>No maternity services at Salford, so if a pregnant patient were to be admitted with SAH (very rare 1-2 per decade) the current pathway is for them to be transferred to Preston neurosurgical unit</p> <p><b>4) Where you have identified adjustments would need to be made for those with disabilities, what action has been taken?</b></p> <p>Carers will be involved/updated</p>			
<p><b>Will this guideline require a full impact assessment? Yes / No</b>  <i>(a full impact assessment will be required if you are unsure of the potential to affect a group differently, or if you believe there is a potential for it to affect a group differently and do not know how to mitigate against this - Please contact the Inclusion and Equality team for advice on <a href="mailto:equality@pat.nhs.uk">equality@pat.nhs.uk</a>)</i></p> <p>Author: Type/sign: <span style="margin-left: 200px;">Paul Ferris</span> <span style="float: right;">Date: 4/12/19</span></p> <p>Sign off from Equality Champion: <i>smulle</i> <span style="float: right;">Date: 05/12/19</span></p>			