Subarachnoid Haemorrhage

1. Purpose of document

To guide management of spontaneous subarachnoid hemorrhage (SAH) in the Department Of Critical Care Medicine (DCCM) within the Auckland District Health Board (ADHB).

2. Responsibility

All medical and nursing staff providing care and treatment for patients admitted to the DCCM with spontaneous SAH.

3. Document management principles and goals

*Spontaneous SAH (*which must be differentiated from *traumatic SAH)* affects people with a mean age of 55years, is associated with high rates of morbidity and mortality and although other aetiologies exist, is most commonly due to arterial aneurysm rupture.

A small magnitude seasonal effect has been described, with higher rates of SAH occurring in winter than summer, and both modifiable (smoking, hypertension, alcohol use, sedentary lifestyle, hyperlipidemia) and non-modifiable risk factors (age, female sex, family history, ADPKD) have been reported[1234].

The main proven interventions following SAH are timely endovascular coiling rather than clipping when feasible and the administration of Nimodipine[56]. Outcome is also probably improved by advanced Neuro-intensive care support and the utilisation of multi-disciplinary teams[7].

Patients admitted to the DCCM following SAH will usually fall into one of two categories; (1) Poor clinical grade SAH (as per World Federation Of Neurosurgeons Grading Scheme - see appendix) requiring ICU specific therapies such as intubation and ventilation or (2) patients suffering complications of SAH, most commonly Delayed Cerebral Ischemia (of which "Vasospasm" is an important component) or less frequently other intra-cranial and extra-cranial complications such as refractory status epilepticus, cardiac arrhythmias or failure, respiratory complications or post-operative complications.

The goals of this document are to provide DCCM team members with information to manage patients admitted following SAH or the complications of SAH, it includes established treatment parameters for patients with secured and un-secured aneurysms and general guidance on investigation and management of SAH and its complications.

Subarachnoid Haemorrhage

4. Inclusion Criteria

All salvageable patients admitted to the DCCM following spontaneous SAH.

5. Exclusion Criteria

Patients may not receive this treatment if:

- The duty Intensivist (usually in consensus with the neurosurgical or neuro-interventional consultant) determines a clinical or other indication for deviation from this document.
- The SAH is un-survivable and the patient has been admitted as a potential organ donor or for palliative care.

6. Process of Treatment

6.1 Initial assessment of the patient with suspected spontaneous SAH

This usually takes place in the emergency department and is performed by the DCCM registrar or Intensivist alongside the ED team and in liaison with the neurosurgical registrar.

The nature of the SAH and other important clinical information will be ascertained and all patients requiring transfer to the DCCM will be discussed with the duty Intensivist.

a. All patients will have a CT brain with arterial phase contrast

Important information to be recorded in the admission note are:

- Location and cause of SAH - if aneurysmal then location of the ruptured aneurysm and if applicable the location and size of any un-ruptured aneurysms.
- Presence of intra-ventricular extension, signs of hydrocephalus, trans-compartmental herniation or significant mass effect.

b. All patients will have a clinical exam performed by the DCCM Registrar or Intensivist

- Record will be made of the GCS, pupillary examination and the character of any focal neurological deficit at presentation as a minimum.

c. All patients will have the requirement for reversal of anticoagulants or coagulopathy considered

- Tranexamic acid is not routinely administered in the setting of SAH.
- Reversal of Warfarin or the DOACs should be undertaken as per ADHB policy or following advice from the on call Haematologist.

Subarachnoid Haemorrhage

- Patients receiving Aspirin, Ticagrelor or Clopidogrel only do not usually require any specific treatment except for cessation of therapy.

d. All patients will be discussed with the Neuro-surgical registrar on call and this discussion including the name of the responsible Neuro-surgical consultant will be documented in the clinical notes

- As a minimum this initial discussion will determine the need for emergent EVD placement and when indicated initiate consensus discussion for patients with un-salvageable SAH.

6.2 Initial Management - All Patients

a. Intubate and ventilate if indicated

b. Initiate standard monitoring including in all patients a CVL, NGT, IDC and arterial line

- Be aware of early SAH related complications – hydrocephalus, cardiac arrhythmias, QT interval prolongation, stress-cardiomyopathy, ARDS, pulmonary aspiration and seizures.

c. Sedation for ventilated patients

- Propofol (e.g. 1-2mg/kg/hr) and an opiate (e.g. Fentanyl 20-50mcg/hr + PRN).
- Neuro-muscular blockade should be used sparingly when indicated as neurological assessment forms an important part of management.

d. Fluid Therapy should be administered to maintain normovolaemia

- There is insufficient evidence to recommend hypervolaemia or haemodilution either prophylactically or therapeutically for vasospasm and this may lead to cardio-respiratory complications.
- Hypovolaemia should be avoided.
- Isotonic crystalloids are the first line therapy, there is insufficient evidence currently to support any theoretical neuro-protective benefits of albumin.

e. Haemoglobin should be maintained above 70g/L

f. Blood pressure Management

Subarachnoid Haemorrhage

- All patients with un-secured aneurysms should have an initial mean arterial pressure target of 80-100mmHg. There is insufficient evidence to support prophylactic blood pressure augmentation for the prevention of clinically significant vasospasm and therefore the lower MAP target may be individualised by the duty Intensivist.
- Patients with a sustained systolic blood pressure exceeding 160mmHg should be assessed for adequacy of analgesia and sedation. If hypertension persists the duty Intensivist should be notified.
- Hypertension should be treated acutely with Hydralazine (10-20mg IV PRN) or Labetalol (10-20mg IV PRN) in the absence of contraindications.
- Hypotension should be treated with Metaraminol or Noradrenaline initially and investigation should be undertaken to determine the aetiology of hypotension. Inotropic therapy may be needed in the setting of cardiac failure.

g. Standard Treatment targets (see also table below)

- Normocarbia (pCO2 4.6-6.0 Kpa).
- Normothermia (36-38^{5} degrees Celsius).
- Normoglycemia – as per DCCM hyperglycemia protocol.
- Normoxia (Oxygen saturations on pulse oximetry of 92-96%).
- Normonatremia (Serum Sodium 135-145mmol/L).

h. Treatment of Hydrocephalus

Hydrocephalus following SAH can be rapidly fatal and emergent insertion of an EVD can be life-saving.

- All SAH patients with a poor clinical grade (WFNS 3-5), intra-ventricular extension (particularly involving the 4th ventricle), signs of hydrocephalus on CT or unexpected neurological deterioration should be discussed with the neurosurgical registrar for consideration of EVD placement.

i. Treatment of raised ICP or Trans-compartmental Herniation

Raised ICP occurs in a significant percentage of patients with SAH and is increasingly likely with worsening clinical grade. Guidelines for SAH have generally not addressed management of raised ICP since there are few studies specific to this population. Routine ICP monitoring or ICP/CPP directed therapies for SAH patients are not currently undertaken in DCCM but some important caveats that mandate discussion with the duty Intensivist are:

Subarachnoid Haemorrhage

- Clinical signs of Trans-compartmental brain herniation (e.g. Pupillary dilation, Cushing's reflex).
- Large volume intra-cerebral hemorrhage with significant mass effect.
- It is reasonable to initiate standard ICP directed medical therapy while awaiting re-imaging or clinical decision making in these circumstances.

6.3 ICU Specific Management of SAH with an Unsecured Aneurysm

Management of patients with an Un-secured Aneurysm will continue as per section **6.2 Initial Management - All Patients** with additional management as per section **6.3 ICU Specific Management of SAH with an Unsecured Aneurysm.**

Section 6.3 applies to all patients prior to aneurysm treatment, patients for whom aneurysm treatment has not fully excluded the aneurysm from the circulation and for patients with additional significant untreated aneurysms. The main priorities during this period are prevention of re-bleed, early identification of complications and prognostication to determine utility of aneurysm treatment.

Additional ICU specific management consists of:

a. Sedation and analgesia

Regular sedative-free neurological assessment of the SAH patient is essential to identify complications such as re-bleed, seizures or the development of Delayed Cerebral Ischemia and for prognostication to identify patients who will or conversely will not benefit from aneurysm treatment. This is performed regularly by the bedside nurse, and as a minimum, by the Intensivist on each ward round.

- Sedation and analgesia will be provided with propofol and short-acting opiates titrated to RASS score documented by the Intensivist on the treatment chart.

b. GCS and pupillary size and response to light will be recorded hourly by nursing staff

- Change in pupillary symmetry, deterioration in GCS or evidence of a new focal neurological deficit will be notified to the ICU registrar urgently.

c. EVD Management

- The EVD is usually positioned at 15cm above the EAM unless

documented otherwise.

- The bedside nurse will notify the registrar if there is no drainage from the EVD or new blood evident in the CSF.
- Remedy of a blocked EVD and removal of the EVD is the responsibility of the Neuro-surgical service.

d. Securing the Aneurysm

The method of aneurysm repair is determined by the multi-disciplinary team taking into account patient characteristics and the anatomy of the aneurysm.

- The DCCM medical team and the Neurosurgical consultant will determine patient suitability for aneurysm treatment.
- The goal is to secure all salvageable patients within 24hrs of presentation.
- Timing of intervention is determined via consensus of the DCCM medical team, the neurosurgical team and the neuro-interventional radiologist.
- The method of aneurysm repair (clip or coil) will be directed by the interdisciplinary neuro-vascular team.
- If a flow-diverting stent is a possible treatment modality the implications of dual-antiplatelet therapy must be considered prior to placement – e.g. timing of EVD placement.
- The requirement for anti-coagulation following any interventional radiology procedure will be determined and directed by the interventional radiologist.

e. Nimodipine therapy

- All patients without contraindications will receive Nimodipine 30mg Q2H (unless Noradrenaline is requirement >2mg/hr) via NG within 96hrs of SAH ictus for a duration of 21 days.
- Dose interval and dose may be modified by the duty Intensivist in the event of hemodynamic intolerance.
- General guidance is to tolerate a maximum Noradrenaline requirement of up to 2mg/hr prior to Nimodipine dose modification or reduction (e.g. 15mg Q2H) or in the event of absolute intolerance, cessation. This policy acknowledges that there is insufficient evidence to provide definitive guidance and therapy should be individualised to the clinical situation.
- The utility of Nimodipine in established clinically significant vasospasm is uncertain and it is reasonable to cease therapy at a lower threshold particularly if therapeutic blood pressure

Subarachnoid Haemorrhage

augmentation is used for these patients.

f. Seizure Prophylaxis

- In the absence of seizures, routine seizure prophylaxis is not administered in the DCCM.

g. Enteral nutrition, glucose management and stress ulcer prophylaxis

- As per DCCM policy.
- 2kCal enteral nutrition (with psyllium 5ml NG Q8H) may be required to maintain normonatremia.

h. DVT prophylaxis

- All patients should receive pneumatic compression stockings.
- Timing of the initiation of chemo-prophylaxis should be discussed with the neurosurgical team but in general can be started immediately after coiling and 24hrs following clipping.
- Heparin is the preferred agent.

I. Volume status

- Volume status will be assessed clinically. Non-invasive, semi-invasive or invasive forms of volume status assessment or cardiac output monitoring are not routinely used for SAH patients in the DCCM.

j. Fever

- Initial targets are 36-38[5] degrees Celsius.
- Fever may be associated with deterioration in neurological status, following exclusion of other causes of neurological deterioration it is reasonable to initiate cooling measures to control fever.
- The aetiology of fever should be investigated in all cases including a sample of CSF in all patients with an EVD - the most useful markers of infection from EVD samples are the glucose concentration, a WCC:RBC >500:1, gram stain and culture.

k. ICP monitoring

- ICP monitoring is not currently routinely undertaken in patients admitted to the DCCM following SAH.

l. Other therapies

- There is insufficient evidence to recommend initiation of statins, magnesium therapy or other pharmacological agents at this time.

- Patients already on a statin should have this continued.

6.4 ICU Specific Management of SAH patients with a Secured Aneurysm

Management of patients with a secured aneurysm will continue as per section **6.2 Initial Management - All Patients** and section **6.3 ICU Specific Management of SAH with an Unsecured Aneurysm** except that given the significant reduction in the risk of re-bleed blood pressure management will be modified.

a. Blood pressure Management

- Immediately following exclusion of the aneurysm (either by clip or coil) the MAP should be maintained at 90-120mmHg until the patient is neurologically assessable, following which, in the absence of neurological deterioration, the lower MAP target may be reduced to 70mmHg.
- There is insufficient evidence to support prophylactic blood pressure augmentation for the prevention of clinically significant vasospasm and therefore the lower MAP target may be individualised by the duty Intensivist.
- Hypertension should be treated acutely with Hydralazine (10-20mg IV PRN) or Labetalol (10-20mg IV PRN) in the absence of contraindications.
- NG or PO anti-hypertensive therapy should be initiated for long-term control of hypertension if not done so already. The agent chosen should take into account co-morbidities and allergies.
- Treatment of hypotension should be undertaken with Metaraminol or Noradrenaline initially and investigation should be undertaken to determine the aetiology of hypotension. Inotropic therapy may be needed in the setting of cardiac failure.

6.5 Investigation of unanticipated neurological deterioration and management of delayed cerebral ischemia

Neurological deterioration following SAH is a neurological emergency and requires urgent investigation and treatment of the underlying cause. A suggested approach is described below.

a. Confirm and characterise neurological deterioration by clinical examination

b. Consider a broad differential diagnosis including but not limited to:

- Re-bleed (more likely in untreated aneurysmal SAH),

Subarachnoid Haemorrhage

hydrocephalus, DCI (usually day 3-14 post-ictus), hypoglycaemia, sedatives/analgesics, seizures, hyponatremia and infection.

c. Inform duty Intensivist and initiate investigation

- Ensure the EVD is patent and draining.
- Attain a CT brain with CT cerebral angiogram to assess for radiographic vasospasm, hydrocephalus or re-bleed.
- Consider seizures and EEG monitoring or empiric treatment.

d. Management of Delayed Cerebral Ischemia and Vasospasm

An important feature of SAH is a secondary phase that occurs during days 3-14 following ictus called delayed cerebral ischemia. This is a neurological emergency and early rapid treatment may prevent progression to irreversible cerebral infarction.

DCI is a complex multi-factorial process of which angiographic vasospasm is an important correlate, but the relationship is complex; 70% of patients with SAH develop angiographic vasospasm but only 30% of those will develop clinical symptoms or cerebral ischemia – only patients with symptomatic vasospasm/DCI require therapy.

DCI has been defined by expert consensus as:

- A new focal neurological deficit (such as hemiparesis, dysphasia, apraxia) or deterioration in GCS by 2 or more points.
- That lasts for at least 1 hour.
- Is not apparent immediately following aneurysm treatment (i.e. not a consequence of aneurysm treatment).
- Cannot be attributed to an alternative cause (e.g. seizure, hyponatraemia, hydrocephalus).

Management of DCI in the DCCM should proceed rapidly in a step-wise fashion in consultation with the duty Intensivist:

1. Ensure Normovolaemia

2. Provided the aneurysm is secure and the risk of re-bleed is low initiate blood pressure augmentation with Noradrenaline;

- Increase the MAP target in increments of 10mmHg every 30mins until clinical response or a maximum MAP of 120-140mmHg as determined by the duty Intensivist.
- The utility of Nimodipine in established clinically significant DCI/vasospasm is uncertain and it is reasonable to cease therapy at a lower threshold if MAP augmentation with vasopressors is used for treatment.

Subarachnoid Haemorrhage

3. If there is no neurological improvement after 30mins of blood pressure augmentation to maximum MAP target then discuss further treatment with the Interventional Neuro-radiologist on call

- Consideration can be given to intra-arterial treatment with a vasodilator (e.g. Verapamil) or occasionally mechanical balloon angioplasty.
- The level 8 anesthetics coordinator should be contacted to arrange peri-procedural Anaesthetic support.

Subarachnoid Haemorrhage

	Unsecured	Secured
MAP (mmHg)	80-100	90-120 until neuro-assessment then 70-120*
Oxygen	Saturations 92-96%	
CO2 (kPa)	4.6 - 6.0 Kpa	
Temp (degrees Celsius)	36 - 38[5] degrees	
Patient Position	2hrs left, 2hrs sitting, 2hrs right, 4hrs flat, 4hrs head-up	
Sodium (mmol/L)	135-145	
Glucose (mmol/L)	4.0-10.0 - as per DCCM protocol	
Enteral nutrition	As per DCCM Protocol - 2.0 kcal/ml (with 5ml Q8H Psyllium) may be required to avoid hyponatremia	
EVD	15cm above external auditory meatus or as per Intensivist	
Sedation	Propofol +/- opiate	
Nimodopine	Standard full dose is 30mg Q2H for 21 days	
DVT Prophylaxis	In discussion with neuro-surgery - Generally 24hours following clipping and immediately following coiling	
Neurological deterioration should be treated as an emergency in accordance with section 6.5 and the duty Intensivist notified		
* Immediately following exclusion of the aneurysm (either by clip or coil) the MAP should be maintained at 90-120mmHg until the patient is neurologically assessable, following which, in the absence of neurological deterioration, the lower MAP target may be reduced to 70mmHg		

Subarachnoid Haemorrhage

Acute Neurological Deterioration Algorithm

Confirm and characterise neurological deterioration by clinical examination

Consider a broad differential diagnosis including but not limited to:

- Re-bleed (more likely in untreated aneurysmal SAH), hydrocephalus, DCI (usually day 3-14 post-ictus), hypoglycaemia, sedatives/analgesics, seizures, hyponatraemia and infection

Inform duty Intensivist and initiate investigation

- Ensure the EVD is patent and draining
- Attain a CT brain with CT cerebral angiogram to assess for radiographic vasospasm, hydrocephalus or re-bleed
- Consider seizures and EEG monitoring or empiric treatment

If DCI (see criteria in section 6.5) is the likely cause, initiate emergent therapy in a step-wise fashion

1. Ensure Normovolaemia

2. Provided the aneurysm is secure and the risk of re-bleed is low initiate blood pressure augmentation with Noradrenaline;

- Increase the MAP target in increments of 10mmHg every 30mins until clinical response or a maximum MAP of 120-140mmHg as determined by duty Intensivist

3. If there is no neurological improvement after 30mins at maximum MAP target then discuss treatment with the Interventional Neuro-radiologist on call for consideration of intra-arterial vasodilator therapy

Subarachnoid Haemorrhage

7. Supporting Evidence

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8. Disclaimer

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