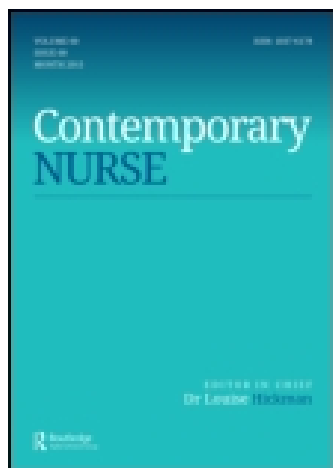


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### Aneurysmal subarachnoid haemorrhage - Part 1: Pre-operative nursing management diagnosis, complications and treatment: A composite case study

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# Aneurysmal subarachnoid haemorrhage – Part 1: Pre-operative nursing management diagnosis, complications and treatment: A composite case study

## ABSTRACT

### KEY WORDS

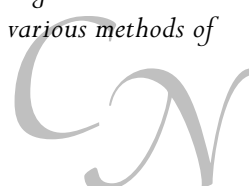
aneurysmal subarachnoid haemorrhage; nursing management; rebleed; CT scan; Glasgow coma scale; intra-arterial digital subtraction angiography; cerebral vasospasm; nimodipine; hydrocephalus; cerebral oedema; seizures; pre-operative care

*Patients who survive an Aneurysmal Subarachnoid Haemorrhage or ASAH describe it as being the worst headache ever, multiplied one hundred-times over. It is a debilitating and life threatening condition, which affects approximately 6.5 people per 100,000 throughout Australia and New Zealand every year (The ACROSS group, 2000). When caring for a patient post Aneurysmal Subarachnoid Haemorrhage or ASAH meticulous monitoring of the patient's neurological, cardiovascular, hepatic, endocrine, renal, and respiratory functions are vital. Due to both the initial ASAH and its complications such as rebleed, cerebral vasospasm, hydrocephalus, cerebral oedema, seizures as well as adverse reactions to counteract these potential problems. All of, which can cause significant long-term morbidity as well as potential mortality if left, undiagnosed and untreated.*

*The following article presets a composite patient highlighting clinical manifestations of ASAH, its associated complications as well as various methods of detecting, preventing and treating them.*

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## INTRODUCTION

Patients who survive an Aneurysmal Subarachnoid Haemorrhage or ASAH describe it as being the worst headache ever, multiplied one hundred-times over. It is a debilitating and life threatening condition, which affects approximately 6.5 people per 100,000 throughout Australia and New Zealand every year (The ACROSS group, 2000). When caring for a patient post Aneurysmal Subarachnoid Haemorrhage or ASAH meticulous monitoring of the patient's neurological, cardiovascular, hepatic,

endocrine, renal, and respiratory functions are vital. Due to both the initial ASAH and its complications such as rebleed, cerebral vasospasm, hydrocephalus, cerebral oedema, seizures as well as adverse reactions to counteract these potential problems. All of, which can cause significant long-term morbidity as well as potential mortality if left, undiagnosed and untreated.

The following article presets a composite patient highlighting clinical manifestations of ASAH, its associated complications as well as various methods of detecting, preventing and treating them.

## INITIAL PRESENTATION OF MRS X

Mrs. X is a 55-year-old lady who presented to a major metropolitan hospital with symptoms of headache, photophobia, nausea and vomiting. Prior to the commencement of her headache she managed a small business. Her only relevant past history is of hypertension and she is a current smoker of 25 years.

On arrival to hospital Mrs X's was neurologically assessed using the Glasgow Coma Scale or GCS (originally graded and still used in some centres as out of a possible 14, is now updated and is graded out of 15, see Table 1a–c). She was eye opening when requested, orientated to time, place and person, obeying commands and

she could move all limbs with full and equal strength. This initial assessment resulted in a score of 14/15 that would be used subsequently to grade the severity of her SAH, and thus the prognosis and potential for complications.

Care for any patient suffering an acute neurological insult requires frequent neurological observations. The Glasgow Coma Scale is a standardised assessment tool of a patient's neurological function and progress (Hickey et al., 1997). This assessment data can guide the clinician to the appropriate treatment, whilst minimising clinician variability. This scale consists of various tasks and questions, which assesses a patient's consciousness, cognitive and motor skills (see Table 1a–c). The tasks and questions are graded from a possible 1 to 6, from the lowest to the highest level of function required to perform each task. Score relevance is shown in Table 2.

## SIGNS AND SYMPTOMS OF A SUBARACHNOID HAEMORRHAGE

A patient with an aneurysmal subarachnoid haemorrhage can exhibit a wide range of symptoms. These symptoms can include a sudden severe onset of headache, photophobia, neck pain, back pain, nausea and vomiting, seizures and or decreased conscious state (Lindsay, Bone

TABLE 1A GLASGOW COMA SCALE (CROWE ET AL., 1997)

### Best Eye opening Response

Spontaneously	4	Patient is observed from a distance before approaching the bedside. If the patient's eyes are open then they are eye opening spontaneously.
To speech	3	If the patient did not open their eyes spontaneously after approaching the bedside the patient's name is spoken and requested to open their eyes without any physical contact. If the patient opens their eyes then they are eye opening to speech.
To Pain	2	If verbal stimulation does not elicit eye opening the patient is given a central painful stimuli, specifically a sternal rub. If the patient opens their eyes then they are eye opening to pain.
No response	1	If the patient does not open their eyes after the above verbal and painful stimuli then the patient is non-eye opening.

If for some reason the eye is unable to be opened an attempt should be made by the nurse to open the patient's eye. If this is unsuccessful then a "C" for closed is documented

**TABLE 1B GLASGOW COMA SCALE (CROWE ET AL., 1997)**

**Best Verbal response**

Orientated	5	The patient is asked the following questions to determine orientation to place, time and person. The wording must be precisely the same each time to ensure objectivity; "Can you tell me where you are?" "What year is it?" "What month is it?" "What day of the week is it?" and "What is your name?" If the patient answers all of these questions correctly, i.e. they know that they are at the Institutes name and the correct year, month and day and they know their own name then they are orientated.
Conversation-confused	4	If the patient answers one or more of the above questions incorrectly, but their answer is relevant to the question. For example, if the patient is asked what year it is and they answer '1864', then the patient is confused. If the patient is orientated to some aspects this should be documented in the nursing notes. It is important to re-orientate the patient if they are confused.
Speech – inappropriate	3	If the patient answers one or more of the above questions incorrectly and their answer is irrelevant to the question. For example, if the patient is asked what year it is and they answer 'banana' then the patient is inappropriate.
Sounds – incomprehensible	2	If the patient answers one or more of the above questions with incomprehensible sounds, such as babbling, mumbling or groaning.
No response	1	If the patient does not make a sound and no attempt is made to answer the question then no verbal response is documented.

- If the patient has a tracheostomy or an endotracheal and is therefore unable to respond verbally, then put a "T" in the square marked no verbal response.
- If the patient is dysphasic the nurse should give the patient three options for each category in order to determine their neurological status. This should be documented in the nursing notes.
- If the patient does not speak or understand English then either an on-site or telephone interpreter should be used.

& Callander, 1997). These symptoms relate to blood in and around the subarachnoid space causing cerebral irritation.

**WHAT IS AN ANEURYSMAL SUBARACHNOID HAEMORRHAGE?**

A Subarachnoid Haemorrhage or SAH is any bleed underneath the arachnoid meninges of the brain. SAH is commonly related to a ruptured cerebral aneurysm (Aneurysmal Subarachnoid Haemorrhage), but can also occur due to trauma, stroke, and arterio-venous malformation or from no identifiable cause (Armstrong, 1994).

An intracranial aneurysm can be described as any weakening of the cerebral arteries. Although some congenital links can be made it is usually very rare to have a positive familial history of

two or more close relatives. Genetic disorders such as Ethlers-Danlos Type IV, Marfan's Syndrome, Neurofibromatosis Type 1, and Autosomal Dominant Polycystic kidney disease can however cause cerebral aneurysms (Pfohman & Criddle, 2001). Cerebral aneurysms are usually described as berry aneurysm, and as the name suggests they are berry like in appearance. Another form of cerebral aneurysm is the fusiform aneurysm. This type of aneurysm is described as a generalised widening of the blood vessel in a localised area. Cerebral aneurysms can be located anywhere throughout the cerebral blood supply. However, junction or branching points are more common and some sites have a much higher incidence of aneurysm than others.

The severity of SAH can be graded using two

**TABLE 1C GLASGOW COMA SCALE (CROWE ET AL., 1997)**

**Best Motor**

This section is primarily concerned with the patient's upper limbs. If the patient responds to the following with both sides then a *dot* is placed in the appropriate column. If the patient only obeys with one side, then it is documented as "L" or "R".

Obeys verbal command	6	Holding both hands out in front of the patient with two fingers extended on each hand the nurse asks the patient "Can you squeeze both of my hands as hard as you can?" It is vital that the assessor does not place their hands in the patient's hands as this tests a reflex. If the patient is for some reason unable to see the nurse's hands, then touching the top of their hands to let the patient know where their fingers are is acceptable. If the patient can reach to squeeze the nurse's hands then they are obeying commands.
Localise to pain	5	If the patient does not obey commands then the nurse proceeds to give the patient central painful stimuli (sternal rub). If the patient's arms move towards the painful stimuli in an attempt to remove the stimuli then the patient is localising to pain. The nurse ensures both arms are tested if there is a different response.
Non-purposal movement	4	Patient is moving all limbs spontaneously but without direction or response to stimuli.
Flexion – abnormal	3	When the patient neither obeys nor localises to central painful stimuli the nurse gives the patient a peripheral painful stimuli (nail-bed pressure). If there is limb flexion at the elbow, abduction of the upper arm and extension of the wrist, then the patient is flexing to pain.
Extension – abnormal	2	When the painful peripheral stimuli is applied, if the patient's limb straightens at the elbow with adduction and internal rotation of the limb, then the patient is extending to pain.
No response	1	If there is no response from central and peripheral painful stimuli then the patient is non-responsive.

**TABLE 2 GLASGOW COMA SCALE (HICKEY ET AL., 1997)**

Glasgow Coma Score	Associated level of Brain Injury
13–14	Mild Brain Injury
9–12	Moderate Brain Injury
3–8	Severe Brain Injury

types of scale. The Hunt and Hess Scale (Table 3) (Cavanaugh & Gordon, 2002) are used by some Neurosurgeons but more commonly the World Federation of Neurological Surgeons haemorrhage scale (Cavanaugh & Gordon, 2002) is used (Table 4). These scales determine the severity of the bleed based on the presenting signs and symptoms of a patient. The higher the score, the greater the bleed and therefore the poorer the prognosis. The results of these

assessments are used to determine the treatment of the patient post SAH.

Mrs. X was tentatively diagnosed as having a possible grade 2 SAH (using the WFNS scale) and sent for further tests.

**DIAGNOSIS OF A ANEURYSMAL SUBARACHNOID HAEMORRHAGE**

A diagnosis of ASAH cannot be confirmed without performing various tests, as there are

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**TABLE 3 HUNT & HESS SCALE OF SUBARACHNOID HAEMORRHAGE (CAVANAUGH & GORDON, 2002)**

GRADE	SYMPTOMS
I	Asymptomatic or minimal headache and slight nuchal rigidity.
II	Moderate to severe headache, nuchal rigidity, and no neurological deficit other than cranial nerve palsy.
III	Drowsy, confused, mild focal deficit.
IV	Stupor, moderate to severe hemiparesis, possible early decerebrate rigidity and vegetative disturbances.
V	Deep coma, decerebrate rigidity, moribund appearance.

**TABLE 4 WORLD FEDERATION OF NEUROLOGICAL SURGEONS: HEMORRHAGE GRADING SCALE (CAVANAUGH & GORDON, 2002)**

**Haemorrhage Grading Scale**

Grade	Glasgow Coma Scale	Motor Deficit
I	15	Absent
II	14–13	Absent
III	14–13	Present
IV	12–7	Present or Absent
V	6–3	Present or Absent

many possible conditions with symptoms similar to that displayed by Mrs X. The first test ordered for Mrs. X was CT scan of her brain.

Computed Tomography (CT) scan is used to diagnose SAH. A CT scan can identify the extent of the subarachnoid haemorrhage and can sometimes narrow down the location of the bleed as well as identify any other related complications such as hydrocephalus (Hickey et al., 1997). A limitation of CT scan is that it cannot visualise cerebral blood flow, which is necessary to isolate where the origin of the bleed is and thus possible treatment options.

Another diagnostic test that maybe used to diagnose SAH is Lumbar Puncture. Up until 20 years ago the use of Lumbar Puncture was the only effective technique that could determine a subarachnoid haemorrhage (Edlow & Caplan, 2000). Although not often used as a diagnostic technique anymore, it is often performed if patient’s symptoms suggest subarachnoid haemorrhage and conventional scanning techniques are inconclusive (Edlow & Caplan, 2000). If a lumbar puncture were to be performed, SAH

would be confirmed if Cerebro-Spinal Fluid or CSF results showed Xanthochromia and or Erythrocytes present. Xanthochromia in the CSF is caused by haemoglobin being metabolised into oxyhaemoglobin and bilirubin (Edlow & Caplan, 2000). Erythrocytes in the CSF are present as they disseminate into the subarachnoid space after a SAH (Edlow & Caplan, 2000). Its pitfall however includes traumatic taps, which may lead to misinterpretation of the results, due to the presence of blood.

Mrs. X’s CT scan showed the presence of subarachnoid blood. As the exact cause was unknown further tests were required. The procedures used are those that can visualise the cerebral blood flow to diagnose or rule out cerebral aneurysm. Mrs X was sent to radiology to have an Intra-Arterial Digital Subtraction Angiography.

Intra-Arterial Digital Subtraction Angiography (IADSA) or Cerebral Angiography is the most commonly used radiological procedure to locate the cause of an ASAH. It is an invasive radiological procedure that allows precise visu-

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alisation of all intracranial vessels and can clearly demonstrate any cerebro-vascular abnormalities including multiple aneurysms, arterio-venous malformations and cerebral vasospasm (Kopitnik & Samson, 1993). Performed under fluoroscopy using local anaesthetic, a catheter is inserted into the femoral artery, passing up into the cerebral arteries, where a radiopaque dye is injected. Simultaneously a series of x-rays are taken and any abnormality can be identified (Allen et al., 1983).

Other techniques such as Magnetic Resonance Angiography or MRA can be used as a non-invasive technique to visualise cerebral vessels. Unfortunately MRA cannot be used in the acute phase of a subarachnoid haemorrhage (up to 48 hours). This is due to resolution limitations caused by a combination of acute blood and turbulent blood flow, creating background artefact (MacDonald, 1989). CT angiography or CTA is another non-invasive procedure that visualises cerebral blood flow, abnormalities or obstructions but it is thought to be only 90% accurate (White et al., 2001). But is not as accurate as IADSA.

Mrs. X's IADSA showed an Anterior Communicating Artery Aneurysm or ACommAA. The Neurosurgical team were consulted and it was decided that surgical intervention was required to treat this condition.

## PREOPERATIVE COMPLICATIONS

Complications post ASAH can have devastating effects on a patient's condition, therefore particular care and monitoring for signs and symptoms must be performed. Preoperatively, it is not unusual to have a patient on hourly or even half-hourly neurological and cardiovascular assessment. This is done in order to detect any deterioration in a patient's condition related to potential complications such as rebleed, hydrocephalus, cerebral vasospasm and seizures. This allows treatment to be initiated to counteract these negative effects quickly, which can prevent any further deterioration.

Prior to surgical intervention Mrs. X had hourly neurological and cardiovascular assessment. She was commenced on intravenous hydration and Nimodipine. Environmental stimulus was kept to a minimum and she was prepared for immediate surgery to prevent possible complication preoperatively.

## REBLEED

Until the damaged cerebral vessel is repaired there is always a risk of rebleed. There are a number of ways an aneurysmal subarachnoid haemorrhage can rebleed, including an increase in blood pressure or intra-cranial pressure or for an unknown reason. Rebleed commonly occurs within 24 hours of the first bleed, and if left untreated the risk of further morbidity or even death increases substantially and sequentially over time (Edlow & Caplan, 2000). Rebleed can be confirmed with a CT scan, which will show more blood in the ventricles (Rusy, 1996).

## Nursing management (rebleed)

Signs and symptoms of a rebleed include severe headache, decreased conscious state, new neurological deficits, nausea and vomiting.

Frequent neurological observations (as per individualised protocol) are performed to closely monitor any changes in a patient's neurological status and to provide a baseline to gauge any improvement or deterioration of the patient's condition as well as to plan for appropriate treatment.

Environmental stimulus should be kept to decrease possible exacerbating factors, such as which may lead to a rise in blood pressure and thus rebleed. This may include dark room, no radio or television, restricted visitors and strict bed rest.

Bowel care such as stool softeners are used to decrease straining which may cause an increase in intra-thoracic pressure and thus increased ICP.

Blood pressure is closely monitored and

active intervention is used to prevent hypertensive episodes that may put increased pressure on an already damaged blood vessel (usually between 120–140 systolic).

## HYDROCEPHALUS

Hydrocephalus can occur through the blockage or obstruction in the reabsorption of CSF (communicating) or flow of CSF (non-communicating). This obstruction can occur because the arachnoid villi (responsible for CSF reabsorption) or CSF pathways are blocked with blood either from the initial bleed, associated surgery or further complications. This causes CSF to build up and the ventricles therefore become dilated (Kopitnik & Samson, 1993). A diagnostic CT scan would identify dilated ventricles associated with the build up of CSF (MacDonald, 1989).

### Nursing management (hydrocephalus)

Hydrocephalus causes a rise in intracranial pressure (ICP) that can result in nausea, vomiting, a decreased conscious state, ataxia, widened gait, severe headache and alteration in neurological observations.

Frequent neurological assessment should allow for early detection of deterioration in a patient's neurological state, which may indicate hydrocephalus.

## SEIZURES

Isolated seizures are more common in-patients post ASAH than the development epilepsy, but the patient can become an epileptic if the initial bleed or complications causes irreversible damage. As seizures are uncommon post ASAH, anti-convulsant cover is not routinely commenced (but may be routine at some centres) until there is evidence of seizure activity (Rees et al., 2002). Depending on the type of anti-convulsant prescribed complications can range from sedation to toxicity. Due to the frequent

nature of neurological assessment seizure activity should be diagnosed quickly.

## CEREBRAL VASOSPASM

Cerebral vasospasm is the most significant cause of morbidity and mortality in patients surviving an aneurysmal subarachnoid haemorrhage (Edlow & Caplan, 2000). As such the majority of care and treatment for a patient sustaining an ASAH is focused on preventing this from occurring. It is caused by unidentified vasogenic substances, which are released after a subarachnoid haemorrhage. This causes structural changes to the vessel walls leading to narrowing of the cerebral vessel lumens, decreasing blood supply to parts of the brain (Armstrong, 1994). Various theories have been put forward for the aetiology of cerebral vasospasm, but its exact cause remains unknown. It can however be related to the presence of blood in the subarachnoid space coming in contact with surrounding blood vessels. The greater the amount of blood in the vicinity of the cerebral blood vessels the higher the incidence of cerebral vasospasm (Ingawa et al., 1995). Cerebral vasospasm usually occurs between four and fourteen days post subarachnoid haemorrhage, peaking around day seven, although it can occur much earlier (day one) or later (day twenty eight).

### Nursing management (cerebral vasospasm)

Signs of Cerebral Vasospasm can include headache, inappropriate behaviour, vagueness, seizures, a possible alteration in conscious state and changes to neurological assessment. Commonly signs and symptoms of cerebral vasospasm relate to the particular cerebral artery involved and thus the area of the brain and the specialised centres it supplies (Edlow & Caplan, 2000). Neurological observations pre operatively as stated before are done at least hourly before surgery to detect and treat any complication as early as possible.

Diagnosis of cerebral vasospasm is routinely



done by Transcranial Doppler, but as cerebral vasospasm does not peak until day seven these are routinely performed post operatively and will be discussed in Part 2 of this article.

### **Nimodipine**

The use of Nimodipine in the prevention of Cerebral Vasospasm is now widely accepted (Rusy, 1996). Unlike other calcium channel blockers, Nimodipine is lipid soluble and readily crosses the blood brain barrier, thus exerting a greater effect on cerebral blood flow (Radhakrishnan & Menon, 1997).

There are various theories on the action of Nimodipine in relation to Cerebral Vasospasm. The most widely held theory is that specific application of nimodipine in neurosurgery is to inhibit calcium influx across the cell membrane of vascular smooth muscles, thereby decreasing peripheral vascular resistance and promoting vasodilatation. Nimodipine selectively affects cerebral vessels with the most important clinical action is to block calcium entry into ischaemic brain hence preventing progression to infarction (Sosnowski, 1990).

Potential side effects of Nimodipine include hepatitis, jaundice, gastrointestinal haemorrhage, anaemia, flushing, diaphoresis, wheezing, thrombocytopenia, light-headedness, dizziness, rebound cerebral vasospasm, palpitations, hypotension, haematuria and haematoma (Peck, 1983). Also, those with other serious underlying conditions such as epilepsy and various heart abnormalities can be severely compromised by the synergistic effect Nimodipine has on Phenytoin and anti-hypertensive drugs (Sosnowski, 1990). Patients with known decreased or altered hepatic functions are at a high risk of developing a life threatening hepatitis due to the potential to exacerbate this condition. As a result careful monitoring of these drug levels must be performed. Due to these complications Nimodipine maybe ceased if it compromises a patient. In particular if the use of Nimodipine decreases a patient's blood pressure less than

120 systolic this may also compromise their cerebral perfusion and thus will be ceased. In Part 2 of this article other methods available to prevent cerebral vasospasm will be discussed.

Although Nimodipine is seen to prevent cerebral vasospasm, the fact that it's exact mechanism and the possible long-term side effects are of great concern. The usual dosage for Nimodipine is 60mg every four hours for oral administration or 30mcg per kilogram per hour for intravenous administration, if blood pressure is maintained within prescribed limits (Crowe et al., 2003).

Mrs X was stabilised and monitored closely overnight without further incident. She was consented to have a clipping of her ACommAA aneurysm the next morning.

### **CONCLUSION**

Stabilising a patient post ASAH can be a very complicated and time-consuming task. Due not to not only the insult of the initial bleed, but also the potential complications of rebleed, cerebral vasospasm, hydrocephalus and seizures, which can be devastating. As Mrs. X has been stabilised in preparation for surgery post-operative considerations will be discussed in Aneurysmal Subarachnoid Haemorrhage, Part 2. Post-operative Nursing. Diagnosis, complications and treatment Management. A composite case Study.

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