	The Walton Co	ontre NHS		
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	Excellence in Neur	oscience		
Subarachnoid Haemorrhage				
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Review Date: June 2021 Version: **1.0** Page **1** of **19**

1. Introduction

Aneurysmal Subarachnoid haemorrhage (aSAH) is a serious medical condition in which outcome can be dramatically impacted by early, aggressive, expert management. (1, 2). Failure to diagnose subarachnoid haemorrhage (SAH) rapidly exposes the patient to the fatal effects of a further bleed, and also to complications which can be avoided or successfully treated. (1, 2, 3, 4)

Subarachnoid Haemorrhage (SAH) is defined as bleeding into the subarachnoid space (and, therefore, into the cerebrospinal fluid (CSF)). Cerebral arteries lie within the subarachnoid space. Therefore, bleeding from associated aneurysms occurs primarily into this space. *The arachnoid layer can sometimes be disrupted causing intracerebral haemorrhage or occasionally, subdural haematoma.*

The rupture of a cerebral aneurysm is a medical emergency. If not diagnosed and transferred to a specialist centre in a timely manner will risk rebleed, which is at its highest in the first 24 hours. This can ultimately result in significant disability or death.

Some patients present in poor clinical condition, requiring complex management of pulmonary oedema, cardiac failure, and arrhythmias.

Others will need emergency evacuation of a cerebral haematoma or decompressive craniectomy to control mass effect, emergency treatment for hydrocephalus or management of seizures; all of which are frequent occurrences with this patient group.

aSAH is a significant cause for morbidity and mortality throughout the world. The annual incidence in the UK is approximately 7/100,000 (5).

In contrast to more common types of stroke, aSAH often occurs at a relatively young age: half the patients are younger than 60 years.

The outcome of patients with aSAH is generally poor: half the patients die within one month of the haemorrhage, and of those who survive the first month, half remain dependent for help with activities of daily living (walking, dressing, bathing). Only 25% of patients can expect to return to a relatively normal life following SAH (6)

1.1. Purpose of this guideline:

This guideline serves to support the medical and nursing management of the patients presenting to the Walton centre following spontaneous subarachnoid haemorrhage.

- 1.2. Presenting symptoms and signs of SAH:
 - Sudden onset of headache (thunderclap). Distinctive and often described as "worst ever" (8).
 - The onset of headache may be associated with additional signs and symptoms such as
 - Nausea and/or vomiting.
 - Neck stiffness.
 - Photophobia.
 - Collapse/Brief loss of consciousness.
 - Focal neurological deficits (including cranial nerve palsies)
 - Seizures occur in approximately 20% of patients after a SAH. These are most common in the first 24 hours.

- Alterations in GCS/consciousness.
- Cardiopulmonary complications (neurogenic pulmonary oedema)
- Neurocardiogenic injury (arrhythmias, ECG changes)
- Most intracranial aneurysms remain asymptomatic until they rupture. Some patients may experience a "warning" or sentinel headache that precedes the SAH. These typically occur 2-8 weeks before the SAH (9).

1.3. Recommendations for diagnosis of SAH:

- SAH should be suspected in any patient who presents with a sudden onset of headache
- Diagnosis is confirmed by non-contrast computerised tomography (CT) brain.
- A CT scan is positive in up to 98% of patients with SAH presenting within 12 hours but is positive in only 50% of those presenting within one week. Cerebrospinal fluid (CSF) bilirubin spectrophotometry can be used to determine the need for angiography in those few CT-negative patients in whom clinical suspicion of SAH remains high; LP may remain positive up to two weeks after the event, therefore, if CT is negative then Lumbar puncture (LP) should be performed no less than 12 hours following ictus; 2-3 % of patients with negative CT scan.
- Positive lumbar puncture will prove SAH (10).
- Sufficient volume of CSF should be taken (the first should be a minimum of 0.5 ml in an EDTA tube for glucose and protein analysis; Microbiology requires 5 mls in 2 sterile CSF containers labelled '2nd' and '3rd'; a further 1 ml (20 drops) minimum should be collected in a sterile CSF container, protected from the light (in the usual specimen bag inside a thick brown envelope) and labelled 'fourth'. Use of a pneumatic transport system should be avoided and a paired serum sample to assess serum bilirubin and total protein should accompany the 4th CSF sample. Time taken should be stated on the request form and specimen carriers. (11)
- Magnetic Resonance Imaging can be considered for diagnosis although doesn't remove the need for LP if negative.
- CT angiography (CTA) may be requested before a patient is accepted for further investigation or treatment.

The management of patients with aneurysmal subarachnoid haemorrhage demands expertise to anticipate, recognise, and promptly treat the many neurological and systemic complications.

For this reason, these patients are best cared for in high-volume medical centre's with multidisciplinary team involvement and should preferably be treated by a specialised centre in specialist ward and/or intensive care unit depending on stability.

After diagnosis the neurosurgical registrar on call at the Walton Centre should be contacted for further advice and acceptance for ongoing management.

They can be contacted via the hospital switchboard on 0151 525 3611.

- Once accepted, the registrar will discuss the safest place to manage the patient with the bed management team or the anaesthetist in charge of the critical care unit as well as informing the neurovascular team.
- If the patient is of poor WFNS grade, at risk of deterioration or is not stable, level 2 or 3 care will be organised.
- Otherwise, the patient will be placed within the highly specialised neurovascular ward (

- If advice is needed over the appropriate placement of the patient then the Neurovascular Specialist Nurses (______) or SMART (______) should be contacted.
- The nursing team from the accepting ward will contact the referring hospital to enquire on the condition of the patient and ensure the patient is placed where they can receive the appropriate level of care supported by the multidisciplinary team (12).

Once a place is found then the referring hospital should contact the receiving ward in order to organise transfer.

A quick reference guide is available on managing the patient in the appendices of this document (appendix 1).

1.4. Before transfer:

Assess:

- Airway Ensure maintenance and stability of airway before and during transfer.
- Breathing O2 to maintain SaO2 >95% using supplemental O2 if needed.
- Circulation Insert intravenous cannula and commence 0.9% saline / plasmalyte 148 at 125 mls/hour if safe to do so. Maintain blood pressure.
- Disability (including blood sugar). Accurate GCS including pupil response. Validate WFNS score
- Examination including fluid balance, co-morbidities, blood results, medication.
- Give appropriate analgesia before transfer.
- If there is any doubt of stability refer back to registrar in Walton Centre for further guidance.
- Appropriate medical personnel and equipment should accompany patient.

The aim of specialist management is to identify the size, location and secure the ruptured aneurysm; ongoing early aggressive management; prevent complications; identify and treat complications in an expert and timely manner

- 1.5. After transfer:
 - Reassess airway; breathing; circulation; disability and examination.
 - Systemic examination of patient and timely senior review.

1.6. Early medical management:

See appendices for a quick reference guide for general management of SAH in the Walton Centre.

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- Nimodipine 60mgs 4 hourly for 21 days. In the event of severe hypotensive effect; 30mgs 2 hourly (19). If unable to tolerate orally and no NG tube then IV into a central cannula or a large vein titrated from 2.5mgs to 10mgs hourly depending on weight and blood pressure stability. If running peripherally this should be with a concurrent infusion of normal saline running at minimum of 80mls/hourly in appropriate infusion line. (Walton Centre locally agreed policy)
- Analgesia. (consider paracetamol, codeine with oramorph for breakthrough pain)
- Antiemetics.
- Fluid administration with isotonic saline/ plasmalyte to maintain 3 litre input in 24 hours if no signs of fluid overload, advanced age and known heart failure assessing each case individually. Aim for euvolaemia.

- Blood pressure control. Severe hypertension should be treated, but there is little evidence to guide the criteria to initiate antihypertensive treatment. On the basis of indirect evidence from retrospective data, systolic blood pressure greater than 160 mm Hg might be associated with increased risk of re-bleeding. (13) It is prudent to avoid sudden drops in perfusion pressure because this may result in ischaemia, particularly in patients with increased intracranial pressure and disordered auto regulation.
- General preventive measures e.g., stool softeners to avoid straining.
- Anti-embolism stockings and Sequential compression devices for the legs to minimise the risk of deep vein thrombosis. The safety of subcutaneous low-dose heparin remains untested in aSAH, therefore shouldn't be given before the aneurysm is secured.
- Bed rest with maximum elevation of 15-30 degrees. Bed rest by itself does not abate the risk of re-bleeding. (14, 15) Typically, activity is limited in patients with an unsecured aneurysm. The patient should be maintained in a quiet environment with limited visitors until after aneurysm securement. All activities that increase BP (and, therefore, ICP) are limited to prevent rebleed. (16).
- Order bloods (Full blood count, urea and electrolytes, CRP, blood sugar, clotting screen, liver function, group and save)
- Order CT Angiogram (CTA) once renal function/ eGFR is determined to identify aneurysmal position, morphology and size.
- ECG
- Half hourly neuro observations for first 2 hours and increase to hourly for first 12 hours as condition stabilises. Changes in the frequency of observation should be made by the senior nurse/ medical team. Report any drop in GCS of 2 points or new focal deficit to the medical team as a matter of urgency.
- Request import of foreign CT scans to Walton system.
- Keep patient and family updated.
- 1.7. Securing the aneurysm:
 - Once an aneurysm has been identified, care will be transferred from the accepting consultant to a consultant neurosurgeon in the neurovascular team (if not already done).
 - A multidisciplinary team (MDT) discussion will take place with the parent consultant neurovascular neurosurgeon and the consultant interventional radiologist after review of the patient, CTA, blood distribution on the diagnosing CT scan as well as the characteristics of the ruptured aneurysm. The purpose of this meeting is to consider best management.
 - If an aneurysm is found and amenable to coil embolisation, this will be the treatment of choice (12, 17).
 - Otherwise, the aneurysm will be dealt with by microsurgical clipping.
 - If the medical team agrees the risk of treatment is too high, then discussions will be made with the patient and family to inform them of this course of action. The aneurysm will then be secured at the earliest safest time.
 - The surgical team should always be involved as to the timing of treatment to allow a safe back up option if endovascular treatment fails (12, 18).
 - Following any procedure to secure a ruptured aneurysm, the patient will be transferred to the Critical Care Unit (CCU) to be closely monitored for at least 12 hours. The consultant in charge of the CCU will be informed and agreeable to this decision. If there is no availability for critical care facility, risk assessment will take place in order to provide optimal care for the patient (12). The neurovascular team including the nurse team should be involved in the decision-making process.

- If no aneurysm is found; refer to 1.19.
- 1.8. Critical care management:
 - The minimal monitoring devices for these patients will be ECG, Arterial Blood Pressure and SaO2 as well as ½ hourly observations of neurological status in the first 2 hours; increasing to hourly if the patient's condition allows (1, 2, 12).
 - The use of more specialised or invasive monitoring and complex management of the critically ill neurovascular patient will be managed individually as patient condition determines.
 - The general goals of anaesthetic/ intensive care management are airway and haemodynamic stability; effective monitoring/ observation; stabilisation and treatment for actual/potential neurological deterioration with the aim to rapid management.
 - Triple-H therapy (Hypervolaemia/ hypertension/ haemodilution) **or** hypertensive therapy and restoration/management of euvolaemia will be instituted in the critical care environment (20).
- 1.9. Management of raised intracranial pressure (ICP):
 - Raised intracranial pressure (>20mmHg) significantly worsens other aSAH complications and reduces the chances of a good outcome. Raised ICP in aSAH occurs frequently, and has prolonged and multiple causes (22, 40).
 - The most common causes of raised intracranial pressure (ICP) are hydrocephalus, intracerebral haemorrhage or global cerebral oedema. Less common causes include subdural haematoma, massive cerebral infarct secondary to delayed cerebral ischaemia (DCI) and extracranial causes such as raised intrathoracic pressure from neurogenic pulmonary oedema, central fever, severe hyponatraemia or overcorrection of hypernatraemia (22).

Close monitoring of the patient is key to recognising rising ICP.

- When a patient rapidly deteriorates in the first instance a CT brain should be considered and urgent bloods obtained whilst ruling out other causes (23, 24).
- The goal is to keep ICP<22 and cerebral perfusion pressure 60-70 or higher if vasospasm is suspected (40).
- ICP monitoring in the intubated and ventilated patient should be a consideration for monitoring ICP
- Close monitoring of the patient who is maintaining their own airway is essential
- Serial observation of GCS and limb response should continue being mindful to the fluctuating nature of delayed ischaemic deficit/ vasospasm (23).
- Regular monitoring of fluid balance, electrolytes and osmolalities should continue in the unstable patient (25, 26, 27).
- Methods used to reduce ICP include hyperosmolar agents, hypothermia and barbiturate coma, decompressive craniectomy and CSF drainage. These are specific to the clinical course and investigation finding (1, 2, 28, 34, 40).
- Hypertonic saline is preferable in aSAH patients as mannitol can lead to hypotension and hypovolaemia which is not recommended in aSAH. It is also beneficial in low serum sodium. (28)

1.10. Management in the ward environment post occlusion of aneurysm:

Once the patient is not in need of expert critical care management, they will be moved to the ward. This decision will be made after a discussion has taken place with the neurosurgical team who will then be taking over primary care of the patient. Review of the patient will be done by the receiving team, neurovascular nurse, SMART or ward team before transfer.

Patients will not be transferred to the ward with intra-arterial catheters in situ. (see guidelines for patients following angiographic procedures).

- Once on the ward, close observation will continue. Observations of haemodynamic status as well as neurology will be at 2 hourly for the first 12 hours, increasing to 4 hourly when the medical/nursing team agree that the patient's condition is stable. Close observation of fluid balance will continue for 10 days and assessed daily thereafter.
- Daily blood for urea and electrolytes will be evaluated for the first 7 days post ictus. This will be reduced if there are no signs of symptomatic vasospasm, raised ICP, hydrocephalus, haemodynamic instability and electrolytes are within normal limits.
- The patient will be mobilised as condition allows.
- DVT prophylaxis will be prescribed assuming there are no contra-indications.
- Pain relief/stool softeners will continue regularly as needed.
- Anti-emetics will be administered as needed.
- If coil Embolisation has been performed then the prescribed prophylactic antiplatelet medication and duration will be determined.
- The patient will normally be encouraged to take 3L oral input, although this prescription will be assessed on an individual basis depending on age/size/medical co-morbidities and fluid/electrolyte balance. If the prescribed input cannot be achieved orally; intravenous infusion will be commence (26, 27).
- Urine output and fluid balance charting will be mandatory with the goal of euvolaemia. This will be reviewed daily or after conditional change by the nurse in charge/medical team. There is class 1 evidence to suggest that maintaining euvolaemia and preventing hypovolaemia reduces the incidence of symptomatic vasospasm (1, 2, 27).
- Specialist management in the ward area; e.g. tracheostomy weaning will be led by the neurovascular team and SMART.
- Input from the multidisciplinary team is essential in order to reduce hospital stay, plan for safe return home or ensure timely rehabilitation (12).
- MUST, Waterlow, thromboembolism risk scores will be assessed and appropriate management commenced in a timely manner in order to reduce avoidable complications of illness.
- Any deterioration of the patients neurology that results in a reduction of GCS of 2 points or more or a new/worsened focal neurological deficit will be reported to the patients medical team, neurovascular nurse specialists (______) and/or SMART (_______ immediately.
- Report any change that causes a concern even if it doesn't include the above. Report any fluid balance that results in >500mls negative or positive. Report any significant fall in serum sodium. Report any clinical deterioration or concerns. In the event of being unable to contact the responsible medical team then the on call team should be contacted.

Expert management of the neurovascular patient is dependent on rapid observation and management of subtle or major changes in neurology.

- 1.11. Management of Delayed Cerebral Ischaemia (DCI):
 - Oral Nimodipine should be administered to all patients with confirmed or suspected aneurysmal SAH as it is clinically proven to improve neurological outcome (19).
 - Maintenance of euvolaemia and normal circulating blood volume to prevent DCI (20, 33)
 - Hypertensive therapy with inotropic support is recommended/ individually considered for patients with DCI unless blood pressure is elevated at baseline or cardiac status contraindicates. (20)
 - Transcranial doppler assessment may be considered if appropriately qualified personal are available to complete and interpret
 - Cerebral angioplasty and/or selective intra-arterial vasodilator therapy in patients with symptomatic cerebral vasospasm can be considered after discussion/ agreement with the interventional radiology team and parent surgical team if it is thought the patient may benefit.
 - CT angiogram can be considered to confirm symptomatic vasospasm.
 - Perfusion imaging with CT or MRI can be useful to identify potential brain ischaemia.
 - Most patients have a baseline MRI/A post coil embolisation of ruptured aneurysm if safe to do so. This should be reviewed before they go home or transferred.

Aneurysmal Subarachnoid haemorrhage is a life -threatening disease. Even those who survive the initial bleed and go on to have a ruptured aneurysm secured are at risk of cerebral ischaemia. It occurs in approximately 30% of cases (22, 23), most commonly between days 3 and 14 hence 'Delayed Cerebral Ischaemia' (DCI)). Onset is often subtle and fluctuant with eventual focal neurological deficit such as limb paresis or dysphasia, or a decrease in level of consciousness typically with gradually reducing or fluctuating consciousness (23).

The proposed definition of clinical deterioration caused by DCI as advocated by Vergouwen *et al* 2010 is: "The occurrence of focal neurological impairment (such as hemiparesis, aphasia, apraxia, hemianopia, or neglect), or a decrease of at least 2 points on the Glasgow Coma Scale (either on the total score or on one of its individual components (eye, motor on either side, verbal)). This should last for at least 1 hour, is not apparent immediately after aneurysm occlusion, and cannot be attributed to other causes by means of clinical assessment, CT or MRI scanning of the brain, and appropriate laboratory studies." (31). The diagnosis of DCI can be subjective as no strict criteria can be given.

Firstly, exclude and correct other reversible causes such as infection, hypotension, hyponatremia, hypovolaemia, seizures, hypoxia, heart failure, hypoglycaemia and the effect of sedatives.

Diagnosis of DCI is individual to each situation:

The minimum diagnostic tool should be CT brain to rule out hydrocephalus and/or rebleed although CTA, CT perfusion and transcranial Doppler's (TCDs) can be useful diagnostic adjuncts.

When DCI is diagnosed then hypertensive therapy should be considered and euvolaemia restored.

(No randomised trials have been performed to prove this but the improvement of patients after it is started and deterioration once withdrawn early is proven efficacy). There is now more literature to suggest that Hypertension, and maintainence of euvolaemia is beneficial rather than triple-H therapy. (20)

Management of a patient with DCI should be undertaken in the critical care unit.

1.12. Management of suspected hydrocephalus:

The incidence of acute hydrocephalus in patients with subarachnoid haemorrhage is up to 20-30%. It usually occurs acutely within 48 hours of subarachnoid haemorrhage. More rarely the build-up is chronic after weeks or even months after haemorrhage (24).

- When obstructive hydrocephalus is suspected, a bactiseal external ventricular drainage catheter (EVD) is preferred for emergency deterioration.
- Lumbar drainage or serial lumbar puncture should be assessed and considered by the patient's clinical lead and depends on the clinical scenario (1)
- Chronic hydrocephalus associated with subarachnoid haemorrhage is usually treated with ventricular shunt placement.
- Whilst only a proportion of patients with SAH associated hydrocephalus develop shunt dependent hydrocephalus, chronic symptomatic hydrocephalus should be treated with permanent cerebrospinal fluid diversion.

1.13. Management of Seizures:

Risk factors for the development of seizures include: Middle cerebral artery aneurysm, increased fisher grade (3 and 4), associated haematoma, rebleed, ischaemia, poor WFNS grade, acute hydrocephalus and PMH of hypertension. (1)

The percentage of seizures after subarachnoid haemorrhage without anti -epileptic drug protection ranges from 3-26%; Late or recurrent epilepsy occurred in 7-12% of patients; particularly in those with fisher grade 3 and 4, established (cortical) infarction and hydrocephalus. (38, 39).

High quality evidence for routine long -term anticonvulsant use is lacking (1) but is supported for short term prophylactic use on the premise seizures in the acutely ill patient may lead to further brain injury or rebleed (38, 39).

- A loading dose of Intravenous Levetiracetam or Phenytoin is the agreed first line management of seizure control post SAH. The agreed maintenance anti-epileptic drug should be levetiracetam. Anti-epileptic drugs should be reviewed with the medical team at 6 month follow up as the patient is seizure free, there may be indication to reduce and withdraw taking into account driving restrictions.
- Sedating drugs such as lorazepam and diazemuls are usually avoided in the acute neurosurgical patient as may lead to reduced blood pressure (thus cerebral perfusion pressure and poor airway control).
- SMART should be involved in a timely manner in the event of status epilepticus that doesn't respond to initial management as the patient may need airway management, stabilisation and transfer to the critical care unit.

1.14. Management of hypernatraemia/ hyponatraemia:

Hyper and hyponatraemia are commonly seen in the acute phase after SAH. (25, 26, 27). The reported incidence of hyponatraemia occurs in 10-30%. Hyponatraemia is associated with confirmed and clinical vasospasm. Hyponatraemia can sometimes be seen a day before the onset of clinical vasospasm (27).

Hyponatraemia can develop from the syndrome of cerebral salt wasting due to the excessive release of natruetic peptides, thus excessive natriuresis and resultant risk of volume contraction with a cerebral pathology. It is distinct from the syndrome of inappropriate antidiuretic hormone secretion, which is characterized by inappropriate retention of free water.

Cerebral salt wasting is more common in patients with poor clinical grade, ruptured anterior communicating artery aneurysms and hydrocephalus. It may be a risk factor for poor outcome. Observational studies have shown that volume resuscitation reduces the risk of cerebral ischaemia after aneurysmal subarachnoid haemorrhage (21, 29, 30).

- Daily observation of U+E's is beneficial in the first 7 days after SAH. Thereafter they should be examined on clinical need or if there is a conditional change in the patient (29).
- Euvolaemia should be maintained with a 3 litre input if patient condition allows. Fluid balance charting should be accurate in patients within the first 7 days after SAH. After that review should take place according to clinical need. Total fluid charts should be kept on all patients with SAH (31).
- Increased fluid input (>3L) should only be prescribed by medical team/ neurovascular nurse specialists/SMART and senior nursing team after review and under strict observation of fluid balance and cardiorespiratory system if condition necessitates and no history / presenting features of cardiac disease, pulmonary disease or low BMI.
- In the event of a fall in sodium below 130 mmol/L then urine and serum electrolytes together with osmolalities, thyroid function studies and cortisol should be analysed. Clinical management will take place after medical review of results, fluid balance and patient condition.
- It is reasonable to replace sodium with supplements in SAH patients (1, 30).
- Consider referral to endocrinology if simple measures to improve serum sodium do not help or the serum sodium falls to 125 mmol/L or below(38).
- Fluid restriction in the high risk period of vasospasm (day 3-14) may be detrimental to the patient (28).
- Neurological observation should be close (2-4 hourly) in the patient who is hyponatraemic.
- 1.15. Management of fever:

Fever of central origin is a common medical complication of aneurysmal subarachnoid haemorrhage. It is associated with increased fisher grade, vasospasm and severity of injury. It may represent an inflammatory state induced by blood and its by-products or infection (35). Fever is a predictor of poor outcome and so it is reasonable to instate early investigation of a source of fever and aggressively manage. (1, 35).

1.16. Management of blood glucose:

Variations in blood glucose strongly predicts poor outcome following subarachnoid haemorrhage. (It is likely that hyperglycaemia on admission is a result of catecholamine surge and persistent hyperglycaemia is proven to exacerbate secondary injury and is a predicter of poor outcome (33). It is reasonable to aim for blood glucose between 4.4 and 7.8 in areas that support robust monitoring and control such as ICU. Treatment for hyperglycaemia > 10 should be considered (1, 31, 41, 44).

1.17. Management of Anaemia:

Anaemia with Hb <100 g/l affects approximately 50% of aSAH patients and is associated with poor outcome.

Fall in haemaglobin may be as a result of suppression of erythropoiesis because of an inflammatory response.

Baseline prediction of likelihood to develop anaemia include female and baseline hematocrit<36% and HB <120.

There is little evidence to support transfusion and it hasn't been fully proven that the risks of transfusion may outweigh the benefit. For that reason, management should involve identifying risk and preventing blood loss where able (39).

1.18. Deep Venous Thrombosis (DVT):

DVT prophylaxis will not normally be considered until after the aneurysm is secured. Any decision prior to this time will be decided by the patients' consultant on a risk: benefit ratio.

Studies suggest all patients should use sequential compression devices and compression stockings unless they are contra-indicated (2).

Prophylactic low molecular weight heparin should be considered 12 hours following clipping of aneurysm and immediately after coiling (2).

If a patient has an angiographically proven negative subarachnoid haemorrhage then they should be encouraged to mobilise following the first angiogram and DVT prophylaxis discussed.

1.19. Non-aneurysmal SAH:

In approximately 15% of cases no vascular cause will be found for the SAH. If the CT scan and/or lumbar puncture is positive for SAH local protocol suggests CTA, DSA and review. If DSA is negative for vascular cause for aSAH consultant neurosurgeon decision with MDT discussion will take place as to whether a second angiogram is necessary. A second DSA is advised depending on blood distribution. Decisions should include categorising the patient to perimesencephalic or non-perimesencephalic location. Repeat DSA is usually recommended in cases where there is non-perimesencephalic blood distribution (2).

The continued use of nimodipine will be reviewed following the first DSA.

Depending on the patient presentation and inpatient course plus the amount and distribution of blood on the CT brain, a delayed DSA or MRI/A brain and MRI spinal cord will be considered. This will be consultant or MDT decision.

If a second or delayed angiogram is negative or is deemed not necessary, then the team would normally consider other rarer cause which may involve input and expertise from the vascular neurology team

Management of equivocal lumbar puncture ("SAH cannot be ruled out") will be done on an individual basis by the patients' consultant depending on presenting symptoms and the lumbar puncture result as well as other significant information.

1.20. Going Home:

Clinical indicators to consider before going home are:

- Baseline MRA review being satisfactory (assuming endovascular procedure for occluding aneurysm).
- Patient informed of provisional plan for any incidental aneurysms.
- No clinical symptoms of delayed ischemia/vasospasm
- Suitable pain control including reduced need for break through pain relief such as oramorph.
- Stable electrolytes
- Adequate dietary input.
- Safe mobilisation/ physiotherapy assessment if needed.
- Cognitive function established and Occupational Therapy input to ensure safety at home and/or follow up care in place
- Speech and language assessment if needed
- Vision has been assessed and systems in place to maintain safety/ ophthalmology referral.
- All patients should be instructed on how to manage headaches and aim to stop pain medication for headaches by 4 weeks after going home. If this cannot be done then the GP should consider specialist management to the refractory headache clinic
- Suitable supervision/care in the home environment if needed.
- If appropriate, rehabilitation should be organised following guidelines set by the rehabilitation network.
- The patient/family should be offered information, contact details and been supported, instructed on self-care and recovery from the neurovascular clinical nurse team before going home or repatriated (12).
- 1.21. Referring back to presenting hospital:
 - If the patient is clinically stable but isn't safe or ready to go home or to a rehabilitation unit then repatriation to the referring hospital should be considered for ongoing care.
 - If rehabilitation is needed, a plan should be put into place either by referral to the Cheshire and Merseyside rehabilitation team or a local one that is suitable for rehabilitation needs prior to repatriation: The patient can be repatriated awaiting a rehabilitation place once a referral is in place.

1.22. Follow up:

- Phone call from neurovascular nurse team at approximately 2 weeks after going home
- Neurosurgical follow up will take place 2-3 months after going home/repatriation/transfer to rehabilitation. This will be organised once the patient has left the Walton Centre. Unless otherwise stated the first follow up will be undertaken in the nurse clinic.
- Follow up protocol after coil embolisation procedures to occlude aneurysm includes MRI/A brain at 6, 18, 60 months. This procedure may be changed on instruction of the interventional neuroradiologist after review.
- All patients will be reviewed by a consultant neurosurgeon or radiologist following the 6 months MR scan. After the subsequent MR scans, written confirmation is the planned choice in advising the patient of the results and further follow up.
- Follow up scanning following surgical clipping will be reviewed at the neurovascular MDT if there is no plan in the notes. This may include DSA or MRA.
- Outcome scores (modified Rankin Scale as a minimum) should be completed at 3 months and 6 months and any follow up clinical review

- "Road to Recovery" information courses are available for patients after they go home. These are run in partnership with the brain haemorrhage support group at least one per year.
- Information booklets are available on subarachnoid haemorrhage. These can be obtained from the Walton Centre or downloaded from the website. Information can be translated into other languages as required.
- Self-help, working lives, and support during recovery can be gained from the Brain Charity (<u>www.thebraincharity.org.uk</u>).
- The Brain Haemorrhage Support Group, Liverpool who can be contacted via facebook or the Brain Charity). These are a group of people who have suffered brain haemorrhage and can be a source of support.

2. References

1. CONNOLLY, E., RABENSTEIN, A., CARHUAPOMA, J., DERDEYN, C., DION, J., HIGASHIDA, R., HOH, B., KIRKNESS, C., NAIDECH, A., OGILVY, C., PATEL, A., THOMPSON, B., VESPA, P., AMERICAN HEART ASSOCIATION STROKE COUNCIL, COUNCIL ON CARDIOVASCULAR RADIOLOGY AND INTERVENTION, COUNCIL ON CARDIOVASCULAR NURSING, COUNCIL ON CARDIOVASCULAR SURGERY AND ANESTHESIA, COUNCIL ON CLINICAL CARDIOLOGY., 2012. Guidelines for the management of aneurysmal subarachnoid hemorrhage: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. Stroke, (43 (6), pp 1711-1737.

2. STEINER, T., JUVELA, S., UNTERBERG, A., JUNG, C., FORSTING, M., RINKEL, G., EUROPEAN STROKE ASSOCIATION., 2013, European stroke organisation guidelines for the management of intracranial aneurysms and subarachnoid haemorrhage. Cerebrovascular Diseases. 34, (2), pp 93-112.

3. YAO, Z., HU, X., YOU, C., HE, M., 2017, Timing of surgery for subarachnoid haemorrhage: a systemic review and meta-analysis. International Journal of Surgery, (48), pp 266-274

4. BRODERICK, J., BROTT, T., DULNER, J., LEACH, A., 1994, Initial and recurrent bleeding are the major causes of death following subarachnoid haemorrhage. Stroke, (25) pp 1342-1347

5. INGAL, T., KJELL, A., MARKKU, M., BONITA, R., 2000, A multinational comparison of subarachnoid haemorrhage epidemiology in the WHO Monica stroke study. Stroke, 31(5), pp1054-61.

6. DIRINGER, M, 2009, Management of aneurysmal subarachnoid haemorrhage. Critical Care Medicine, 37(6), pp 2142-3.

7. HUANG, J., VAN GELDER, J., 2002, The probability of sudden death from rupture of intracranial aneurysms: a meta-analysis. Neurosurgery (51)., pp 1101-05.

8. BASSI, P., BADERA, R., LORIERO, M., TOGNUOMI, G., MANGONI, A., Warning signs in subarachnoid haemorrhage: a comparative study. Acta Neurologica Scandanavica. (84), pp277-281

9. DE FALCO, F., 2004, Sentinal headache. Neurological Sciences, pp, S2150217.

10. VAN DER WEE, RINKEL, G., HASAN, D., VAN GIJN, J., 1995. Detection of subarachnoid haemorrhage on early CT: is lumbar puncture still needed after a negative scan? Journal of Neurology, Neurosurgery and Psychiatry, (58), pp357–359.

11. CRUIKSHANK, A., AUID., P., BEETHAM, R., BURROWS, G., EGNER, W., HOLBROOK, I., KEIR, G., LEWIS, E., PATEL, D., WATSON, I., WHITE, P., UK NEQAS SPECIALIST ADVISORY GROUP FOR EXTERNAL QUALITY ASSURANCE OF CSF PROTEINS AND BIOCHEMISTRY, 2008, Revised national guidelines for analysis of CSF for bilirubin in suspected SAH. Annals of Clinical Biochemistry, (45), pp 238-244.

12. NCEPOD, 2013, Managing the flow? A review of the care received by patients who were diagnosed with an aneurysmal subarachnoid haemorrhage [online]. Available at www.ncepod.org.uk [accessed 23 02 2019].

13. OHKUMA, H., TSURUTANI, H., SUZUKI., S., 2001. Incidence and significance of early aneurysmal rebleeding before neurosurgical or neurological management., Stroke, (32), pp 1176-1180.

14. HENDERSON, W., TORNER, J., NIBBELINK, D., 1997, Intracranial aneurysms and subarachnoid hemorrhage: report on a randomized treatment study, IV-B: regulated bed rest: statistical evaluation., Stroke, (8), PP, 579–589.

15. MA, Z., WANG, Q., LIU, M., 2013, Early versus delayed, mobilisation for aneurysmal subarachnoid haemorrhage., Cochrane Database, Systemic Review, (5)., [online]. Available from: www.cochranelibrary.com [Accessed12 February 2019].

16. SUAREZ, J., TARR, R., SELMAN, W., 2006., Aneurysmal subarachnoid haemorrhage. New England Journal of Medicine., (354), pp 387-39.

17. MOLYNEUX, A., KERR, R., YU, L., CLARKE, M., SNEADE, M., YARNOLD, J., SANDERSOCK, P., 2005, International Subarachnoid Aneurysm Trial (ISAT) of neurosurgical clipping versus endovascular coiling in 2143 patients with ruptured intracranial aneurysm: a randomised comparison of effects on survival, dependency, seizures, rebleeding, subgroups, and aneurysm occlusion. Lancet., (366), pp 809-817.

18. KIRKPATRICK P., 2002., Subarachnoid haemorrhage and intracranial aneurysms: What neurologists need to know. Journal of Neurology, Neurosurgery and Psychiatry, (73), pp i28-i33.

19. PICKARD, J., MURRAY, G., ILLINGWORTH, R, SHAW, M., TEADALE, G., FOY, P., HUMPHREY, D, LANG, A., NELSON, R., RICHARDS, P., Effects of oral nimodipine on cerebral infarction and outcome after subarachnoid haemorrhage: British aneurysm nimodipine trial. British Medical Journal, (298), pp 636-642.

20. DANKBAAR, J., SLOOTER, A., RINKEL, G., SCAAF, I, 2010. Effect of different components of triple-H therapy on cerebral perfusion in patients with aneurysmal subarachnoid haemorrhage: a systematic review. Critical Care Medicine, (14), R23.

21. CHANDY, D., SY, R., ARONOW, W., LEE, W., MAGUIRE, G., MURALI, R., 2006, Hyponatraemia and cerebrovascular spasm in aneurysmal subarachnoid hemorrhage. 2006. Neurology India. Sep;54 (3):273-5.

22. ROOS, Y., HAAN, R., BEENEN, L., GROEN, R., ALBRECHT, K., VERMEULEN, M., 2000. Complications and outcome in patients with aneurysmal subarachnoid haemorrhage: a

prospective hospital based cohort study in the Netherlands. Journal of Neurology, Neurosurgery and Psychiatry, (68), 337–341.

23. HIJDRA, A., VAN GIJN, J., STEFANKO, S., VAN DONGEN, K., VERMEULEN, M., VAN CREVEL, H., 1986. Delayed cerebral ischemia after aneurysmal subarachnoid hemorrhage: clinicoanatomic correlations. Neurology, (36), pp 329–333

24. GERMANWALA, A., HUANG, J., TAMARGO, R., 2010, Hydrocephalus after aneurysmal subarachnoid haemorrhage. Neurosurgery Clinics of North America., 21 (2), pp 263-70.

25. JAMES, I., 1972., Electrolyte changes in patients with subarachnoid haemorrhage. Clinical Science., (42), pp79-187.

26. DISNEY, I., WEIR, B., GRACE, M., ROBERTS, P., 1989., Trends in blood pressure, osmolality, and electrolytes after subarachnoid haemorrhage from aneurysms. Canadian Journal of Neurological Sciences, (16), pp299-304.

27. CHANDY, D., SY, R., ARONOW, W., LEE, W., MAGUIRE, G., MURALI, R., 2006, Hyponatraemia and cerebrovascular spasm in aneurysmal subarachnoid hemorrhage. 2006. Neurology India. Sep;54 (3):273-5.

28. AL-RAWI, P., TSENG, M., RICHARDS, H., NORTJE, J., TIMOFEEV, I., MATTA, B., HUTCHINSON, P., KIRKPARICK, P., 2010. Hypertonic saline with poor grade subarachnoid haemorrhage improves cerebral blood flow, brain tissue oxygenation, and pH., Stroke., (41), pp122-128

29. MARAPUDI, N., MITTAL, S., 2015., Diagnosis and management of hyponatremia in patients with aneurysmal subarachnoid haemorrhage., Journal of Clinical Medicine, 4 (4), pp 756-767.

30. RAHMAN, M., FREIDMAN, W., Hyponataemia in neurosurgical patients: Clinical guidelines development. Neurosurgery, (65), pp925-935.

31. VERGOUWEN, M., VERMEULEN, M., VAN GIJN, R., RINKEL, G., WIJDICKS, E., MUIZELAAR, J., MENDELOW, A., JUVELA, S., YONAS, H., TERBRUGGE, K., MACDONALD, R., DIRINGER, M., BRODERICK, J., DREIER, J., ROOS, Y., 2010, Definition of Delayed Cerebral Ischemia after Aneurysmal Subarachnoid Haemorrhage as an Outcome Event in Clinical Trials and Observational Studies. Stroke., pp 2391-2395.

32. BROWN, R., EPLING, B., FORTUNATO, G., GRADY, J., MCCULLOCH, L., 2015. Polyuria and cerebral vasospasm after aneurysmal subarachnoid haemorrhage. BMC Neurology., (15), p201.

33. EGGE, A., WATERLOO, K., SJOHOLM., H., SOLBERG, T., INGEBRIGTESN, T., ROMNER, B., 2001., prophylactic hyperdynamic postoperative fluid therapy after aneurysmal subarachnoid haemorrhage: a clinical prospective, randomised controlled study. Neurosurgery, (49), pp 593-605.

34. LENNIHAN, L., MAYER, S., FINK, M., BECKFORD, A., PAIK, M., ZHANG, H., WU., Y., KLEBANOFF, L., RAPS, E., SOLOMON, R., 2000, Effect of hypervolemic therapy on cerebral blood flow after aneurysmal subarachnoid haemorrhage: a randomised controlled trial. Stroke., (31), pp383-391.

35. DOUDS, L., TADZONG, B., AGARWAL, D., KROSHNAMURTHY, S., LEHMAN, E., COCKCROFT, K., 2012., Influence of fever and hospital acquired infection on the incidence of delayed neurological deficit and poor outcome after aneurysmal subarachnoid haemorrhage., Neurology Research International (2012), pp 1-6.

36. NAVAL, N., STEVENS, R., MIRSKI, M., BHARDWAJ, A., 2006., Controversies in the management of aneurysmal subarachnoid haemorrhage. Critical Care Medicine, (34), pp511-524.

37. HARRIGAN, M., 2001, Cerebral salt wasting syndrome. Critical Care Clinics., (17), pp125-138.

38. KYU-SUN C., HYOUNG-JOON, C., HYEONG-JOONG, Y., YONG, K., YOUNG-SOO, K., JAE-MIN, K., 2009. Seizures and epilepsy following aneurysmal subarachnoid haemorrhage: incidence and risk factors., Journal of Korean Neurosurgical Society. 46 (2), pp 93–98.

39. BUTZKUEVEN, C., EVANS, A., PITMAN, A., LEOPOLD, C., JOLLEY, D., KAYE, A., KILPATRICK, C., DAVIS, S., 2000. Onset seizures independently predict poor outcome after subarachnoid haemorrhage. Neurology. (55), pp1315-1320.

40. ALOTAIBI, N., WANG, J., PASARIKOVSKI, C., GUHA, D., AL-MUFTI, F., MAMDANI, M., SAPOSNIK, G., SCHWEIZER, T., MACDONALD, R., 2017. Management of raised intracranial pressure in aneurysmal subarachnoid haemorrhage: time for a consensus? Neurosurgical Focus. 43 (5), E13 [online]. Available from https://thejns.org/view/journals/neurosurg-focus/43/5/article-pE13.xml. [Accessed 3 December 2018].

41. NAIDECH, A., LEVASSEUR, K., LIEBLING, S., GARG, R., SHAPIRO, M., AUIT, M., AFIFI, S., BATJER, H., 2010., Moderate hypoglycaemia is associated with vasospasm, cebebral infarction and a 3-month disability after subarachnoid haemorrhage. Neurocritical Care., 12 (12), pp181-187.

42. MISTRY, A., MISTRY, E., KUMAR, G., FROEHLER, M., FUSCO, M., CHITALE, R., 2016. Corticosteroids in the management of hyponatraemia, hypovolaemia and vasospasm in subarachnoid haemorrhage: a meta-analysis. Cerebrovascular Diseases., (42), pp 263-271.

43. AYLING, O., IBRAHIM, G., ALOTAIBI, N., GOODERHAM, P., MACDONALD, R., 2013. Anaemia after aneurysmal subarachnoid haemorrhage is associated with poor outcome and death. Stroke., 49(8), pp1859-1865.

44. KRUYT, N., JAN BIESSELS, G., DEVRIES, H., LUITSE, M., VERMEULEN, M., RINKEL, G., VANDERTOP., WP., ROOS, Y., 2010. Hyperglycaemia in aneurysmal subarachnoid haemorrhage: a potentially modifiable risk factor for poor outcome. Journal of Cerebral Blood Flow and Metabolism. 30 (9), pp1577-1587.

2.1. Appendices

Appendix 1 - Scoring systems

The clinical grading system proposed by the World Federation of Neurological Surgeons is intended to be a simple, reliable and clinically valid way to grade a patient with subarachnoid haemorrhage.

World federation of Neurological Surgeons (WFNS) grading system for Subarachnoid Haemorrhage: -

WFNS grade	Glasgow Coma Score	Motor deficit
1	15	Absent
2	13-14	Absent
3	13-14	Present
4	7-12	Absent/Present
5	3-6	Absent/Present

Interpretation:

- Maximum score of 15 has the best prognosis
- Minimum score of 3 has the worst prognosis

Scores of 8 or above have a good chance for recovery

• Scores of 3-5 are potentially fatal, especially if accompanied by fixed pupils or absent oculovestibular responses

In assessing outcome of subarachnoid haemorrhage, the WFNS recommends use of the Glasgow Coma Scale:

Glasgow Coma Scale: -

Eye Opening			
Spontaneous	4		
To speech	3		
To pain	2		
None	1		
Verbal response			
Orientated	5		
Confused	4		
Inappropriate words	3		
Incomprehensible	2		
sounds			
None	1		
Motor response			
Obeys commands	6		
Localises to pain	5		
Flexes to pain	4		
Abnormal flexion to	3		
pain			
Extension to pain	2		
None	1		

Appendix 2 - Quick reference guide for management of acute subarachnoid haemorrhage



Review Date: June 2021 Version: **1.0** Page **18** of **19** Appendix 3: Quick reference guide for management of patients with SAH after transfer to WCNN



Version: **1.0** Page **19** of **19**