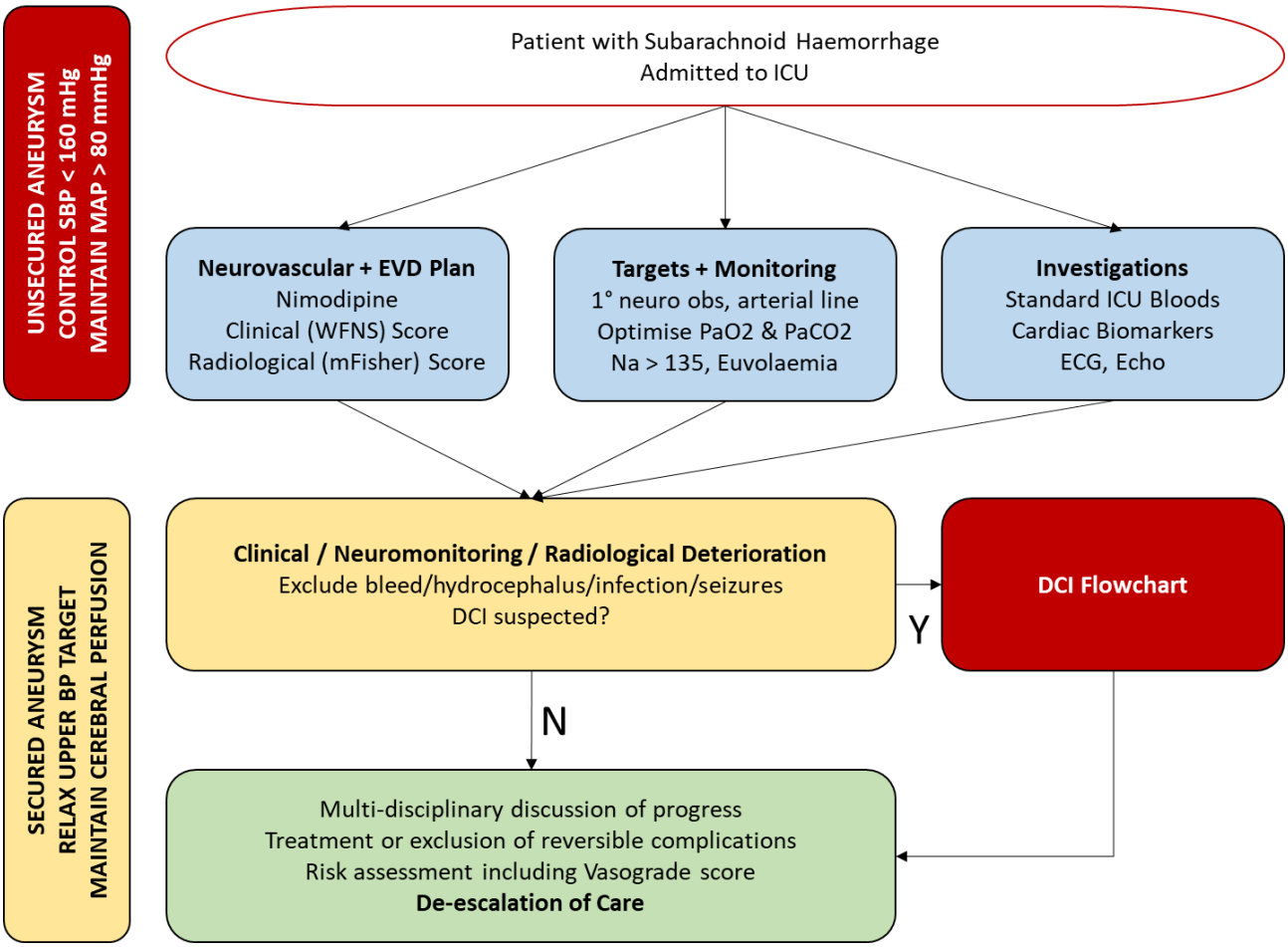
Guidelines for the Management of Subarachnoid Haemorrhage

AIM: To provide guidance on the management of patients with subarachnoid haemorrhage
SCOPE: Royal Sussex County Hospital & Princess Royal Hospital Intensive Care Units

INDEX

SUMMARY FLOW CHART
ICU MANAGEMENT OF SUBARACHNOID HAEMORRHAGE



CLINICAL PRESENTATION

The classic history of subarachnoid haemorrhage (SAH) is one of sudden onset headache, often described as the 'worst imaginable.' However, this is non-discriminatory and only around 1% of patients presenting to emergency departments with headache will subsequently be diagnosed with SAH [4]. Other presentations can include the following:

- Nausea or vomiting (75%)
- Symptoms of meningeal irritation
- Photophobia and visual changes
- Focal neurologic deficits (15%)
- Sudden loss of consciousness
- Seizures during the acute phase (7%)
- Delirium (1%)

Physical examination findings may include the following:

- Mild to moderate BP elevation
- Temperature elevation
- Tachycardia
- Papilledema
- Retinal haemorrhage/intraocular subhyaloid haemorrhages (14%)
- Global or focal neurologic abnormalities

Prodromal signs and symptoms usually are the result of sentinel leaks, mass effect of aneurysm expansion, emboli, or some combination thereof. They can include the following:

- Sensory or motor disturbance (6%)
- Seizures (4%)
- Ptosis (3%)
- Bruits (3%)
- Dysphasia (2%)

DIAGNOSIS

A subarachnoid haemorrhage should be diagnosed if a non-contrast CT scan shows blood in the subarachnoid space. This modality reliably excludes SAH if performed within six hours of symptom onset. Lumbar puncture is therefore not routinely indicated in this context. A lumbar puncture should be considered if a CT scan performed after six hours is negative for a SAH [68,69].

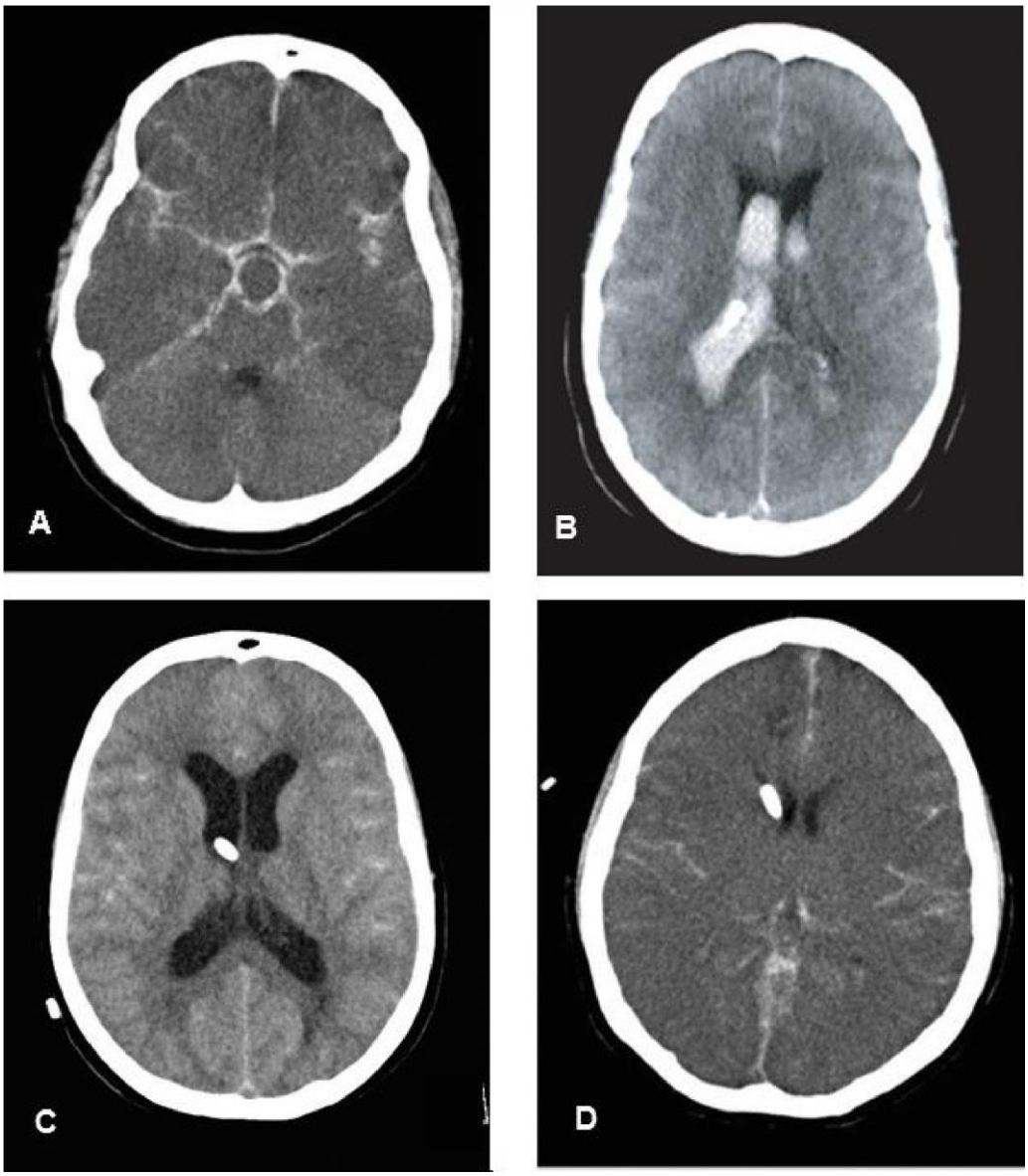


Figure 1 Non-contrast CT scans showing (A) subarachnoid haemorrhage; (B) subarachnoid haemorrhage with extensive intraventricular blood and associated hydrocephalus; (C) subarachnoid haemorrhage with intraventricular drain in situ; (D) subarachnoid haemorrhage and cerebral oedema with loss of appearance of the brain sulci and gyri, and effaced ventricles (with ventricular drain in situ) [5]

Confirmation of the presence of red blood cells, or their metabolites, in the cerebrospinal fluid identifies an additional 3% of patients who subsequently have an aneurysm detected by cerebral angiography. The diagnostic sensitivity of lumbar puncture is increased when performed at least 12 hours after the initial ictus, although this results in a delay in initiating treatment [69].

The opening pressure of cerebrospinal fluid must be recorded and samples analysed for protein, cells, and glucose (paired with a serum sample) CSF bilirubin, CSF spectrophotometry and CSF microbiology

An increase in CSF bilirubin is the key finding, which supports the occurrence of SAH but is not specific for this. In most positive cases, bilirubin will occur with oxyhaemoglobin. Please see pathology for details of CSF collection.

INITIAL ICU MANAGEMENT

All patients:

- Site arterial cannula, aim MAP 80-100
- Systolic BP < 160 mmHg IF aneurysm unprotected
- CVC if vasopressor required to achieve MAP targets
- Maintain oxygenation (SpO2 > 94%)
- Stop oral antihypertensives, use short-acting IV antihypertensives if necessary
- Multi-disciplinary review

Invasively ventilated patients:

- Sedate to achieve RASS -2 to -3
- Head up 30 degrees
- PaO2 > 13 kPa
- PaCO2 4.7-5.3 kPa
- ICP < 22 mmHg and CPP > 60mmHg (if monitor in situ)
- PbtO2 > 20 mmHg (if monitor in situ)

Investigations

- U+Es, Mg, LFTs
- FBC, clotting, G&S
- Troponin & BNP
- ABG
- ECG
- Echo

Immediate management is similar to that for any critically ill patient and focuses on the support of the airway, breathing and circulation. Specific attention must be given to maintenance of cerebral perfusion (mean ABP 80-100 mmHg), whilst minimising the risk of re-bleeding and achieving a rapid diagnosis. **Hypotension should be meticulously avoided.**

Unconscious patients, or those with a deteriorating GCS, should be intubated and ventilated to maintain an initial PaO2 > 13 kPa and PaCO2 4.7-5.3 kPa. The optimum gas exchange targets may be unknown; clinical assessment and multi-modal monitoring should be used where possible to titrate oxygenation and ventilation. Short-term moderate hyperventilation (PaCO2 4.3-4.6 kPa) may be indicated ONLY as rescue therapy for intracranial hypertension, e.g. in the presence of hydrocephalus, an expanding intraparenchymal haematoma or cerebral oedema. [6]

Hypertension is a normal response to SAH, although high blood pressure increases the risk of rebleeding, whereas excessive reductions in blood pressure risk the development of cerebral ischemia. Extreme hypertension (mean ABP $\geq$ 130 mmHg), should be treated cautiously with short-acting agents (labetalol infusion first line, hydralazine second line). Modest elevations in blood pressure (mean ABP <110 mm Hg) do not require treatment. **Analgesia should also be considered in all patients with hypertension.**

Patient should be nursed in bed for first 24 hours with head of bed slightly elevated 30 degrees (although nursing flat may be considered as a temporary measure if vasospasm is suspected).

GRADING

Clinical

The World Federation of Neurological Surgeons grading scale standardises clinical evaluation over time and helps estimate prognosis (Table 1 below):

WFNS Grade	GCS	Motor Deficit
I	15	N
II	13-14	N
III	13-14	Y
IV	7-12	Y or N
V	3-6	Y or N

Table 1: World Federation of Neurological Surgeons grading scale. *GCS – Glasgow Coma Scale*

Radiological

The Modified Fisher scale (table 2) grades SAH according to the CT scan appearances. Worse grades are associated with a progressively higher risk of developing delayed cerebral ischaemia:

Modified Fisher Scale	Cisternal Blood	IVH	Risk of DCI
1	Thin	Absent	24%
2	Thin	Present	33%
3	Thick	Absent	33%
4	Thick	Present	40%

Table 2: Modified Fisher grading of CT appearances in SAH, with associated risk of DCI. *DCI – delayed cerebral ischaemia; IVH – intraventricular haemorrhage.*

Combined

It is also possible to combine the above scores to further stratify patients according to their risk of developing DCI. This has been published as the VASOGRADE score [57] and may assist with targeting of more intensive investigations, and facilitate de-escalation where appropriate:

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

DIAGNOSTIC IMAGING

The choice of diagnostic imaging should be discussed with neuroradiology/ neurosurgery and will normally consist of:

1. CT angiogram (CT-A) interpretation by an experienced neuroradiologist reliably identifies aneurysms > 4 mm. (7)
2. 4-vessel digital subtraction angiography

Negative angiographic findings do not rule out aneurysm. Approximately 10-20% of patients with clinically diagnosed SAH (on CT and/or lumbar puncture) have negative angiographic findings. A repeat angiogram is usually required after 10-21 days in such cases.

The following may also be considered in individual cases:

1. MRI or MRI angiography
2. CT perfusion scans

ANEURYSM CONTROL

Re-bleeding

Re-bleeding was previously the primary cause of death following poor grade SAH but rates have dramatically reduced since the shift towards early securing of the ruptured aneurysm. The greatest risk of re-bleeding occurs within the first 24 hours and is highest in those with the poorest grade. The overall re-bleeding rate is 4-7%, with a 1.5% risk per day for up to two weeks after the ictus and is highest in first 72 hours (5-10%). [2]

Tranexamic acid is **NOT** recommended routinely. Although the risk of rebleeding may be reduced, subsequent cerebral perfusion can be impaired and TXA is not associated with improved functional outcomes [51]. It may be considered for patients with an unavoidable delay in obliteration of aneurysm but only in short term (<72 hours). [3]

Securing the aneurysm

Early aneurysm control reduces the risk of re-bleeding and allows higher ABP to prevent or treat cerebral hypoperfusion. The choice of aneurysm control will be different for each patient, dependent on site and type of aneurysm and will normally be either endovascular technique (coiling) or surgical technique (clipping) as decided by the neurovascular multidisciplinary meeting.

The International Subarachnoid Aneurysm Trial (ISAT) compared endovascular and surgical techniques and confirmed an improvement in early survival in selected patients receiving endovascular therapy, with a small excess of late bleeds. [8] Some criticisms of ISAT include a relatively high exclusion rate, and a lower frequency of posterior circulation and middle cerebral artery aneurysms [9]. However, the proportion of cases that are unsuitable for endovascular treatment is likely to be much lower today because of advances in technology and expertise since publication of the original trial [52].

Most aneurysms are now treated by endovascular options, but coiling is still not a total replacement for surgical treatment. Aneurysms in selected locations, those > 25 mm, those with a wide neck or with branches arising from the aneurysm, may not be amenable to coiling. [9] Long-term follow-up demonstrates improved quality of life outcomes following coiling as compared to clipping [53].

ACUTE DETERIORATION AFTER ANEURYSM TREATMENT

Acute neurological deterioration following coiling of a cerebral aneurysm may be due to thromboembolism related to the coils or bleeding. Thromboembolism can often be treated effectively by the prompt administration of ReoPro (Abciximab). ReoPro is a potent anti-platelet antagonist which acts on platelet aggregation, the cause of coil related thromboembolism. Rarely, acute neurological deterioration is due to haemorrhage, despite an apparently successful coiling procedure.

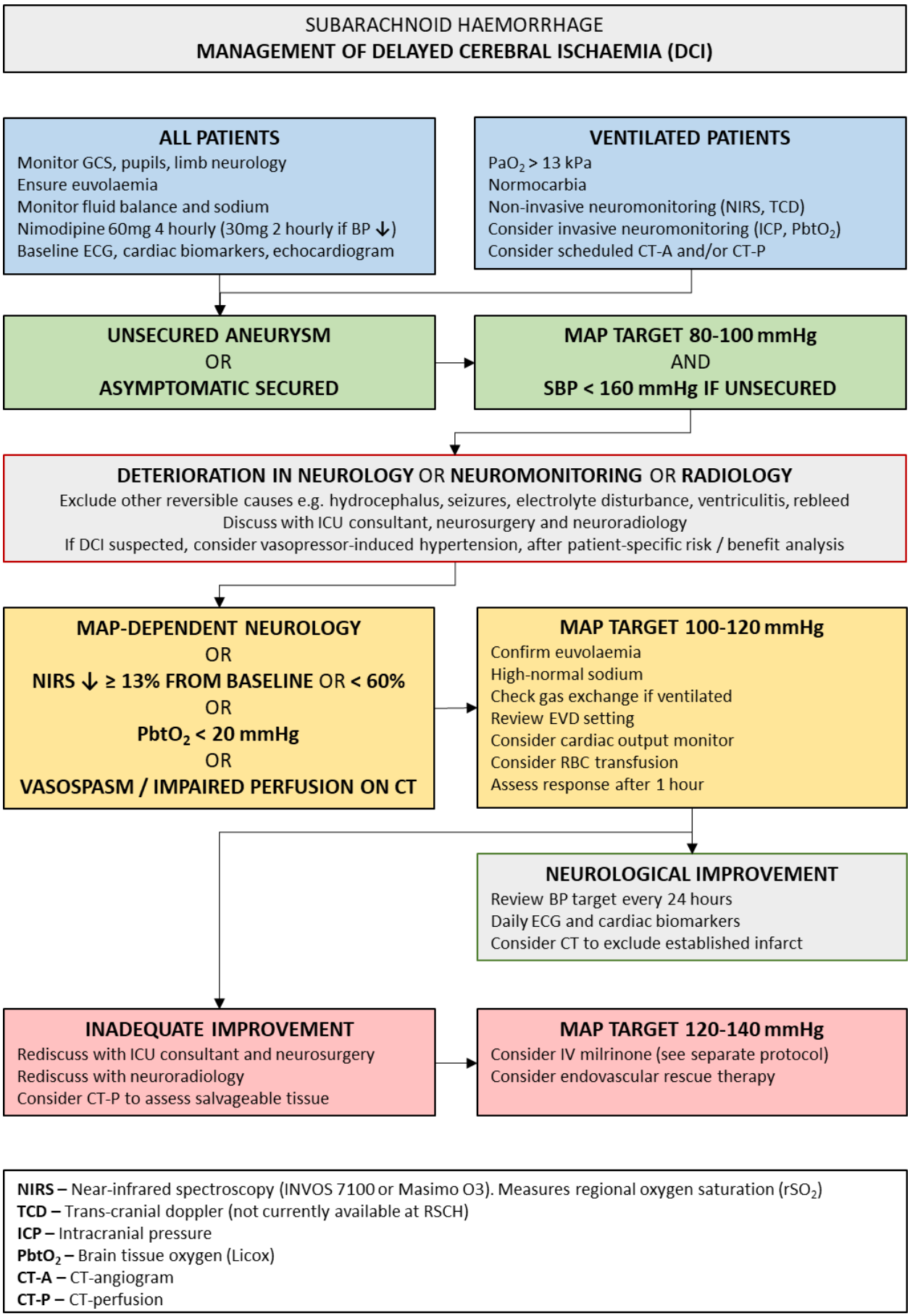
If a patient deteriorates neurologically in the first 24 hours post-coiling:

1. Arrange a CT head and discuss the case immediately with the duty neurosurgical registrar, who should then contact the interventional neuroradiologist, depending on findings
2. Abciximab may be indicated if thrombosis is suspected
3. Discuss subsequent heparinisation and/or aspirin therapy with the interventional neuroradiologist
4. If the aneurysm is large or giant (> 12mm), consider treatment with intravenous dexamethasone to reduce possible effects of peri-aneurysmal oedema related to thrombosis
5. Continue close observation of femoral artery puncture sites, arterial lines, ventricular access devices etc., following administration of abciximab

DELAYED CEREBRAL ISCHAEMIA

DCI is a term applied to any neurological deterioration, including focal neurological deficits and altered consciousness, which persists for more than one hour and cannot be explained by other abnormalities identified by radiographic, laboratory or electrophysiological investigations. It may be unrecognised clinically in some patients because of their poor clinical grade or the concurrent administration of sedatives.

DCI occurs in around 30% of patients, peaks between 4 and 10 days after the ictus and persists for several days. It can occur up to day 21 post-bleed. It is second only to the initial haemorrhage as a cause of morbidity and mortality after SAH. A summary flowchart of DCI management is detailed below:



NIRS – Near-infrared spectroscopy (INVOS 7100 or Masimo O3). Measures regional oxygen saturation (rSO_2)
TCD – Trans-cranial doppler (not currently available at RSCH)
ICP – Intracranial pressure
$PbtO_2$ – Brain tissue oxygen (Licox)
CT-A – CT-angiogram
CT-P – CT-perfusion

Pathophysiology

Although DCI has been attributed to cerebral vasospasm, the exact relationship between the two is unclear. DCI can occur in the absence of vasospasm and *vice versa*, and ischemia often involves more than one vascular territory [11]. Other mechanisms contributing to DCI include vascular dysautoregulation, micro thrombi, direct neurotoxic effects and cortical spreading depolarisation [12].

Diagnosis

DCI is detected clinically by a reduction in level of consciousness with or without a focal neurologic deficit. In unconscious or sedated patients, detection can be difficult and multiple modes of neuromonitoring may be of assistance:

- Brain tissue oxygen monitoring (PbtO2) [61]
- Near infrared spectroscopy [62,63]
- Angiography
- CT angiography and CT perfusion [64,65]
- Transcranial doppler - blood flow velocity (not recommended in NICE guidelines)
- Microdialysis (experimental)

TREATMENT

Nimodipine

All patients should receive enteral nimodipine 60 mg four-hourly immediately after diagnosis until day 21. Nimodipine is a calcium channel blocker of the dihydropyridine group with preferential activity on cerebral vessels. This reduces the incidence of DCI and improves outcome [14]. Nimodipine can cause hypotension; this may be managed by changing the dose to 30mg 2-hrly in the first instance.

Hypertensive therapy

In the presence of a secured aneurysm, maintenance of ABP at supra-normal levels is widely used in the management of DCI [15], although there is a lack of randomised controlled trials to support this [60]. The target blood pressure should be increased in a stepwise fashion, guided by assessment of neurological function, neuromonitoring or radiological evidence of improved perfusion. Prophylactic haemodynamic augmentation should be avoided [68].

Triple H therapy - hypervolemia, haemodilution and hypertension - was previously used to prevent and treat DCI. However, this was not supported by good quality evidence, and while fluid therapy is a key component of the management of SAH, prophylactic hypervolaemic therapy is not effective in raising CBF or improving neurological outcome. There is also some evidence of harm from overly aggressive filling [17]. Consensus guidance recommends that euvolaemia rather than hypervolemia should be the target for both prophylaxis and treatment of DCI, and that haemodilution should not be used [16, 68].

Isotonic crystalloids (Hartmanns or normal saline) are the initial fluids of choice, although hypertonic saline may sometimes be required to manage hyponatraemia. In the presence of adequate volume status, noradrenaline is widely used to augment blood pressure. Central venous pressure is an unreliable indicator of volume status after SAH and although invasive ABP and cardiac output monitoring are often used to guide volume and vasopressor therapy, no technology has been demonstrated to improve outcome. A PICCO may be useful if noradrenaline requirements are either rapidly increasing, in the presence of concurrent sepsis or when noradrenaline requirements are greater than 0.2 mcg/kg/min.

Milrinone

Milrinone is a phosphodiesterase-3 (PDE-3) inhibitor used as an unlicensed treatment for DCI secondary to cerebral vasospasm. The inhibition of PDE-3 present in cerebrovascular smooth muscle is thought to lead to vasodilation and thus increases cerebral perfusion [67]. Through its effect on interleukin 6, milrinone also exhibits some anti-inflammatory effects which may prevent abnormal proliferation of vascular smooth muscle and remodelling caused by DCI. The exact mechanism of milrinone in treating DCI is unknown but there is evidence to show it may be an effective treatment in otherwise refractory cases. For further details, please see separate guideline on Microguide.

Endovascular rescue

There is some evidence that angioplasty and/or intra-arterial vasodilators may have a role if medical therapy has failed and it should be discussed with interventional neuroradiology [1, 68].

Treatments not currently recommended for DCI

There is no current evidence to support the following:

1. **Hypermagnesaemia**. Magnesium was demonstrated in a randomised clinical trial to have no beneficial effect on incidence of DCI, cerebral infarction or clinical outcome [18], and a subsequent post hoc analysis showed worse clinical outcomes [19] In view of this magnesium should be kept in normal range to avoid arrhythmias
2. **Statins**. The STASH trial did not detect any benefit in the use of simvastatin for long-term or short-term outcome in patients with aneurysmal subarachnoid haemorrhage [20]
3. **Antiplatelets** A Cochrane review suggested a non-significant trend towards improved outcome in patients treated with antiplatelet agents but accompanied by increased risk of haemorrhagic complications [22]

OTHER CAUSES OF DETERIORATION

Acute hydrocephalus

Acute hydrocephalus is common following SAH and may present either incidentally on CT scan or with increasing headache or decreasing GCS. In the acute stage, it is treated initially with either an external ventricular drain (EVD), lumbar drains or serial lumbar punctures. If chronic, a shunt may subsequently be required [3].

Intracranial hypertension

A small number of patients with SAH may develop intracranial hypertension [1]. The management should focus on treatment of hydrocephalus, evacuation of intracranial haemorrhage or cerebral oedema secondary to ischaemic infarction. The basic principles of ICP management are similar to those used in traumatic brain injury (see separate TBI guideline), although higher blood pressure targets may be required.

Seizures

Seizures occur in approximately 20% of patients after SAH. These must be treated aggressively and an EEG requested, but universal prophylaxis is not recommended. Levetiracetam is currently first-line (see separate anti-epileptic guidelines). Non-convulsive status epilepticus should be excluded in patients with poor grade SAH who fail to improve or have neurological deterioration of unknown aetiology [3].

Ventriculitis

EVD-related infection is a significant source of morbidity. If clinically suspected, appropriate antimicrobial therapy should be discussed with the microbiology team. Some centres administer prophylactic intrathecal vancomycin [55], although this is not currently used at RSCH.

NON-NEUROLOGICAL COMPLICATIONS

Non-neurological complications are common after SAH [25] and are associated with worse outcomes. Their incidence is detailed in table 3 below:

Complication	Incidence
Fever	54%
Anaemia	36%
Hyperglycaemia	30%
Hypertension	27%
Hypernatraemia	22%
Pneumonia	20%
Hypotension	18%
Pulmonary oedema	14%
Hyponatraemia	14%
Life-threatening arrhythmia	8%
Myocardial ischaemia	6%

Table 3 Non-neurological complications of subarachnoid haemorrhage

In the Cooperative Aneurysm Study, the proportion of deaths related to non-neurological complications was 23% (similar to that of DCI at 22%) [26]. The intensive care management of non-neurological organ dysfunction and failure presents significant challenges, because optimum treatment for the failing systemic organ system may have potentially adverse effects on the injured brain [28]. The risks and benefits of brain-specific therapy, such as induced hypertension, should be weighed against the risks and benefits to other organ system. This assessment will always be patient specific.

Cardiac complications

Cardiac dysfunction is common after SAH and is associated with DCI and poor outcome [29]. It can be identified with an abnormal ECG, impaired function on echocardiography and elevated cardiac biomarkers (troponin and BNP). It occurs in 20-40% of patients and manifests as spectrum of ventricular dysfunction collectively referred to as the neurogenic stunned myocardium (NSM) syndrome [30]. Possible ECG changes are detailed below:

ECG abnormality	Reported incidence
ST-segment changes	15-51%
Inverted or isoelectric T waves	12-92%
QTc prolongation	11-66%
Prominent U waves	4-47%
Sinus bradycardia	16%
Sinus tachycardia	8.5%

Table 4 ECG abnormalities after SAH

Cardiac dysfunction can be caused by excessive noradrenaline release from myocardial sympathetic nerve terminals resulting in a physiological myocardial denervation in the presence of normal coronary perfusion [31].

This results in a characteristic pattern of LV regional wall motion abnormalities involving the basal and middle portions of the anteroseptal and anterior ventricular walls, with relative apical sparing. Takotsubo cardiomyopathy, also referred to as left apical ballooning, is also a rare cause of ventricular dysfunction after SAH, when it is associated with increased mortality [34].

Although LV dysfunction is usually temporary, it is associated with higher mortality after SAH. In severe cases, can lead to cardiogenic shock and pulmonary oedema. Inotropic support may be required and should be directed by cardiac output monitoring.

DISORDERS OF SODIUM REGULATION

Hyponatraemia

Hyponatraemia after subarachnoid haemorrhage is common and multi-factoral. Cerebral salt wasting (CSW) or the syndrome of inappropriate ADH secretion (SIADH) may be responsible and can co-exist. On occasion iatrogenic haemodilution or drugs, such as a PPI or citalopram, may be responsible. As plasma tonicity reduces, fluid shifts may precipitate cerebral oedema and neurological symptoms, therefore the plasma sodium should be kept $\geq$ 135 mmol / l.

CSW is associated with raised atrial and brain natriuretic peptide and excessive renal sodium and water loss, leading to circulating volume contraction with associated hypotension and tachycardia, although patients can remain cardiovascularly stable due to a combination of fluid replacement and a secondary ADH secretion [40,41]. SIADH occurs because of excess antidiuretic hormone secretion, causing water retention, volume overload and dilutional hyponatraemia.

It is important to distinguish between SIADH and CSW, as the treatment of the two is distinct [40]. The primary treatment of CSW syndrome is volume and sodium resuscitation. Fludrocortisone may also be used to limit the natiuresis, but care must be taken to monitor for hypokalaemia and hyperglycaemia [2]. Once the volume status is stabilised, sodium supplementation can be commenced if required. If the enteral route is available, slow sodium tablets can be administered, otherwise intravenous supplementation is used, titrating the concentration to effect.

In contrast, electrolyte-free water restriction, (e.g. 1000-1500 mL/day), is the usual initial treatment of SIADH but this may be very hazardous in SAH. Cardiovascular instability and cerebral hypoperfusion can be precipitated, **therefore fluid restriction should generally be avoided during the first 21 days after SAH** [2]. Hypertonic saline may be required to raise sodium levels earlier than would be the case in other cohorts. Pharmacological therapies such as demeclocycline and ADH-receptor antagonists are NOT recommended [3].

In all patients with hyponatraemia (serum Na less than 135mmol/L):

1. Check serum and urine osmolality and urinary sodium
2. Consider measuring serum cortisol and TFTs
3. Replace sodium with either slow sodium tablets (600mg- 3g qds) or NaCl added to the NG feed; 3g of sodium can be added to a litre bag of feed
4. Consider fludrocortisone (50-100 mcg BD) to control naturiesis
5. If Na < 130mmol/L, consider hypertonic saline until serum Na is greater than 135 mmol/L
6. Serum Na should be checked 4 hourly on 1.8% NaCL to prevent increases greater than 8mmol/24h
7. Sodium replacement and fludrocortisone should continue till Na $\geq$ 140 mmol/L

Pseudohyponatraemia

If there is a large discrepancy between the laboratory serum Na and blood gas Na consider the possibility of pseudohyponatraemia. This is due to the lab analysers effectively measuring concentration rather than activity. Calculate the osmolar gap (= difference between measured serum osmolality and calculated serum osmolarity), which should be less than 10:

Calculated serum osmolarity = (2 x ($[Na^+]$ + $[K^+]$) + [glucose] + [urea] mmol/L

A laboratory plasma glucose is needed for this calculation. If the calculated serum osmolarity is less than the measured serum osmolality then either there is excessive "space occupation" by triglyceride or protein

(they have to be very abnormal for this to be a substantial issue) or else there is something else osmotically active present (e.g. mannitol, ethanol, methanol, ethylene glycol).

Hypernatraemia

Hypernatraemia independently increases the risk of adverse cardiac outcome and death after SAH and patients with hypernatremia should be monitored for evidence of cardiac dysfunction [42]. Hypernatraemia in SAH can be a consequence of either the use of osmotic diuretics, or diabetes insipidus (DI) which is commonly associated with pituitary ischaemia. This may result from raised intracranial pressure (e.g. following intracranial haemorrhage or hydrocephalus or cerebral oedema) therefore identification of DI should prompt consideration of an urgent CT Scan. Other causes of hypernatraemia include dehydration or excessive saline infusion.

Diabetes insipidus should be suspected if urine output > 250mls/hr. for more than 3 hours and specific gravity <1005. **Confirm** by measuring plasma and urinary osmolalities and electrolytes. In DI, plasma osmolality rises with a marked rise in Na^+ > 150 mmol/l and urine osmolality is very low, with low electrolyte concentrations. Remember that in some patients a diuresis may be appropriate; assess for low plasma and urine osmolality, and a previous high cumulative fluid balance.

If confirmed on laboratory results, a continuing high urine output and a plasma Na+ >155 mmol/l, **refractory to management with fluids,** consider desmopressin (DDAVP) 0.5 micrograms intravenously. It must be recognised that **inappropriate administration of desmopressin in the context of neurocritical illness carries substantial risks of promoting cerebral oedema**. Discussion with the ICU consultant and/or neurosurgical team is essential if there is any doubt about its appropriateness. In the acute phase, desmopressin is preferably given intravenously, usually at a dose of 0.5 micrograms (repeated maximum 1-hrly). Desmopressin must not be administered concurrently with hypotonic fluids, due to the risk of cerebral oedema.

OTHER CONSIDERATIONS

Temperature Management

Targeted temperature management should be initiated if the patient's temperature increases above 37.5 degrees C, once an appropriate infection screen has been performed. If neurogenic fever is detected, targeted temperature management should be initiated as described in Appendix 1 below. TTM should be maintained for as long as there is the potential for secondary brain damage. Active management of shivering may be required, as detailed in Appendix 2 below.

Glycaemic control

Hyperglycaemia is common after SAH, occurring in around 30% of patients, and is associated with adverse outcome [45]. Standard ICU guidelines on management of blood glucose should be followed.

Anaemia

Anaemia is very common and associated with poor outcome after SAH, although transfusion is itself similarly associated with adverse outcome effects [49]. Current guidance recommends that packed red cells be administered to maintain haemoglobin concentration between 80-100 g/l, although higher thresholds might be appropriate in isolated patients. The SAHARA trial is currently investigating transfusion thresholds in this cohort.

DVT prophylaxis

SAH induces a prothrombotic state that may lead to development of DVT and pulmonary embolus. The incidence of DVT ranges from 1.5%-18% [2] with highest incidence in poor grade patients. Mechanical thromboprophylaxis should be used in all patients. The use of low molecular weight and unfractionated heparin in patients may be considered 48 hours after aneurysm has been secured and should be discussed with neurosurgeon/interventional neuroradiologist due to the increased risk of bleeding with these drugs. The presence of an EVD is not a contraindication to chemical thromboprophylaxis but requires prior discussion with neurosurgery [58,59].

GI

- Follow trust guidelines for confirming correct positioning of nasogastric tubes
- Follow unit protocol for establishing enteral nutrition
- Follow unit bowel protocol

Nicotine replacement

Although smoking increases the risk of aneurysmal haemorrhage, nicotine replacement therapy following SAH may reduce the risk of DCI and is associated with improved outcomes [66]

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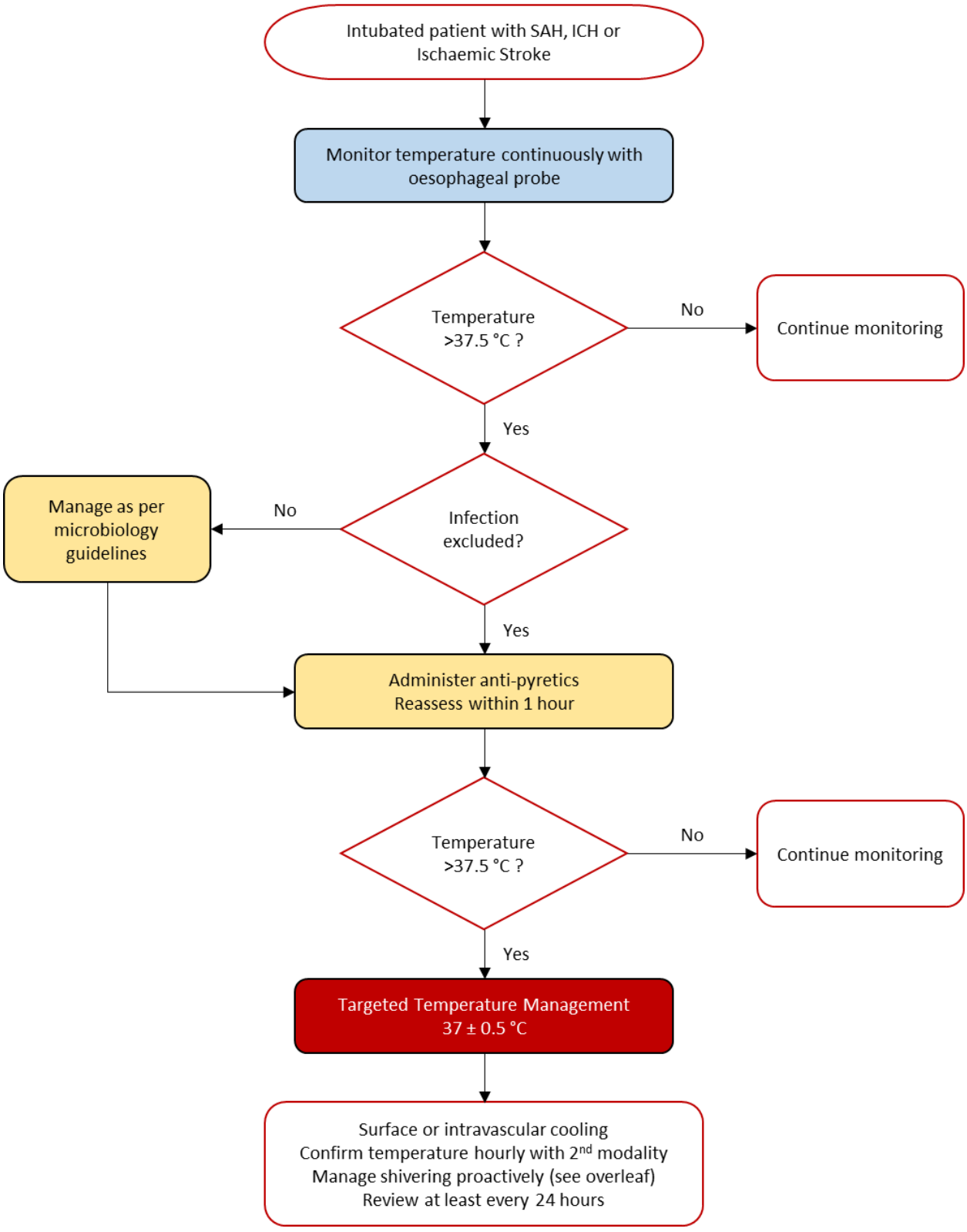
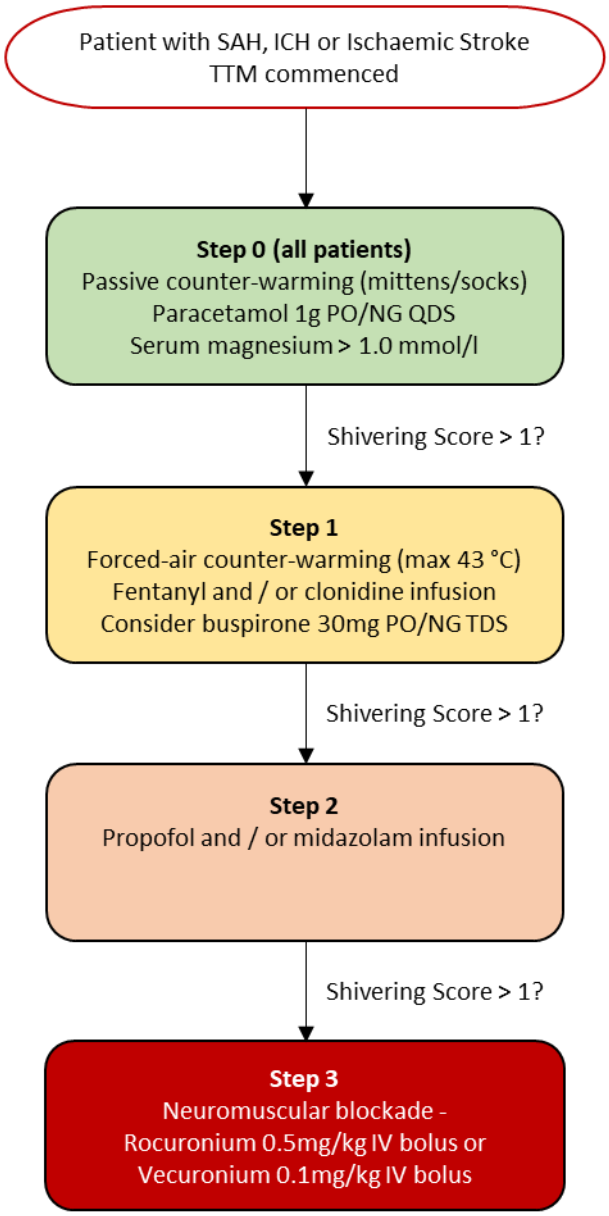
Appendix 1

TEMPERATURE MANAGEMENT IN SAH, INTRACEREBRAL HAEMORRHAGE AND ISCHAEMIC STROKE



Appendix 2

ANTI-SHIVERING PROTOCOL DURING TARGETED TEMPERATURE MANAGEMENT



Shivering score	Type	Features
0	None	No shivering detected on palpation of masseter, neck or chest
1	Mild	Shivering localised to neck or chest, or seen on ECG only
2	Moderate	Shivering involves intermittent movement of upper extremities
3	Severe	Gross movements of upper and lower extremities