Surgery in the “beach chair” position

In August 2011 a 50-year-old former rugby player died as a result of a stroke, which occurred during arthroscopic shoulder surgery in the “beach chair” position. The NSW Deputy State Coroner found this was the result of cerebral hypoperfusion “caused by a failure to estimate and maintain an appropriate level of mean arterial pressure in the blood supply of the brain”.

In March 2013, prior to the NSW Coroner’s determination, a committee of surgeons and anaesthetists from Australia and New Zealand was set up by Mr Greg Hoy, the President of the Australian and New Zealand Shoulder and Elbow Society, to examine safety issues surrounding shoulder surgery in the beach chair position. Clinicians on this committee are highly experienced specialists with a particular interest in shoulder surgery and/or safety issues. Members with a range of views were deliberately invited.

Following extensive collaboration, and with the support of ANZCA, suggested guidelines for shoulder surgery in the beach chair position have been developed. This document was prepared by Hugh Pearce, Paul Soeding and Greg Hoy. Contributions were also received from Mark Hayman and Emma Halliday.

Guidelines for shoulder surgery in the beach chair position

Background

The beach chair position (BCP) is extensively used as a routine approach for shoulder surgery in Australia, New Zealand and around the world. Beach chair surgery requires appropriate surgical and anaesthetic management to ensure patient safety. Protection of cerebral perfusion is paramount.

Surgical indications for BCP

Many surgeons consider BCP preferable as it confers certain advantages over the lateral decubitus position, namely:

- Improved access.
- Increased operative arm mobility and ease of examination under anaesthetic.
- Reduced bleeding as a result of improved venous drainage.
- Easier conversion from arthroscopic to open procedures.
- Avoidance of brachial plexus traction injury.

Risk of cerebral injury with BCP

BCP is commonly associated with a fall in blood pressure, and therefore has a potential risk of cerebral hypoperfusion and cerebral injury. The catastrophic complication of global cerebral ischaemia is rare, and has been reported in the literature. This event is considered to occur as a result of significantly reduced cerebral blood flow during general anaesthesia. Normally when cardiac output and arterial blood pressure decrease, cerebral blood flow is autoregulated to maintain adequate flow. If arterial pressure falls below a certain level, this normal protective physiological mechanism is unable to compensate and prevent cerebral ischaemia.

Hypotensive bradycardic events (HBE) under anaesthesia

HBE is an idiosyncratic event occurring with BCP characterised by a significant decrease in mean arterial pressure (MAP), pulse rate and cardiac output. These effects may be exaggerated in BCP.

This problem is well understood by the anaesthetic community and surgeons should also be aware of it when proposing to operate on patients in BCP. Surgeons and anaesthetists must be prepared to take the necessary steps to minimise the occurrence of HBEs and reduce any risk of cerebral hypoperfusion.

Patient selection

It is important to identify patients preoperatively who may be susceptible to the risk of cerebral ischaemia during BCP. Co-morbidities that may increase the risk of cerebral hypoperfusion include:

- Diabetes and autonomic neuropathy.
- Cerebrovascular disease.
- Severe hypertension and generalised vascular disease.
- Cardiac disease.
- History of fainting.
- Fibrile conditions.
- Patients considered to be at increased risk should be referred to a specialist physician or anaesthetist for preoperative assessment and work up, and the anaesthetist (if not already involved) should be forewarned.

Surgeons must be aware that some patients may be clinically unsuitable for BCP by reason of an unacceptable risk to the cerebral circulation. In these cases the surgical approach may need to be reconsidered. A surgeon may have to reduce the angle of elevation of the table or alter their surgical approach with the patient in a supine or lateral decubitus position.

Only specialist anaesthetists who are aware of the potential complexities should provide anaesthesia for these cases.

Operating table

The operating table used for BCP must be a properly designed device meeting current Australian and New Zealand standards. Specifically, it must be secure in the upright position and easily and rapidly laid flat. Positioning of the head and neck must be anatomical and without focal areas of pressure on parts of the neck to ensure normal vascular flow as well as the general avoidance of injury. The airway is positioned to maintain ventilation and the eyes must be protected.

Aims of intraoperative management

A primary aim of anaesthetic management is to maintain MAP and to ensure adequate cerebral perfusion. This involves appropriate monitoring, vigilance and intervention when required. The potential adverse effect of BCP on the circulation may be limited by:

- Ensuring that the patient is properly hydrated within normal fasting guidelines.
- Intravenous fluid loading before induction.
- The use of compression stockings.
- Gradual elevation to BCP.
- Pretreatment with vasopressors.
- Choice of anaesthetic technique.
- Avoidance of hyperthermia.
- Avoidance of hypothermia in ventilated patients.

Monitoring

Full monitoring as per ANZCA guidelines is required. This includes arterial pressure monitoring, ECG, oxygen saturation [SpO2] and other airway gases when general anaesthesia is used.
Arterial pressure must be monitored frequently and accurately. Arterial pressure may be monitored either non-invasively (NIBP) or with intra-arterial pressure monitoring (IABP) as selected by the anaesthetist. NIBP is measured on the nonsurgical arm, not the leg. IABP has the advantage of being continuous and can provide a measure of arterial pressure at the level of the brain when the transducer is placed at the level of the tragus. When NIBP is used a correction for the height between arm and brain is required, since a difference of up to 15-20 mm Hg exists.

**Blood pressure measurement and management**

During BCP surgery, clinicians should aim to keep MAP > 70mm Hg when using NIBP at the arm in healthy patients, and higher in those with hypertension or known cerebrovascular disease, at the discretion of the anaesthetist.

Similarly, deliberate BP reduction below normal levels to control bleeding (controlled hypotension) should be avoided. A blood pressure that is close to normal, for that particular patient, is recommended.

<table>
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<th>Blood pressure management includes:</th>
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<tr>
<td>• Ensuring an adequate MAP following anaesthetic induction and before BCP.</td>
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<td>• Measurement of MAP immediately after BCP.</td>
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<td>• Treating a fall in MAP of greater than 25 per cent from resting baseline or a MAP of less than 70mm Hg.</td>
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<tr>
<td>• Aggressive treatment of HBE using intravenous fluids, cardiac stimulants and vasopressors and if non-responsive to these measures, laying the patient supine. HBE lasting for three minutes or more may lead to permanent cerebral damage.</td>
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**Choice of anaesthetic technique**

Early evidence suggests that anaesthetic technique can influence the frequency and severity of hypotensive events and the need for intervention. It may be inferred that by reducing the incidence of HBE patient safety is increased.

In higher risk patients it may be preferable to use a technique of intravenous sedation with interscalene regional anaesthesia and avoid the use of intermittent positive pressure ventilation (IPPV) with general anaesthesia. Regional anaesthesia with sedation is associated with a lower incidence of HBE compared to general anaesthesia with IPPV. A possible mechanism is IPPV-induced interference with venous return and therefore cardiac output.

The role of neurological monitoring during BCP, including the use of transcranial Doppler or cerebral oximetry is emerging. These techniques require validation before recommendation as a standard of care can be made.

The authors are of the view that these minimum recommendations need to be adhered to in order to minimise the risk of the unlikely event of catastrophic global hypoxic cerebral damage.

**References:**


An updated list of safety alerts is distributed in the first week of each month in the “Quality and safety” section of the ANZCA E-Newsletter. They can also be found on the ANZCA website: www.anzca.edu.au/fellows/quality-safety/safety-alerts