SAH READMISSIONS TO NCCU

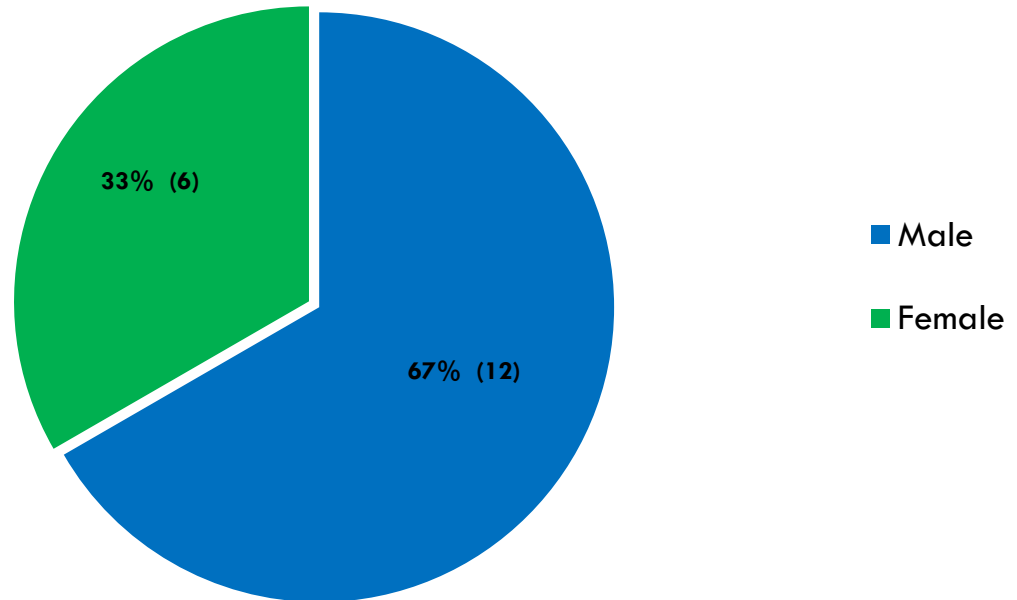Are they preventable ?

João Amaral
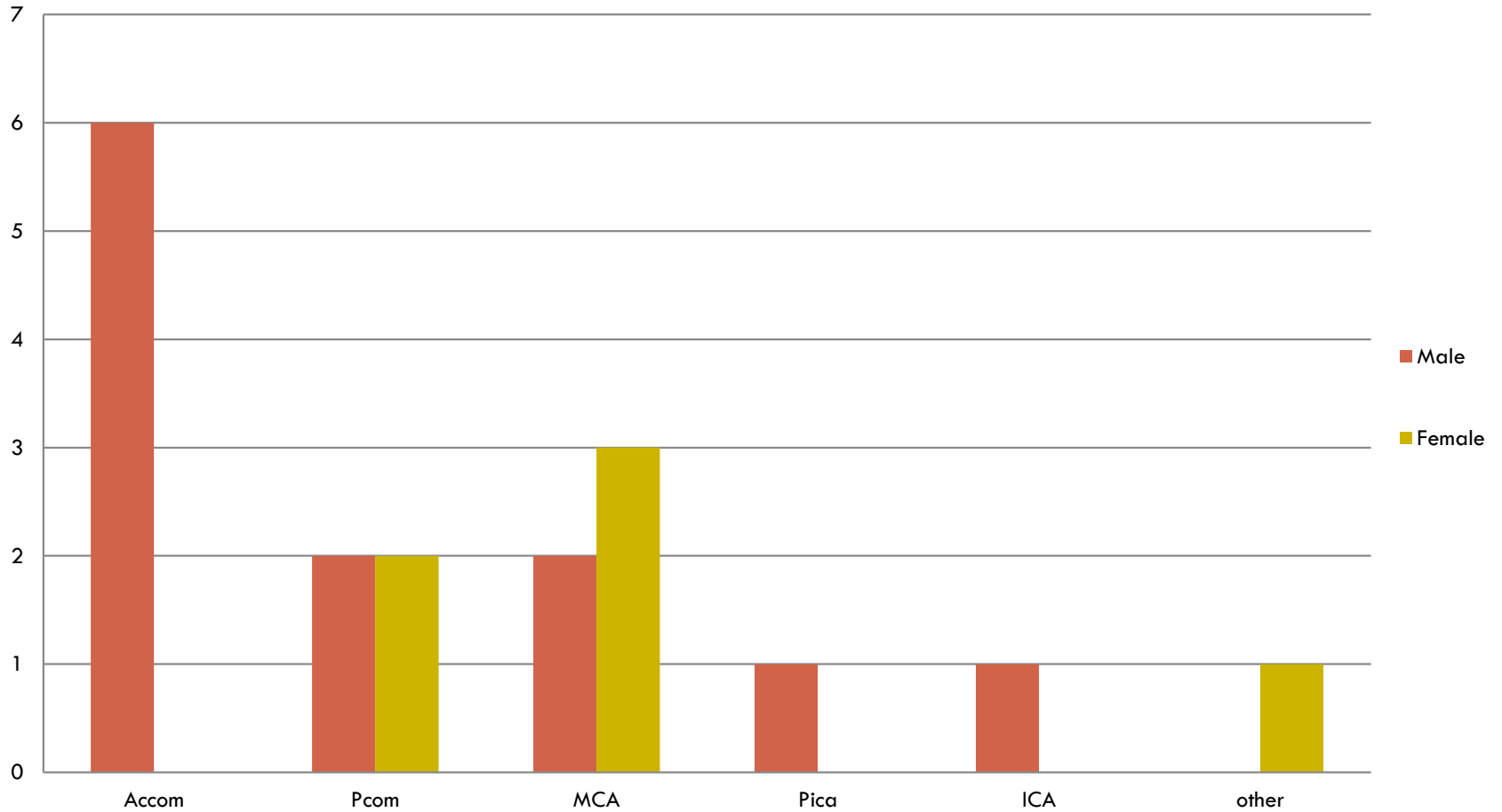*Rebecca Gorf*
Critical Care Outreach Team - NHNN

2015

- Total admissions to NCCU =862
- Total SAH admitted to NCCU= 104 (93e) (12.0%)
- Total SAH **readmissions**= 18 (19.3%)
- Total SAH **readmissions in 48hrs** = 13 (72.2%)
- Average patient's age 58.2yo [32-87]
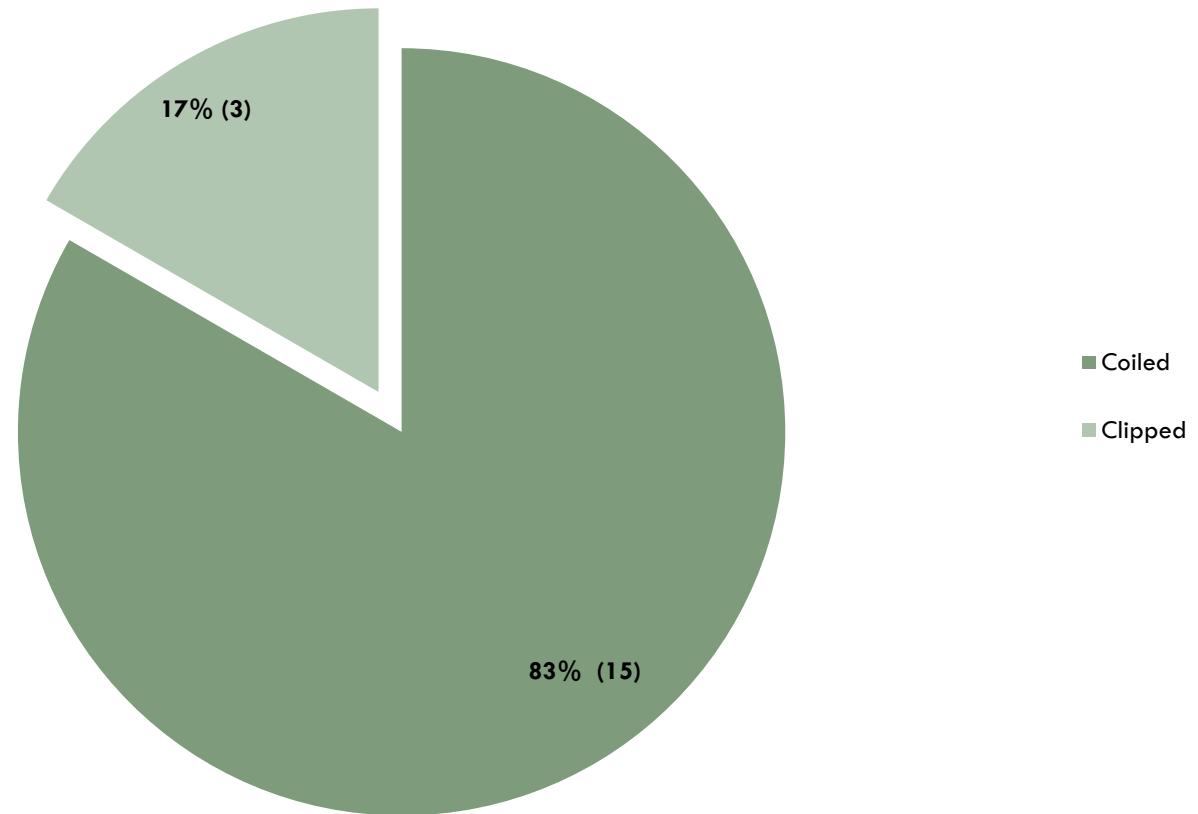- 87% (81) patients seen by CCOT

Demographics
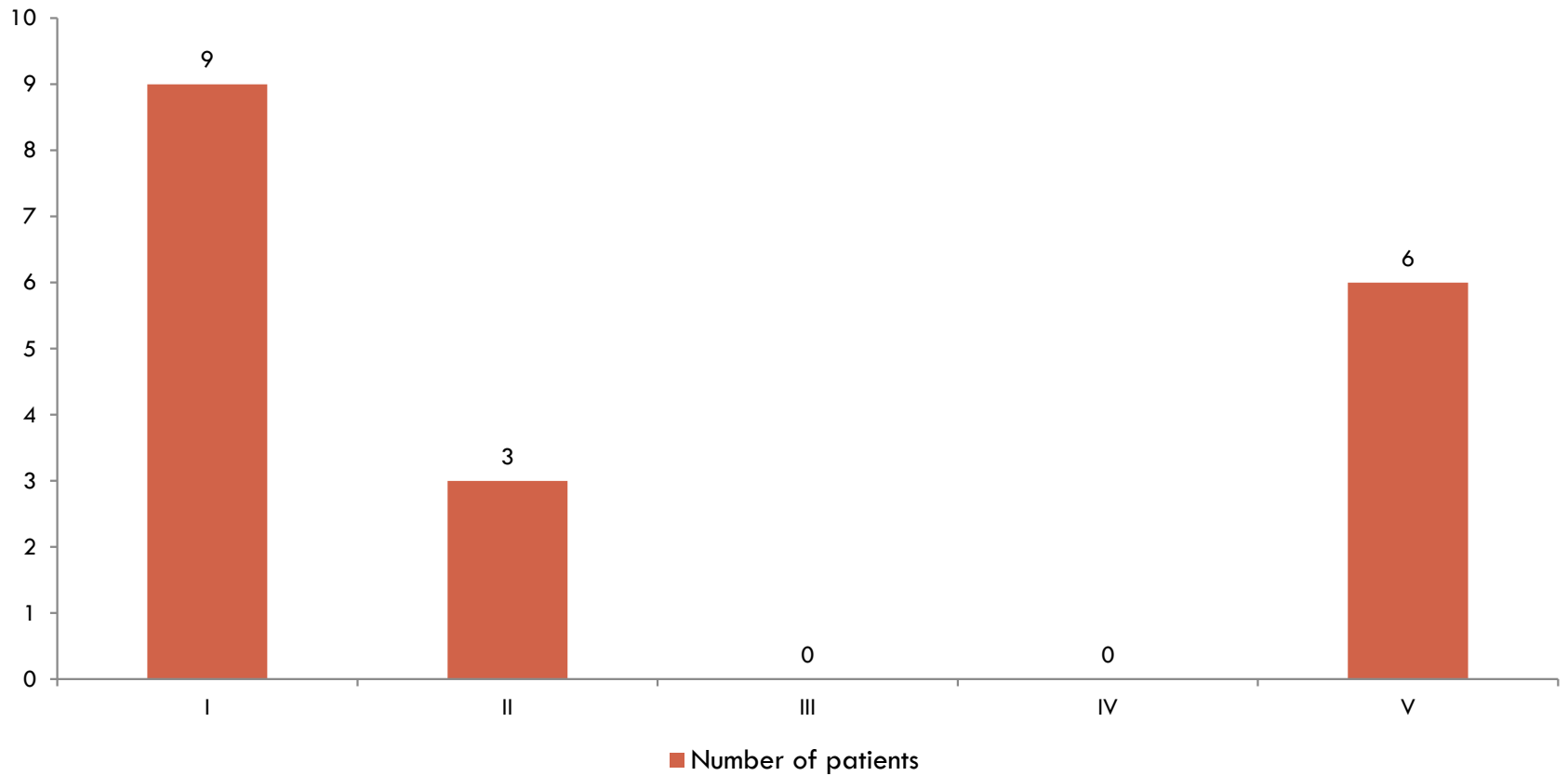
Distribution by gender (readmissions to NCCU)

Aneurysm location

Coiled Vs Clipped

Pie chart showing 83% (15) Coiled and 17% (3) Clipped.

Distribution by Grade (WFNS)

Clinical Causes for readmission

n=18

Legend:
- Vasospasm
- Pneumonia
- Hyponatre
- Hydroceph
- ICH
- Pain Manag

Pie chart values:
- 10 / 56% (Vasospasm)
- 2 / 11% (Pneumonia)
- 1 / 5% (Hyponatre)
- 3 / 17% (Hydroceph)
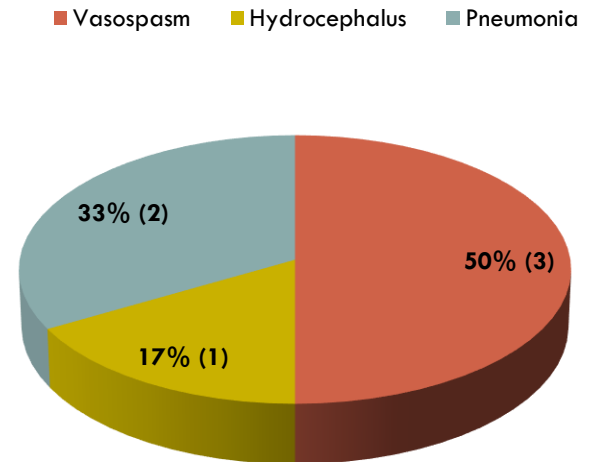- 1 / 5% (ICH)
- 1 / 6% (Pain Manag)
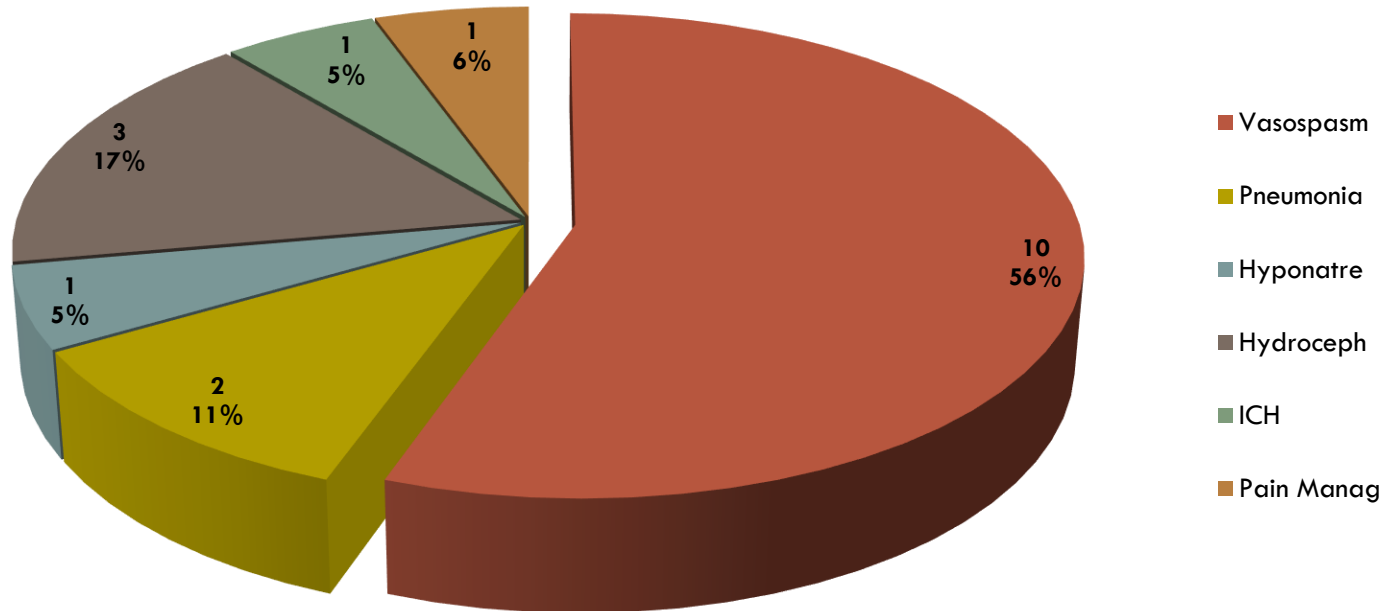
Causes for readmission

WFNS Grade I, II
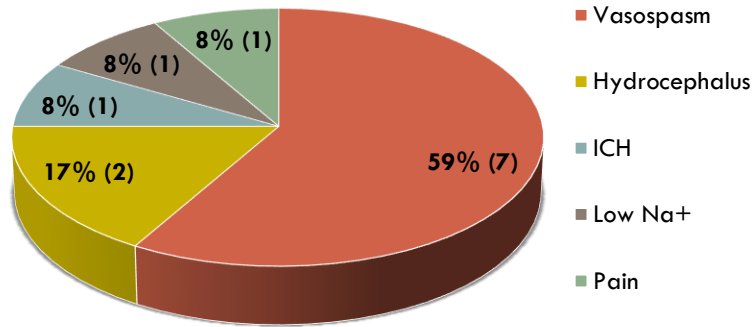
WFNS Grade V

Inotropes and Vasopressors required after readmission to NCCU

15 d in NCCU

Avoidable??

Didn't required
33% (6)

Inotropes or Vasopressors
67% (12)
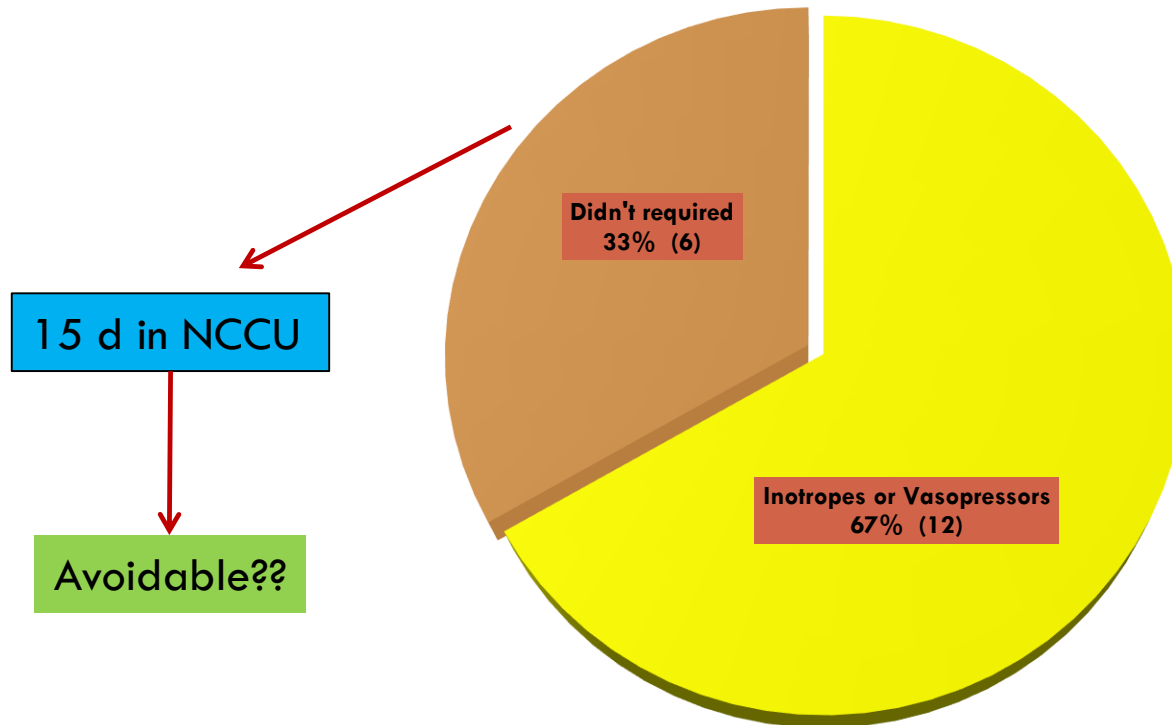
SAH grade Vs vasopressors or inotropes required
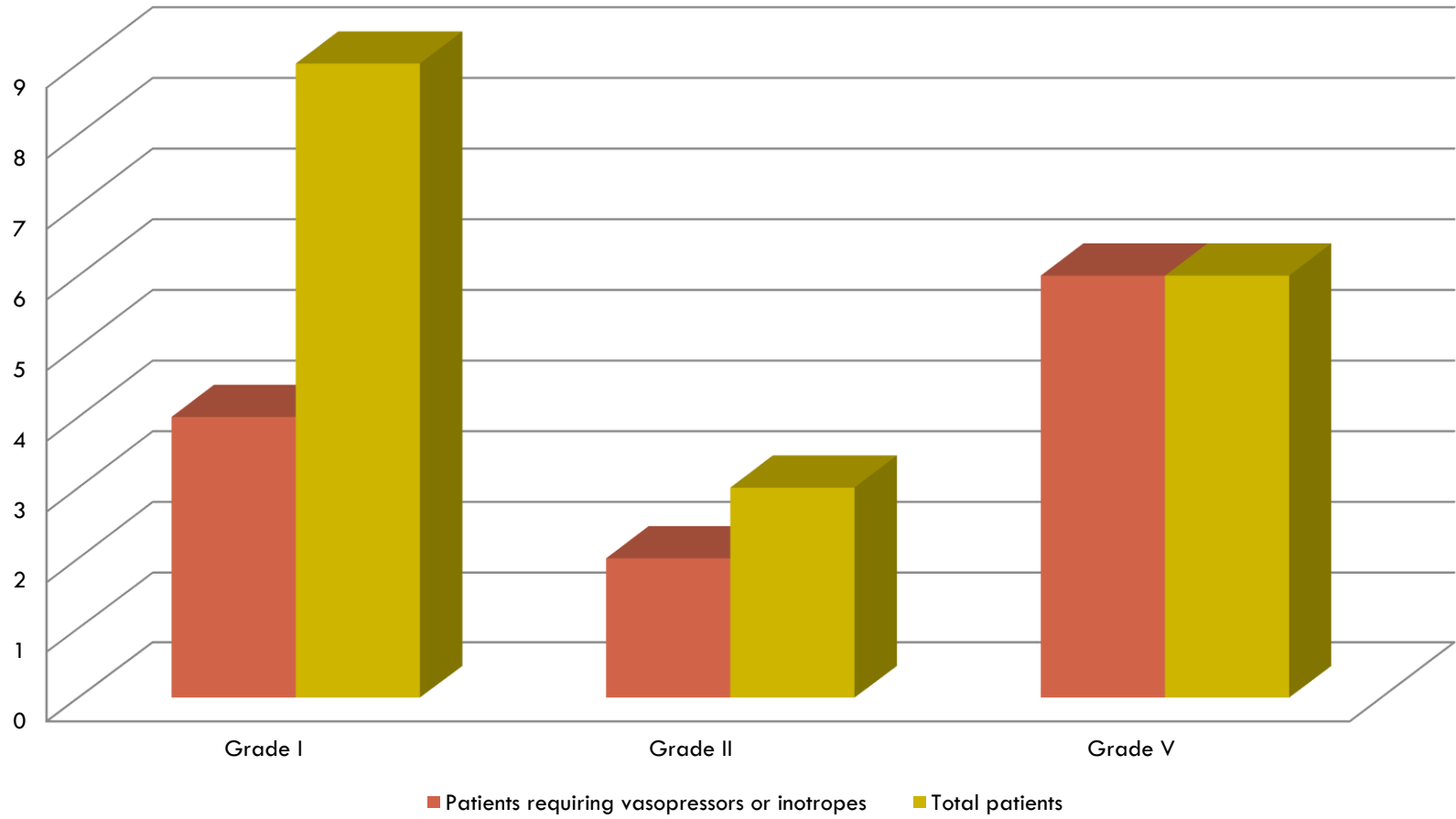
Length of 1ˢᵗ Stay in NCCU

Ward LOS prior readmission to NCCU

72.2% of all SAH readmissions
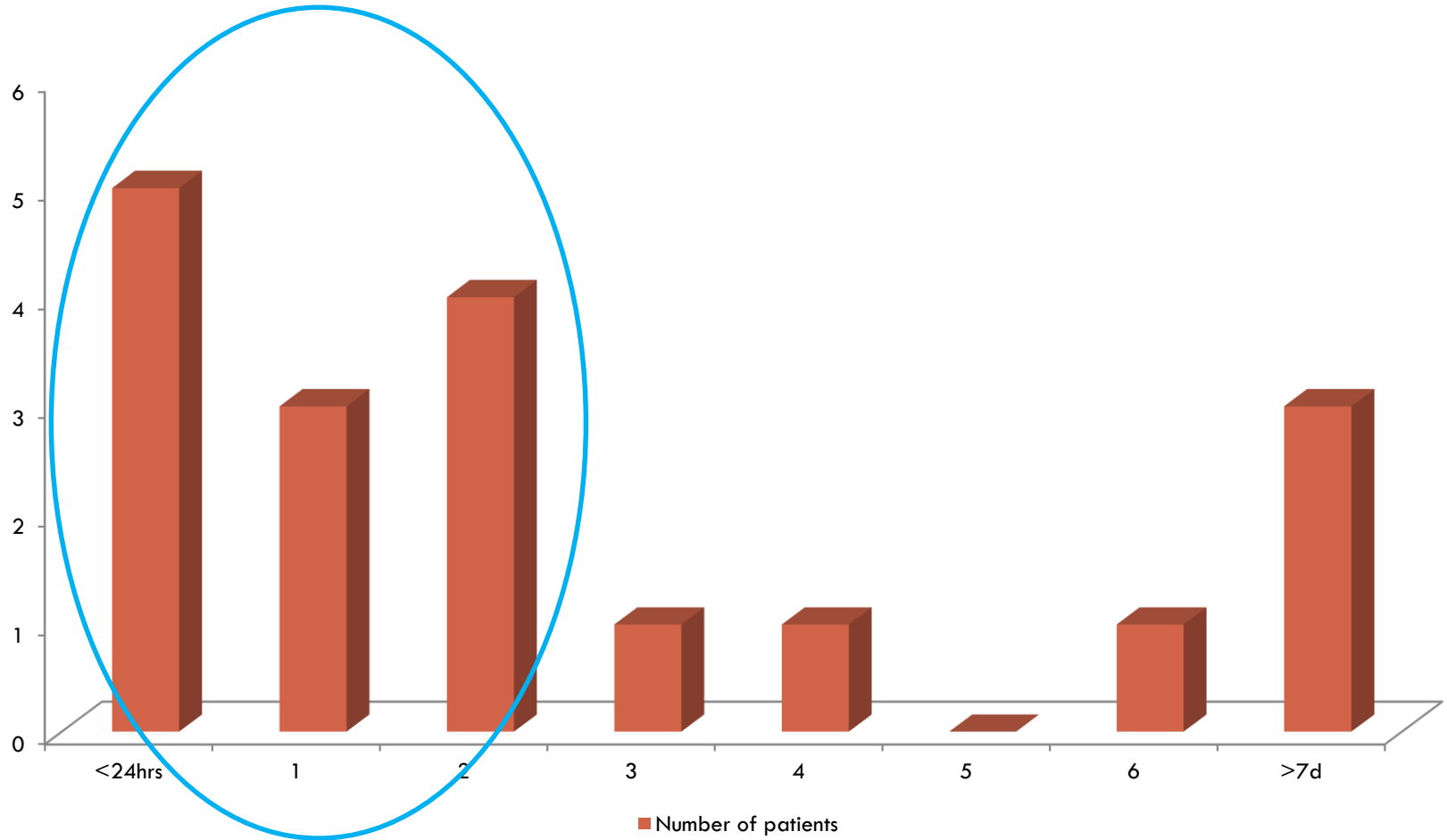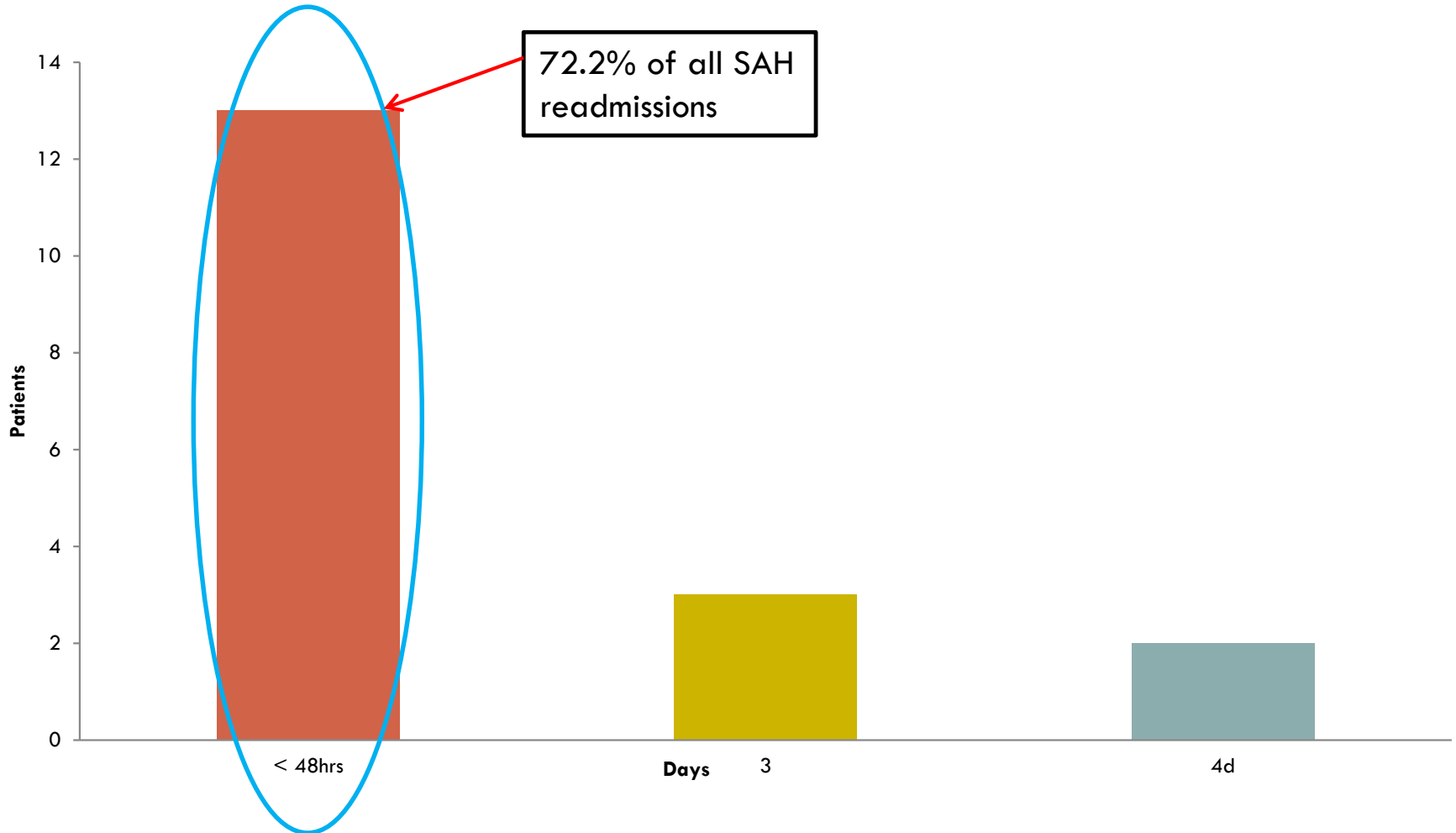
Ward LOS prior readmission to NCCU

- ***Grade I, II***

  Average 1.41d [<24h-4d]

- ***Grade V***

  Average 1.1d [< 24h-4d]

LOS in NCCU after readmission
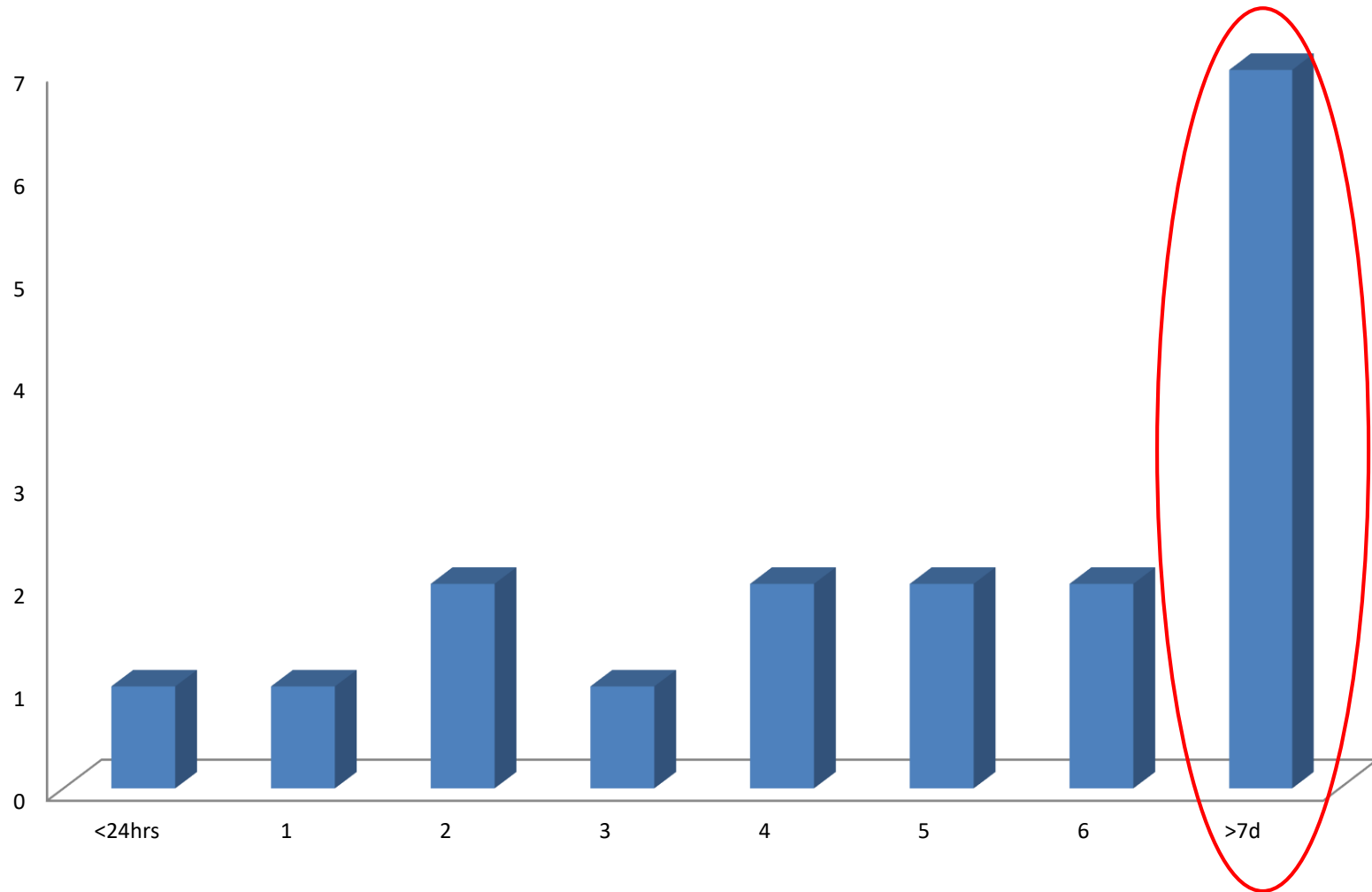
LOS in NCCU after readmission

- **_Grade I, II_**

 Total days= 67 days

  Average 5.58d [<24h-12d]

- **_Grade V_**

 Total days= 54 days

  Average 9.0d [2-16d]

Non-readmitted vs readmitted (LOS)
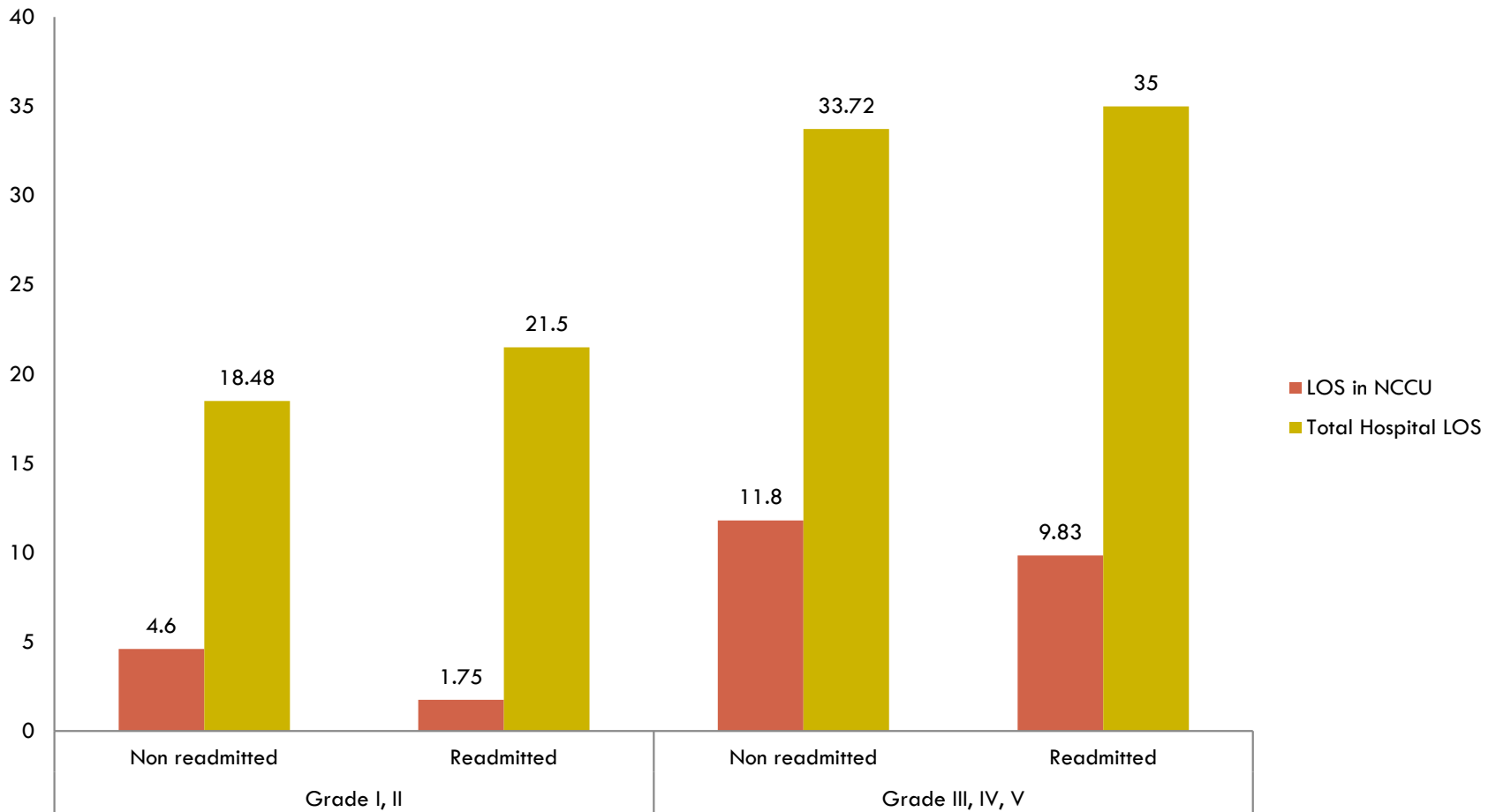
Unplanned readmissions within 48 hrs (2015)

➢ n=13 (72.2%)

Clinical Causes for readmission

n= 1 (8%)
n=2 (15%)
n=1 (8%)
n=2 (15%)
n=7 (54%)

- Vasospasm
- Hydrocephalus
- Pain Management
- Pneumonia
- ICH
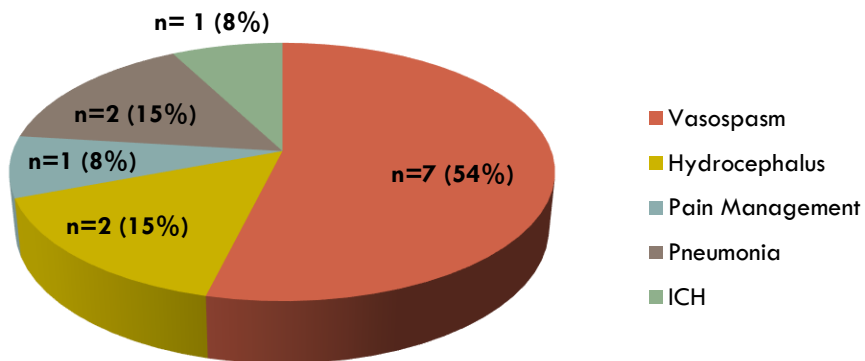
LOS in NCCU after readmission
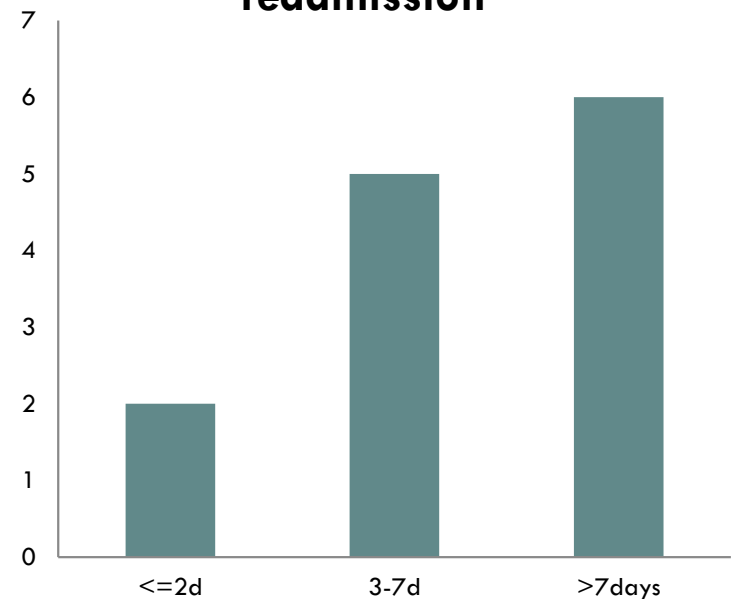
Unplanned readmissions within 48 hrs (2016)

➢ *n=7(41%)*

Clinical Causes for Readmission

Cardiac arrest
n= 1 (14%)

HTN/Low GCS
n=2 (29%)

Vasospasm
n=4
(57%)

LOS in NCCU after readmission

<=2 d   3-7 d   > 7days

Subarachnoid Haemorrhage (SAH)
Management of Cerebral Vasospasm

University College London Hospitals NHS Foundation Trust

MONITORING AND DIAGNOSIS

MONITORING

Observations
GCS, pupil and limb assessment
Vital signs - set BP target

*Neurocritical care patients will have daily **Transcranial Dopplers** during risk period*
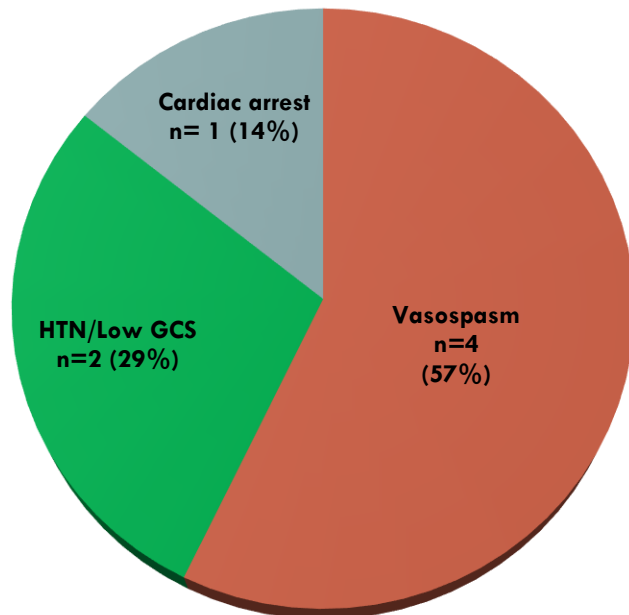
Ventilated patients

BP 'high normal' for patient

Blood gas targets
PaO$_2$ >13kPa
Normal PaCO$_2$
Do not hyperventilate due to added risk of ischaemia from vasospasm

Daily Transcranial Dopplers

If there is good evidence of vasospasm from angiography or direct observation of blood vessels at operation a period of prophylactic hypertensive therapy may be specified if the patient is not clinically assessable

DIAGNOSIS

Detected clinically:
Reduction in conscious level
Focal neurological deficit
- Dysphasia
- Limb weakness
(May be subtle sign e.g. arm drift)

Diagnosis of exclusion
∴Patient needs CT scan to exclude hydrocephalus or established stroke

Clinical signs of vasospasm? — NO → **Continue monitoring and preventive measures**

YES →

PREVENTION AND TREATMENT

Nimodipine
60mg / 4 hourly PO or NG
Commenced as soon as SAH diagnosed and continued for 21 days

If hypotension observed after administration give 30mg / 2 hourly

If patient not absorbing give IV (1-2mg/hr) via dedicated central line lumen (run concurrently with 0.9% saline 40ml/hr)

BP

BP 'high normal' for patient

Euvolaemia
2-3 litres in 24 hours will be target for most patients but consider individual requirements

Clinical signs of vasospasm? — NO → **Continue monitoring and preventive measures**

YES →

FLUID THERAPY

Monitor fluid balance

1st choice:
IV crystalloid

Give fluid bolus

Clinical signs of vasospasm? — NO → **Continue monitoring and preventive measures**

YES →

VASOPRESSORS

Secured aneurysm

Induced hypertension
Patient requires HDU bed
Metaraminol infusion
(see protocol)
If still required after 24 hours insert central line and start noradrenaline
Noradrenaline infusion
Make stepwise increases and titrate to neurology to determine BP target

↑ Systolic BP to reverse neurological deficit
(may be up to 180mmHg)

BP target set by ICU Consultant *after d/w Interventional Neuroradiologist and Neurosurgeon*

Adjust target based on patient response to initial elevation of BP

Unsecured aneurysm

*If aneurysm thought to have ruptured is unsecured **cautious BP elevation** may be attempted*

Unsecured aneurysm not thought responsible for acute SAH should not influence haemodynamic management

*If no reversal of neurological deficit seen after **1 hour** of hypertensive therapy discuss with Interventional Neuroradiologist*

1. Transcranial Dopplers (TCD)
2. CT perfusion – after d/w Interventional Neuroradiologist
3. Proceed to angiography after d/w Interventional Neuroradiologist

Clinical signs of vasospasm? — NO → **Continue monitoring and preventive measures**

YES →

Patient may require transfer to HDU for more invasive monitoring and treatment

Duration of induced hypertension
- Review every 24 hours
- Review TCD results
- Consider trial of lowering BP targets to determine continued need for induced hypertension
-Stepwise reduction in vasopressor based on neurology
- Set new BP target
- Consider rescan to rule out established infarct ∴ contra-indication to induced hypertension

Endovascular options
- Angioplasty
- Direct intra-arterial injection with vasodilators

CEREBRAL VASOSPASM

With current emphasis on early protection of a ruptured aneurysm, cerebral vasospasm leading to delayed ischaemic neurologic deficit (DIND) is the most common cause of late morbidity and mortality

Vasospasm is angiographically demonstrable in about two thirds of patients and one third will go on to develop clinical symptoms of cerebral ischaemia

Vasospasm occurs in a delayed fashion and may be reversible with aggressive preventive and treatment strategies in intensive care

PATHOPHYSIOLOGY

Peaks at 4-10 days after ictus and persists for several days but can occur up to 1 month after ictus

Exact cause remains obscure but its development is directly correlated with blood load in basal cisterns

Constituents of oxyhaemoglobin are likely spasmogenic factors

Most significant consequence of vasospasm is development of DIND secondary to reduced regional cerebral perfusion

Summary

- ❑ SAH patients are unpredictable
- ❑ Preventing SAH readmissions may be impossible
- ❑ Specially with regards to DCI and Vasospasm
- ❑ Non-viability to keep SAHs in HDU > 14 days

Data presented was provided by Case Mix Programme (CMP) database-ICNARC and collected from electronic (ICIP) and paper patient's notes including nursing and medical records.

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Thank you