Neurosurgery

Positioning for neurosurgery

Venous air embolism

Problems associated with raised ICP

Transsphenoidal surgery for acromegaly

Paediatric neurosurgery

Surgery following subarachnoid haemorrhage

Awake craniotomy

UCSF Neuroanesthesia Recommendations

Fluid Management in Neuroanesthesia
Positioning for neurosurgery

General considerations
- Usually prolonged surgery
- Careful identification of pressure areas
- Avoidance of traction on nerves
- Thromboembolic precautions

Supine
- Used for frontal, temporal or parietal access
- Extreme of head rotation may cause venous obstruction, carotid dissection
- Slight head-up usually desirable for venous drainage
- Hip and knee flexion reduces back strain: beach-chair position

Semilateral (“Jannetta”)
- Used for retromastoid procedures
- Table tilted 10-20°, shoulder roll, head rotation
- Avoid extreme head rotation

Lateral
- Used for posterior parietal and occipital access
- Axillary roll to prevent brachial plexus injury
- Stabilization with vacuum bean-bag or lateral rests (potential pressure areas)

Prone
- Used for spinal, occipital, cranial suture and posterior fossa procedures
- For cervical spine and posterior fossa usually head-up and neck flexed
- Requires planning for turning
  - Secure airway and lines, 100% \( O_2 \), removal of most monitoring
  - Unstable cervical spine may require awake intubation and positioning
- Facial support much not cause eye compression and retinal ischaemia
- Other pressure areas: elbows, breasts, iliac crests, genitalia, knees, toes
- Avoid pressure on abdomen: ↑ PAW, IVC obstruction
- Neck flexion may cause compression of base of tongue and pharynx
  - Especially with instrumentation: ETT, TOE

Sitting
- Used for some posterior fossa and cervical spine surgery
- Possibly greater dangers than alternative positions
  - Hypotension, cerebral ischaemia (↓ venous return, ↓ CPP)
  - Perfusion pressure should be measured at ear level
  - Lightly anaesthetized patients may compensate with ↑ SVR, ↓ CO
  - Volume loading and pressors to maintain CPP ≥60 mmHg
  - TEDs stockings or calf compression devices
  - Tongue and pharynx compression or spinal injury from neck flexion
  - Pressure areas: buttocks, potential brachial plexus distraction
  - Venous air embolism ± paradoxical embolism
  - Pneumocephalus
    - May be worsened by \( N_2O \) diffusion after dural closure
    - Cease \( N_2O \) with dural closure
    - PA catheter tip may be in West’s zone 1 (alveolar pressure > PA pressure)
  - Surgery in this position may involve the brainstem
    - Haemodynamic, respiratory, homeostatic disturbance

Some advantages
- Better venous and CSF drainage, possibly better access
Venous air embolism

Incidence
- Depends on procedure, position and method of detection
  - Sitting position posterior fossa surgery with TOE: 76%
  - Less for other positions, surgery and monitors

Aetiology
- Open vessels at lower than ambient pressure
  - Cerebral sinuses, emissary veins, diploic vessels in head-up position
  - Gas under pressure in ventricles, subdural space
  - Gas under pressure in non-neurosurgical procedures: laparoscopy, hysteroscopy, gas-cooled lasers

Detection
- High sensitivity
  - TOE, praecordial Doppler (right sternal edge 3rd-6th intercostal spaces)
- Lower sensitivity, indication of severity and recovery
  - ETCO₂, PAP
- Low sensitivity, indication of incipient arrest
  - BP, ECG, SpO₂

Management
- ABC
  - Prevent further air entry
    - Notify surgeon, flood field
    - Jugular compression, lower head
  - Manage intravascular air
    - 100% O₂, cease N₂O, cease PEEP
    - Aspirate right heart catheter if present
  - Circulatory support: fluid, pressors, chest compression
  - Head-down right lateral position theoretically advantageous
    - Not feasible in most neurosurgery, no evidence for efficacy

Paradoxical embolism
- Requires PFO (25% prevalence) and transient RAP > LAP
  - PFO may be detected by TOE after induction
- RAP to LAP gradient
  - Transiently positive during cardiac cycle
  - Increased by PEEP, greatest with release of Valsalva manoeuvre
  - Reduced by fluid loading
Problems associated with raised ICP

ICP

Intracranial pressure
Usually measured with LP in lateral position or intraventricular catheter
Used to calculate cerebral perfusion pressure
CPP = MAP - greater of JVP and ICP
Rises with intracranial expansile mass
Monro-Kellie Doctrine: volume of cranium is constant
Initial compensation by reduced venous blood volume
Then rapidly rising ICP, ↑ capillary pressure increases cerebral oedema
CBF becomes pressure-passive
Potential for herniation through tentorium

Cerebral blood flow (CBF)
Autoregulated under normal conditions
CPP 60-160 mmHg
Affected by PaO$_2$, PaCO$_2$, cerebral metabolic activity (CMRO$_2$)

Surgery
To relieve intracranial pressure
Craniotomy, resection of lesion, drainage of haematoma
Incidental
Trauma patient with head injury and other injuries
Chronic intracranial hypertension

Assessment
Routine, plus
Neurological findings
Headache, nausea, vomiting, visual disturbance, cranial nerve lesions, irritability and confusion
Intracranial pathology: malignancy, haemorrhage
Timecourse of symptoms
Cardiorespiratory history
Usual blood pressure (baseline for autoregulation of CBF)
Specific diseases
Diabetes, pituitary dysfunction, trauma
Medications
Steroids, anticonvulsants, antihypertensives, mannitol, frusemide
Airway assessment, risk of aspiration
Usual investigations plus imaging

Preoperative
Premedication
Avoid hypercapnia, so no opiates
Usual machine and equipment checking
Monitoring
Routine, plus
ECG arrhythmias common
Arterial line, CVC or long line
IDC, temperature

Intraoperative
Induction
Most agents suitable except ketamine
Barbiturates, propofol reduce CMRO$_2$, CBF and ICP
Non-depolarizing relaxants safe
Histamine release should be avoided
Suxamethonium relatively contraindicated
ICP rise is small and blocked by pre-dosing with NDB
Lignocaine 1.5 mg/kg may reduce ICP rise at intubation
Protect eyes and face
Use armoured tube

**Maintenance**

Continuous deep muscle relaxation
TIVA or inhalational techniques
All agents except ketamine cause ↓ CMR, ↓ CBF in parallel
High concentration of volatiles impair autoregulation (H >> E > I, S, D)
N₂O is a cerebral vasodilator alone (↑ CBF, ↓ CMRO₂)
Propofol TIVA is probably best

Techniques to reduce ICP (in consultation with surgeon)

**Cellular**

Surgical resection
ICF, ECF
Osmotic diuretics (mannitol 0.25-2 g/kg)
Limited by serum osmolarity ≤320 mOsm/l
May cause rebound swelling, hypovolaemia, hypotension
Loop diuretics
↓ ECF and impair idiogenic osmole formation
May reduce rebound swelling
Fluid restriction
Steroids
↓ ICP over 48-72 h, may worsen outcome overall

**CSF**

Surgical drainage

**Blood**

Head-up position (also reduces perfusion pressure)
Lower CMR (with intact autoregulation)
Barbiturates, anticonvulsants, hypothermia
Acute hyperventilation (controversial)
Transient response, risk of ischaemia, rebound on cessation
Avoid agents which impair autoregulation
High dose volatiles, vasodilators
Avoid coughing, straining or high PAW → venous pressure
Hypotension for vascular lesions
Worsens cerebral perfusion

Once the head is open, CPP is a higher priority
Support MAP

**Neuroprotection**

Drugs: barbiturates
Hypothermia
Transsphenoidal surgery in acromegaly.

Acromegaly

Excessive growth hormone secretion
>99% due to pituitary adenomas
Gradual onset of clinical features
Pre-puberty: pituitary giantism, ↑ linear growth plus adult features
Adult
Continued growth of facial, hand and foot bones
Hypertrophy of soft tissues, viscera, skin tags, mucosal polyps
Cardiomyopathy, hypertension, IHD
Diabetes
Proximal myopathy
Local effects
Failure of other pituitary secretion: LH, FSH, ACTH…
Headache
Bitemporal hemianopia

Usually diagnosed in 3rd or 4th decade
Medical therapy with bromocriptine, octreotide, radiation
Surgical excision usually curative

Surgery
Elective, moderate risk
Performed through the nose or an incision under the upper lip
Shared airway, commonly soiled by surgery

Preoperative
Assessment
Routine, plus
Features of acromegaly
Airway compromise: large tongue and jaw, nasal polyps, mucosal folds, recurrent laryngeal nerve palsy
Cardiorespiratory complications
Other disease complications
Diabetes, IHD
Pituitary tumours
Commonly secrete prolactin, occasionally GH, ACTH or TSH
Compress normal tissue with loss of other hormone secretion
Supplement hypoadrenalism (hyponatraemia, hypovolaemia) or hypothyroidism

Investigation
Routine bloods, glucose, crossmatch
Imaging of the head may give information about the airway

Premedication
Important if fibreoptic intubation planned

Intraoperative
Monitoring
Routine plus arterial line, but 50% positive Allen test
Large IV

Induction
Large mask required, mask ventilation may be difficult
Oral intubation, consider awake FOB if likely to be very difficult
Small tube due to incidence of subglottic narrowing
Armoured tube plus throat pack

Positioning
May be heavy, nerve hypertrophy increases risk to ulnar nerve
Head-up reduces bleeding but may cause air embolism

Maintenance
Neuro-type balanced technique
Vigilance for complications
  Disconnection
  Dissection into cavernous sinus with haemorrhage
  Pressure on face or eyes
Antiemetic
Emergence
  Clear blood or CSF from airway
  Aim to minimize coughing
Postoperative
  Ward care
  Attention to complications
    Diabetes insipidus (usually transient)
    Panhypopituitarism
  Analgesia
    Oral ± IM narcotic
Paediatric neurosurgery

Positions
supine, prone, sitting, lateral/park bench, knee-chest
purpose
surgical access, physiological effect (ICP, bleeding control)
considerations
airway
usually IPPV with oral ETT
raises CVP, ICP
compensate with head-up, minimize airway P using deep
paralysis, long inspiratory time, improve compliance with
position (e.g. pressure off abdomen)
SV occasionally in brainstem surgery, still ETT
access
monitoring
pressure areas
especially eyes
specific complications
air embolism in sitting position
diagnosis by TOE, fall in CO₂, fall in SpO₂, calibrate arterial
pressure at head level for hypotension
manage Valsalva, 100% O₂, flood field, neck tourniquet, aspirate
CVC

Control of ICP
Monroe Kellie doctrine
volume of cranium is constant
true after closure of sutures
physiologic control
normal 5-15 cmCSF
remains constant due to redistribution of CSF and venous blood volume
rises sharply after critical point in elastance curve as intracranial "mass"
expands
Physiologic interventions
positioning
head up reduces both ICP and CPP
hypovolaemia, hypotension
PCO₂
fall causes transient fall in ICP due to vasoconstriction
not used below 30 mmHg as may impair CPP
effect is transient
opening the cranium: surgery
Pharmacologic interventions
reducing mass effect
steroids reduce reactive oedema
diuretic agents
mannitol (acute volume-expanding effect)
frusemide
IDC required
can also reduce MAP
agents to reduce CBF, CMRO₂
general anaesthesia, barbiturates
avoiding agents which raise ICP
high PCO₂, Valsalva, coughing
drugs: suxamethonium (but commonly indicated in trauma etc.)
agents which impair autoregulation: volatiles

Protection of the patient
  positioning
    pressure areas, joint hyperextension or malposition
    vascular compromise
    neuropraxia
  temperature
    conservation and warming
  neuroprotection
  drugs
    maintain cerebral autoregulation, reducing CMRO$_2$
    barbiturates, volatiles, propofol
    hypothermia
Surgery following subarachnoid haemorrhage

Subarachnoid haemorrhage

Aetiology, natural history

- Rupture of arterial aneurysm or bleed from AVM
- 40% immediate major morbidity or mortality
- 30% major morbidity or mortality after surgery

Grading

<table>
<thead>
<tr>
<th>Grade</th>
<th>GCS</th>
<th>ICP (cmH₂O)</th>
<th>Mortality</th>
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<tbody>
<tr>
<td>I</td>
<td>15</td>
<td>&lt;10</td>
<td>2%</td>
</tr>
<tr>
<td>II</td>
<td>13-14 without motor deficit</td>
<td>&lt;10</td>
<td>5%</td>
</tr>
<tr>
<td>III</td>
<td>13-14 with motor deficit</td>
<td>15-20</td>
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<tr>
<td>IV</td>
<td>7-12</td>
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<tr>
<td>V</td>
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Surgical management

- Operation before 72 h or after 14 days (reduced risk of vasospasm)
- Ischaemia managed with fluid loading and hypertension
- Nimodipine
  - Believed to reduce vasospasm, probably cell protection
  - Requires CVC administration
  - May cause hypotension

Anaesthetic priorities

- Avoid acute hypertension
- Intraoperative brain “relaxation”
- Maintain high-normal CPP
- Preparation for BP manipulation at clipping or rupture

Preoperative Assessment

- Routine, plus
- Neurological assessment

Complications of SAH

- SIADH or salt-wasting (hyponatraemia, hypovolaemia, high urine Na⁺)
- Vasospasm related to clot around Circle of Willis
- ECG abnormalities: T inversion, QT prolongation, ST depression, U waves
  - No correlation with LV function
  - No specific therapy unless ischaemic pattern

Premedication

- No sedation (may raise PCO₂)

Monitoring

- Routine, plus
- Arterial line, long line for central venous access, large bore IV, IDC, temperature, nerve stimulator

Intraoperative Induction

- Aim to minimize BP rise
- Rebleeding at induction (1%) is usually fatal
- Vasodilator and pressor agents drawn up
- Topical anaesthesia to airway
- Lignocaine, β-blocker, narcotic to smooth intubation
- Suxamethonium probably safe

Maintenance

- Air, O₂, propofol probably causes least cerebral vasodilation in “tight” cases
- Volatile, N₂O probably safe for elective cases
- Maintain low-normal PCO₂, check on ABG
- BP manipulation
Blunting response to pinning (as for intubation)
Maintained planned CPP (e.g. 70 mmHg)
Induced hypotension for uncontrolled bleeding: SNP fastest agent
Induced hypertension for temporary occlusion: metaraminol or phenylephrine

ICP manipulation
Hypocapnia controversial
Lumbar CSF drain, mannitol may be requested by surgeon

Cerebral protection
Propofol commonly used
Thiopentone proven effective but delays awakening so not common
Consider bolus 5 mg/kg for temporary occlusion
Mild hypothermia 32-34°C

AVM surgery
“Perfusion pressure breakthrough”
Closure of AVM and loss of shunt causes sudden increase in perfusion of adjacent brain which has always been vasodilated
Failure of autoregulation response causes rapid oedema of brain

Other considerations
EEG monitoring, angiography with femoral access

Emergence
Avoidance of hypertension, coughing
Consider extubation deep if fasted

Postoperative
Maintain slight head-up position or as required by surgeons
Close monitoring of haemodynamic and neurological status
ICU or HDU level of care
Awake craniotomy

Surgery
Usually for an epileptogenic focus in the temporal lobe

Preoperative
Assessment
Routine, plus
Detailed history of epilepsy
Nature of aura and seizures for intraoperative recognition
Medication and complications

Investigation
Wada test
Unilateral carotid injection of sodium amytal
Determines lateralization of speech, short term memory

Videotelemetry
Continuous EEG with subdural, parenchymal or foramen ovale
electrodes to localize focus of seizures

Premedication
Anticonvulsant agents avoided (benzodiazepines)

Monitoring
Routine, plus
Gas analysis to confirm airway patency
Continuous neurological assessment
Careful attention to patient comfort and warming

Intraoperative
Sedation, analgesia
Must allow patient responsiveness during cortical stimulation
Must not inhibit seizure activity

Drug regimens
Local anaesthetic block and infiltration for pins and incision
Droperidol 2.5-7.5 mg plus narcotic
alfentanil 5-10 µg/kg plus 0.25-0.5 µg/kg/min, or
fentanyl 0.7 µg/kg plus 0.7 µg/kg/h
Propofol infusion or PCA plus narcotic
If provoking agent is required for seizures, methohexitone 0.3 mg/kg
For seizure termination if necessary, thiopentone 1 mg/kg

Surgery
Usually prolonged
Pin placement (if necessary) and craniotomy are painful
Brain parenchyma is insensate
Airway access may be difficult, especially if head is pinned
Fluid Management in Neuroanesthesia

Definitions

Mole

Quantity of a substance containing the same number of particles as in 12 g of $^{12}$C

Osmolality

Number of osmotically active particles per kilogram of solvent
1 Osmolal solution = 1 mole/kg of osmotically active particles
Measured directly by freezing point depression
1 osm/kg of water depresses freezing point by 1.86˚C

Approximated from electrolyte results
2 ($[Na^+] + [K^+] + [glucose] + [urea]$)
Normal 280-290 mOsm/l
Less than the sum of concentrations of solutes because of particle interactions
Approximation is an underestimate when other solutes are present in high concentrations
Mannitol, ketoacidosis, alcohol

Osmolarity

Number of osmotically active particles per litre of solution
1 Osmolar solution = 1 mole/l of osmotically active particles
Theoretical value calculated from concentration of ions and molecules assuming complete dissociation
Measured values slightly less than calculated due to incomplete dissociation
e.g. LR: calculated 275 mOsm/l, measured 254 mOsm/kg

Osmotic pressure

The pressure required to prevent net diffusion of water through a membrane with differing osmolalities of the solutions on each side.
Proportional to the number of osmotically active particles in solution
Calculated using van’t Hoff’s Law
$\pi = CRT$
where $\pi$ is osmotic pressure, $C$ is concentration of solutes in osmoles/l, $R$ is the gas constant and $T$ the absolute temperature
1 mOsm/l exerts 19.3 mmHg pressure at 37˚C
Normal osmolarity of plasma is 282 mOsm/l (5443 mmHg)
Whole-body equilibration of osmotic pressure takes less than 30 minutes

Colloid oncotic pressure

The osmotic pressure exerted across the capillary wall by the non-diffusible elements of plasma.
Ions and small molecules diffuse readily across capillary walls (reflection coefficient close to 0)
Plasma proteins have high reflection coefficients, close to 1
Total plasma protein osmotic pressure is about 19 mmHg in plasma and 8 mmHg in interstitial fluid
Most pressure (12 mmHg) is due to albumin as it is in high concentration and is a smaller molecule than most other plasma proteins
The Gibbs-Donnan effect results in increased cation concentration in plasma to balance anionic non-diffusible proteins. The “excluded volume” effect results from proteins not being an “ideal solute” and increases oncotic pressure. These effects increase plasma oncotic pressure by another 9 mmHg, total 28 mmHg

Equivalent

Quantity of ions which will combine with 1 mole of $H^+$
Equal to number of mole multiplied by charge
e.g. 1 mmol of $Ca^{2+}$ = 2 mEq of $Ca^{2+}$
Starling hypothesis

Flow of water across a membrane is determined by hydrostatic and osmotic pressure gradients and permeability of the membrane.

Hydrostatic pressure in capillaries and interstitium

\[ P_c, \text{ mean capillary pressure } 17.3 \text{ mmHg} \]
\[ P_i, \text{ interstitial pressure } -3 \text{ mmHg} \]

Oncotic pressure in capillaries and interstitium

\[ \pi_c, \text{ plasma oncotic pressure } 28 \text{ mmHg} \]
\[ \pi_i, \text{ interstitial oncotic pressure } 8 \text{ mmHg} \]

Mean net force is 0.3 mmHg out of capillaries

Filtration coefficient 7 ml/mmHg/min for whole body

Net lymph flow 2 ml/min at rest

In the CNS there is little lymphatic drainage

Fluid moves into CSF or via pinocytosis into vessels

Varies widely from tissue to tissue with capillary pressure, permeability, protein concentration changes

\[ J_v = K_f (P_c - P_i - \pi_c + \pi_i) \]

<table>
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Edema formation

Depends on Starling forces and interstitial compliance

Brain interstitium is non-compliant

The cranium is a non-compliant container

Under normal conditions, edema does not form

Compliance increases with disruption of microscopic structure or craniectomy for decompression

Blood brain barrier (BBB)

Cerebral vessels are relatively impermeable

Effective pore size 7Å vs 65Å for systemic vessels

So small ions can exert osmotic pressure across cerebral vessels

Osmotic effect of Na⁺, K⁺, Cl⁻ etc are far greater than protein effects

With an intact BBB, plasma osmolality is far more important than colloid concentration

Small solutes are effective osmotherapy: mannitol, hypertonic saline

Hypotonic solutions promote edema: Lactated Ringer’s

Disrupted or abnormal vessels are readily permeable to both small and large molecules

Neither small nor large solutes exert osmotic pressure

Osmotic therapy is ineffective

Fluid restriction

No proven benefit

Better than administration of hypotonic solutions

May reduce hydrostatic pressure
Hypertonic solutions
  Reduce cerebral edema with an intact BBB
  Hypertonic saline as effective as mannitol or hypertonic colloids
    Choose the solution with least adverse effects
      Coagulopathy with some colloids
      Hypernatremia with saline
  Mannitol the most common choice
    Transient increase in perfusion pressure with rapid administration is
detrimental
    Subsequent reduction in cerebral volume is desirable
    Gradual accumulation in interstitium may cause edema later
Furosemide
  Not a hypertonic solution, but slowly increases plasma osmolality due to water
loss
  Decreases CSF formation
  Synergistic with mannitol
Glucose-containing solutions
  Hyperglycemia is proven to be harmful in cerebral ischemia
  Worsens neurological outcome
  Hypoglycemia is also harmful
Hemoglobin
  Oxygen delivery is related to blood oxygen content and flow
    Content falls with anemia and hypoxemia
    Flow rises with anemia
  High PaO₂ is desirable
  Optimal Hct is about 30% (Hb 100 g/l)
    Active hemodilution is not necessary and not of proven benefit