We have examined cerebral pressure autoregulation while awake, and during 0.5 and 1.5 MAC of sevoflurane anaesthesia in 10 patients undergoing non-intracranial neurosurgical procedures. All patients received a standardized anaesthetic comprising premedication with temazepam 20 mg orally, a sleep dose of propofol, fentanyl 1 microgram kg⁻¹ and vecuronium 0.1 mg kg⁻¹. After tracheal intubation, the lungs were ventilated with a mixture of air and oxygen to mild hypocapnia. Routine monitors included ECG, continuous and intermittent non-invasive arterial pressure, pulse oximetry and end-tidal capnography. In addition, blood flow velocity (vmca) was measured by sonating the middle cerebral artery transtemporally using a 2-MHz transcranial Doppler probe. Cerebral pressure autoregulation was tested by increasing mean arterial pressure (MAP) by approximately 20 mm Hg using an infusion of phenylephrine and simultaneously recording vmca. The index of autoregulation (IOR) during each period of the study, calculated as the ratio of percentage change in estimated cerebral vascular resistance (CVRₑ = MAP/vmca) to percentage change in MAP, was compared using ANOVA. vmca during 0.5 and 1.5 MAC of sevoflurane anaesthesia was significantly lower than that while awake (mean 79 (SD 24), 54 (15) and 51 (12) cm s⁻¹, respectively; P < 0.05). There was no significant change in vmca with the increase in MAP while awake, or during 0.5 or 1.5 MAC of sevoflurane anaesthesia and IOR was similar under the three conditions (0.82 (0.11), 0.83 (0.04) and 1.0 (0.03), respectively). We conclude that cerebral pressure autoregulation remained intact during sevoflurane anaesthesia in humans.


Rapid increases in the inspired concentration of desflurane cause transient increases in heart rate and blood pressure. Desflurane also impairs cerebral autoregulation at clinical concentrations. Sevoflurane does not share these hemodynamic side effects. We compared the cerebral and systemic hemodynamic responses to the introduction of desflurane or sevoflurane after the induction of anesthesia with propofol. Twenty healthy adult patients scheduled for nonneurological surgery were recruited. After the induction of anesthesia with propofol, either desflurane or sevoflurane (n = 10 per group) was introduced at 7.2% or 2.2%, respectively, and increased to 10.8% or 3.3%, respectively, 2 min later. Middle cerebral artery blood flow velocity was measured continuously by using a 2-MHz transcranial Doppler ultrasound probe. Heart rate and blood pressure were recorded at 1-min intervals during the 12-min study period. Those patients receiving desflurane had significantly greater middle cerebral artery blood flow velocities, heart rates, and blood pressures than those receiving sevoflurane (P < 0.01). Implications The introduction of desflurane after the induction of anesthesia leads to significant disturbances in cerebral and systemic hemodynamics suggesting loss of cerebral autoregulation and cerebral hyperemia. This may have implications for patients undergoing anesthesia for intracranial surgery.

Bleck TP. Alternatives to evidence based medicine. Different rating scale could be used. Bmj. 2000;321(7255):239.

The aim of this study was to determine the effects of desflurane, at 1 and 1.5 MAC, on cerebral autoregulation. Data were analysed from eight patients undergoing non-neurosurgical procedure. The blood flow velocity in the middle cerebral artery was measured by transcranial Doppler ultrasound and cerebral autoregulation was assessed by the transient hyperaemic response test. Partial pressure of the end-tidal carbon dioxide (Pe’CO2) and mean arterial pressure were measured throughout the study. Anaesthesia was induced with propofol and was maintained with desflurane at end-tidal concentrations of 7.4% (1 MAC) or 10.8% (1.5 MAC). The order of administration of the desflurane concentrations was determined randomly and a period of 15 min was allowed for equilibration at each concentration. The transient hyperaemic response tests were performed before induction of anaesthesia and after equilibration with each concentration of desflurane. An infusion of phenylephrine was used to maintain pre-induction mean arterial pressure and ventilation was adjusted to maintain the pre-induction value of Pe’CO2 throughout the study. Two indices derived from the transient hyperaemic response test (the transient hyperaemic response ratio and the strength of autoregulation) were used to assess cerebral autoregulation. Desflurane resulted in a marked and significant impairment in cerebral autoregulation; at concentrations of 1.5 MAC, autoregulation was almost abolished. Br J Anaesth 2001; 87: 193–7


BACKGROUND:
The authors report a positron emission tomography (PET) study on humans with parallel exploration of the dose-dependent effects of an intravenous (propofol) and a volatile (sevoflurane) anesthetic agent on regional cerebral blood flow (rCBF) using quantitative and relative (Statistical Parametric Mapping [SPM]) analysis.

METHODS:
Using H(2)(15)O, rCBF was assessed in 16 healthy (American Society of Anesthesiologists [ASA] physical status I) volunteers awake and at three escalating drug concentrations: 1, 1.5, and 2 MAC/EC(50), or specifically, at either 2, 3, and 4% end-tidal sevoflurane (n = 8), or 6, 9, and 12 microg/ml plasma concentration of propofol (n = 8). Rocuronium was used for muscle relaxation.

RESULTS:
Both drugs decreased the bispectral index and blood pressure dose-dependently. Comparison between adjacent levels showed that sevoflurane initially (0 vs. 1 MAC) reduced absolute rCBF by 36-53% in all areas, then (1 vs. 1.5 MAC) increased rCBF in the frontal cortex, thalamus, and cerebellum (7-16%), and finally (1.5 vs. 2 MAC) caused a dual effect with a 23% frontal reduction and a 38% cerebellar increase. In the propofol group, flow was also initially reduced by 62-70%, with minor further effects. In the SPM analysis of the "awake to 1 MAC/EC(50)" step, both anesthetic agents reduced relative rCBF in the cuneus, precuneus, posterior limbic system, and the thalamus or midbrain; additionally, propofol reduced relative rCBF in the parietal and frontal cortices.

CONCLUSIONS:
Both anesthetic agents caused a global reduction of rCBF (propofol > sevoflurane) at the 1 MAC/EC(50) level. The effect was maintained at higher propofol concentrations, whereas 2 MAC sevoflurane caused noticeable flow redistribution. Despite the marked global changes, SPM analysis enabled detailed localization of regions with the greatest relative decreases.

BACKGROUND AND OBJECTIVE:
This study was designed to evaluate early postoperative cognitive recovery after total intravenous anaesthesia with remifentanil-propofol or sufentanil-propofol in patients undergoing craniotomy for supratentorial expanding lesions.

METHODS:
Sixty patients were consecutively enrolled, and randomly assigned to one of two study groups: remifentanil-propofol or sufentanil-propofol anaesthesia. To evaluate cognitive function the Short Orientation Memory Concentration Test (SOMCT) and Rancho Los Amigos Scale (RLAS) were administered to all patients in a double-blind procedure before surgery at 15, 45 min and 3 h after extubation.

RESULTS:
Mean extubation time was similar in the two groups (13 +/- 5 min vs. 19 +/- 6 min). A significantly larger number of patients in the remifentanil-propofol group than in the sufentanil-propofol group required antihypertensive medication postoperatively to maintain mean arterial pressure within 20% of baseline (18/30 vs. 4/29; P = 0.0004). Intergroup analysis showed no differences in baseline SOMCT scores (28 +/- 1 vs. 28 +/- 1) whereas mean SOMCT scores at 15, 45 min and 3 h after extubation were significantly higher in the remifentanil-propofol group (30 patients) than in the sufentanil-propofol group (29 patients) (22 +/- 3 vs. 16 +/- 3; P < 0.0001 and 27 +/- 1 vs. 22 +/- 3; P < 0.0001; 28 +/- 1 vs. 26 +/- 2; P = 0.0126).

CONCLUSIONS:
In conclusion, propofol-remifentanil and propofol-sufentanil are both suitable for fast-track neuroanaesthesia and provide similar intraoperative haemodynamics, awakening and extubation times. Despite a higher risk of treatable postoperative hypertension propofol-remifentanil allows earlier cognitive recovery.


Neuroanaesthesia continues to develop and expand. It is a speciality where the knowledge and expertise of the anaesthetist can directly influence patient outcome. Evolution of neurosurgical practice is accompanied by new challenges for the anaesthetist. Increasingly, we must think not only as an anaesthetist but also as a neurosurgeon and neurologist. With the focus on functional and minimally invasive procedures, there is an increased emphasis on the provision of optimal operative conditions, preservation of neurocognitive function, minimizing interference with electrophysiological monitoring, and a rapid, high-quality recovery. Small craniotomies, intraoperative imaging, stereotactic interventions, and endoscopic procedures increase surgical precision and minimize trauma to normal tissues. The result should be quicker recovery, minimal perioperative morbidity, and reduced hospital stay. One of the peculiarities of neuroanaesthesia has always been that as much importance is attached to wakening the patient as sending them to sleep. With the increasing popularity of awake craniotomies, there is even more emphasis on this skill. However, despite high-quality anaesthetic research and advances in drugs and monitoring modalities, many controversies remain regarding best clinical practice. This review will discuss some of the current controversies in elective neurosurgical practice, future perspectives, and the place of awake craniotomies in the armamentarium of the neuroanaesthetist.

**BACKGROUND:**
Propofol target-controlled infusion (TCI) in effect site mode has become popular since it became commercially available. **OBJECTIVE** We have performed a study to assess the pharmacokinetic performance of the Marsh model in effect site mode in an unselected group of patients during neurosurgery during the maintenance phase of anaesthesia.

**DESIGN:**
Fifty American Society of Anesthesiologists (ASA) physical status classes 1 to 3 adults underwent elective neurosurgery receiving propofol TCI using the Marsh model in effect site mode. Propofol dose titration and level of patient monitoring was determined by the attending anaesthesiologist. Arterial blood was sampled at regular intervals during the maintenance phase of anaesthesia and measured plasma propofol concentrations were compared with those estimated using TCI.

**SETTING:**
Large tertiary referral centre in Birmingham, UK, with a specialist neuroanaesthesia service.

**PATIENTS:**
Fifty ASA status I to III adult patients undergoing elective neurosurgery. **MAIN OUTCOME MEASURES:**
Performance of Marsh model as assessed by median performance error (bias) and median absolute performance error (imprecision). **RESULTS** Performance of the Marsh model showed a positive bias (median performance error) of 27.6%, and imprecision (median absolute performance error) of 29.4%. Analysis of pooled data demonstrated greatest bias in the early phase (15 to 30 min) of anaesthesia (mean prediction error of 51.6%). Analysis of covariates demonstrated that obesity (BMI >30 kg m⁻²) contributed around half of the bias detected (mean prediction error 47 vs. 23%, P < 0.001). Patients with advanced age and significant comorbidity (ASA physical status class >2) actually demonstrated significantly lower prediction errors.

**CONCLUSION:**
Pharmacokinetic analysis suggests that the performance of the Marsh model in effect site mode is poor in this broad patient population. The greatest bias demonstrated occurred in the early maintenance phase of anaesthesia. Of the covariates analysed, obesity contributed most significantly to an increased bias. Despite overall poor performance of the Marsh model, attending anaesthesiologists modified targeted propofol concentrations only 0.3 times per hour on average, using remifentanil dose modification nine times more frequently, with good surgical conditions in all patients.


**BACKGROUND:**
Both propofol and volatile anesthetics are commonly used for maintenance of anesthesia in patients undergoing neurosurgical procedures. The effects of these two classes of drugs on cerebral hemodynamics have been compared in many clinical trials. The objectives of this review were to evaluate the cerebral hemodynamic effects, operative conditions, recovery profiles, postoperative complications, and neurological outcomes of propofol-based vs volatile-based anesthesia for craniotomy.

**METHODS:**
MEDLINE®, EMBASE™, Cochrane, and other relevant databases were searched for randomized controlled trials that compared propofol-maintained anesthesia with volatile-maintained anesthesia in adult patients undergoing elective craniotomy. The primary outcome measure was the intraoperative brain relaxation score. Secondary outcome measures included intraoperative cerebral hemodynamics (intracranial pressure [ICP], cerebral perfusion pressure [CPP]), cardiovascular changes, recovery profiles, postoperative complications, and clinical outcomes (neurological morbidity, mortality, quality of life). A meta-analysis was conducted using a random effects model to compare the outcomes of the two anesthetic techniques.

RESULTS:
Fourteen studies (1,819 patients) met inclusion criteria and were analyzed. Brain relaxation scores were similar between the two groups after dural opening; however, ICP was lower (weighted mean difference of -5.2 mmHg; 95% confidence interval -6.81 to -3.6) and CPP was higher (weighted mean difference of 16.3 mmHg; 95% confidence interval 12.2 to 20.46) in patients receiving propofol-maintained anesthesia. Postoperative complications and recovery profiles were similar between the two groups, except for postoperative nausea and vomiting being less frequent with propofol-maintained anesthesia. There were inadequate data to perform a meta-analysis on clinical outcome.

CONCLUSION:
Propofol-maintained and volatile-maintained anesthesia were associated with similar brain relaxation scores, although mean ICP values were lower and CPP values higher with propofol-maintained anesthesia. There are inadequate data to compare clinically significant outcomes such as neurological morbidity or mortality.


BACKGROUND:
Details of current UK anaesthetic practice are unknown and were needed for interpretation of reports of accidental awareness during general anaesthesia (GA) within the 5th National Audit Project.

METHODS:
We surveyed NHS anaesthetic activity to determine numbers of patients managed by anaesthetists and details of ‘who, when, what, and where’: activity included GA, local anaesthesia, sedation, or patients managed awake. Anaesthetists in NHS hospitals collected data on all patients for 2 days. Scaling enabled estimation of annual activity.

RESULTS:
Hospital response rate was 100% with 20,400 returns. The median return rate within departments was 98% (inter-quartile range 0.95-1). Annual numbers (% of total) of general anaesthetics, sedation, and awake cases were 2,766,600 (76.9%), 308,800 (8.6%), and 523,100 (14.5%), respectively. A consultant or career grade anaesthetist was present in more than 87% of cases. Emergency cases accounted for 23.1% of workload, 75% of which were undertaken out of hours. Specialties with the largest workload were orthopaedics/trauma (22.1%), general surgery (16.1%), and gynaecology (9.6%): 6.2% of cases were non-surgical. The survey data describe: who anaesthetized patients according to time of day, urgency, and ASA grade; when anaesthesia took place by day and by weekday; the distribution of patient types, techniques, and monitoring; where patients were anaesthetized. Nine patients out of 15 460 receiving GA died intraoperatively.

CONCLUSIONS:
Anaesthesia in the UK is currently predominantly a consultant-delivered service. The low mortality rate supports the safety of UK anaesthetic care. The survey data should be valuable for planning and monitoring anaesthesia services.


False-positive loss of transcranial electrical motor evoked potentials (TCE-MEPs) limits the efficacy of motor tract monitoring during spine surgery. Although total intravenous anesthesia (TIVA) is widely regarded as the optimal regimen for TCE-MEPs, inhalational anesthesia is an alternative regimen. To compare the rates of false-positive TCE-MEPs during spine surgery for patients anesthetized with TIVA and inhalation anesthesia. A retrospective analysis of data collected from consecutive patients undergoing TCE-MEP monitoring during spinal surgery. Consecutive adult patients from multiple surgical centers undergoing spine surgery inclusive of cervical or thoracic spinal levels during 2008–2009 who received TIVA or inhalation anesthesia. The primary outcome measure was the rate of false-positive alerts using TCE-MEPs, defined as a persistent loss of 90% or greater of the amplitude of TCE-MEP in one or more muscles not attributed to technical or transient systemic factors (hypotension or hypoxia) and not associated with any postoperative neurologic deficit. Patients were divided into two groups according to anesthetic regimen: those anesthetized with one or more inhalational agents (n=1,303) and patients anesthetized with TIVA (n=511). The Fisher exact test and unpaired t test were used to compare group characteristics and false-positive rates. Each group was further subdivided by spinal region (cervical, thoracic, and thoracolumbar) and by presence of preoperative motor deficit. A Pearson chi-squared test was used to identify differences according to spinal region. This study was not supported by any financial sources nor do the authors have any financial relationships to disclose. Patient with inhaled anesthesia showed significantly higher rates of false-positive TCE-MEP changes (15.0% vs. 3.2%) compared with the TIVA group. These differences were significant across all surgical subgroups. The inhaled group had a larger number of patients with preoperative motor deficits compared with TIVA (45.0% vs. 37.4%), a potential confounder for false-positive results. However, a significantly higher rate of false-positive TCE-MEP changes was still observed in the inhaled group (11.4% vs. 0.6% for TIVA) when analyzing only those patients without preoperative motor deficits. Use of inhalation anesthesia during adult spinal surgery is associated with significantly higher rates of false-positive changes compared with TIVA during TCE-MEP monitoring. This relationship appears independent of preoperative motor status. Further study and multivariate analysis of anesthetic agents, diagnosis, and symptoms is necessary to elucidate the impact of these variables. The potential confounding effects of inhalational anesthesia on TCE-MEP monitoring should be considered when determining anesthetic regimen.


Various clinical trials have assessed how intraoperative anesthetics can affect early recovery, hemodynamics and nociception aftersupratentorial craniotomy. Whether or not the difference in recovery pattern differs in a meaningful way with anesthetic choice is controversial. This review examines and compares different anesthetics with respect to wake-up time, hemodynamics, respiration, cognitive recovery, pain, nausea and vomiting, and shivering. When comparing inhalational anesthetics to intravenous anesthetics, either regimen produces similar recovery results. Newer shorter acting agents accelerate the process of emergence and
extubation. A balanced inhalational/intravenous anesthetic could be desirable for patients with normal intracranial pressure, while total intravenous anesthesia could be beneficial for patients with elevated intracranial pressure. Comparison of inhalational anesthetics shows all appropriate for rapid emergence, decreasing time to extubation, and cognitive recovery. Comparison of opioids demonstrates similar awakening and extubation time if the infusion of longer acting opioids was ended at the appropriate time. Administration of local anesthetics into the skin, and addition of corticosteroids, NSAIDs, COX-2 inhibitors, and PCA therapy postoperatively provided superior analgesia. It is also important to emphasize the possibility of long-term effects of anesthetics on cognitive function. More research is warranted to develop best practices strategies for the future that are evidence-based.


OBJECTIVE:
This study aimed to evaluate differences in transcranial electrical motor evoked potential (tcMEP) amplitudes between desflurane/remifentanil and propofol/remifentanil anesthesia treatment plans in patients without preexisting motor deficits (PMDs) undergoing carotid endarterectomy (CEA).

METHODS:
This prospective trial included 21 patients who were randomly assigned to an effect group (Group(DESFLURANE); n=14) or a control group (Group(STANDARD-PROPOFOL); n=7). tcMEP amplitudes were measured 35 min post-induction (T1) either with desflurane or propofol. Treatment was then changed to propofol in Group(DESFLURANE). After an additional 35 min, the tcMEP amplitudes were reevaluated (T2). Differences in amplitudes (DW) between T1 and T2 were calculated for each patient, and the means of these differences were compared between groups.

RESULTS:
TcMEPs were recorded in all 21 patients. At T1, the mean amplitude was 840.1 (SD 50.3) μV and 358.9 (SD 74) μV for Group(STANDARD-PROPOFOL) and Group(DESFLURANE), respectively. The absolute mean difference (T1-T2) between groups was -496.75 μV (p=0.0006).

CONCLUSION:
Desflurane reduces the tcMEP amplitude significantly more than propofol in patients without PMDs undergoing CEA.

SIGNIFICANCE:
TcMEPs were recorded in all patients regardless of the anesthesia regimen. In patients with initially small amplitudes, desflurane may limit tcMEP recording because it produces a remarkable amplitude reduction, even in patients without PMDs.


BACKGROUND:
Anaesthesia for awake craniotomy aims for an unconscious patient at the beginning and end of surgery but a rapidly awakening and responsive patient during the awake period. Therefore, an accurate pharmacokinetic/pharmacodynamic (PK/PD) model for propofol is required to tailor depth of anaesthesia.

OBJECTIVE:
To compare the predictive performances of the Marsh and the Schnider PK/PD models during awake craniotomy.

**DESIGN:**
A prospective observational study.

**SETTING:**

**PATIENTS:**
Twelve patients undergoing elective awake craniotomy for resection of brain tumour or epileptogenic areas.

**INTERVENTION:**
Arterial blood samples were drawn at intervals and the propofol plasma concentration was determined.

**MAIN OUTCOME MEASURES:**
The prediction error, bias [median prediction error (MDPE)] and inaccuracy [median absolute prediction error (MDAPE)] of the Marsh and the Schnider models were calculated. The secondary endpoint was the prediction probability PK, by which changes in the propofol effect-site concentration (as derived from simultaneous PK/PD modelling) predicted changes in anaesthetic depth (measured by the bispectral index).

**RESULTS:**
The Marsh model was associated with a significantly (P = 0.05) higher inaccuracy (MDAPE 28.9 ± 12.0%) than the Schnider model (MDAPE 21.5 ± 7.7%) and tended to reach a higher bias (MDPE Marsh -11.7 ± 14.3%, MDPE Schnider -5.4 ± 20.7%, P = 0.09). MDAPE was outside of accepted limits in six (Marsh model) and two (Schnider model) of 12 patients. The prediction probability was comparable between the Marsh (PK 0.798 ± 0.056) and the Schnider model (PK 0.787 ± 0.055), but after adjusting the models to each individual patient, the Schnider model achieved significantly higher prediction probabilities (PK 0.807 ± 0.056, P = 0.05).

**CONCLUSION:**
When using the 'asleep-awake-asleep' anaesthetic technique during awake craniotomy, we advocate using the PK/PD model proposed by Schnider. Due to considerable interindividual variation, additional monitoring of anaesthetic depth is recommended.


Daily interruption of sedative therapy and limitation of deep sedation have been shown in several randomized trials to reduce the duration of mechanical ventilation and hospital length of stay, and to improve the outcome of critically ill patients. However, patients with severe acute brain injury (ABI; including subjects with coma after traumatic brain injury, ischaemic/haemorrhagic stroke, cardiac arrest, status epilepticus) were excluded from these studies. Therefore, whether the new paradigm of minimal sedation can be translated to the neuro-ICU (NICU) is unclear. In patients with ABI, sedation has ‘general’ indications (control of anxiety, pain, discomfort, agitation, facilitation of mechanical ventilation) and ‘neuro-specific’ indications (reduction of cerebral metabolic demand, improved brain tolerance to ischaemia). Sedation also is an essential therapeutic component of intracranial pressure therapy, targeted temperature management and seizure control. Given the lack of large trials which have evaluated clinically relevant endpoints, sedative selection depends on the effect of each agent on cerebral and systemic haemodynamics. Titration and withdrawal of sedation in the NICU setting has to be balanced between the risk that interrupting sedation might exacerbate brain injury (e.g. intracranial pressure elevation) and the potential benefits of enhanced neurological function and reduced complications. In this review, we provide a concise summary of cerebral physiologic effects of sedatives and analgesics, the advantages/disadvantages of each agent, the
comparative effects of standard sedatives (propofol and midazolam) and the emerging role of alternative drugs (ketamine). We suggest a pragmatic approach for the use of sedation-analgesia in the NICU, focusing on some practical aspects, including optimal titration and management of sedation withdrawal according to ABI severity.


BACKGROUND:
Brain tumour surgery usually is carried out with the patient under general anaesthesia. Over past years, both intravenous and inhalational anaesthetic agents have been used, but the superiority of one agent over the other is a topic of ongoing debate. Early and rapid emergence from anaesthesia is desirable for most neurosurgical patients. With the availability of newer intravenous and inhalational anaesthetic agents, all of which have inherent advantages and disadvantages, we remain uncertain as to which technique may result in more rapid early recovery from anaesthesia.

OBJECTIVES:
To assess the effects of intravenous versus inhalational techniques for rapid emergence from anaesthesia in patients undergoing brain tumour surgery.

SEARCH METHODS:
We searched the Cochrane Central Register of Controlled Trials (CENTRAL; 2014, Issue 6) in The Cochrane Library, MEDLINE via Ovid SP (1966 to June 2014) and Embase via Ovid SP (1980 to June 2014). We also searched specific websites, such as www.indmed.nic.in, www.cochrane-sadcc.org and www.Clinicaltrials.gov (October 2014). We reran the searches for all databases in March 2016, and when we update the review, we will deal with the two studies of interest found through this search that are awaiting classification.

SELECTION CRITERIA:
We included randomized controlled trials (RCTs) that compared the use of intravenous anaesthetic agents such as propofol and thiopentone with inhalational anaesthetic agents such as isoflurane and sevoflurane for maintenance of general anaesthesia during brain tumour surgery. Primary outcomes were emergence from anaesthesia (assessed by time to follow verbal commands, in minutes) and adverse events during emergence, such as haemodynamic changes, agitation, desaturation, muscle weakness, nausea and vomiting, shivering and pain. Secondary outcomes were time to eye opening, recovery from anaesthesia using the Aldrete or Modified Aldrete score (i.e. time to attain score ≥ 9, in minutes), opioid consumption, brain relaxation (as assessed by the surgeon on a 4- or 5-point scale) and complications of anaesthetic techniques, such as intraoperative haemodynamic instability in terms of hypotension or hypertension (mmHg), increased or decreased heart rate (beats/min) and brain swelling.

DATA COLLECTION AND ANALYSIS:
We used standardized methods in conducting the systematic review, as described by the Cochrane Handbook for Systematic Reviews of Interventions. Two review authors independently extracted details of trial methods and outcome data from reports of all trials considered eligible for inclusion. We performed all analyses on an intention-to-treat basis. We used a fixed-effect model when we found no evidence of significant heterogeneity between studies, and a random-effects model when heterogeneity was likely. For assessments of the overall quality of evidence for each outcome that included pooled data from RCTs only, we downgraded the evidence from 'high quality' by one level for serious (or by two levels for very serious) study limitations (risk of
bias), indirectness of evidence, serious inconsistency, imprecision of effect or potential publication bias.

**MAIN RESULTS:**
We included 15 RCTs with 1833 participants. We determined that none of the RCTs were of high methodological quality. For our primary outcomes, pooled results from two trials suggest that time to emergence from anaesthesia, that is, time needed to follow verbal commands, was longer with isoflurane than with propofol (mean difference (MD) -3.29 minutes, 95% confidence interval (CI) -5.41 to -1.18, low-quality evidence), and time to emergence from anaesthesia was not different with sevoflurane compared with propofol (MD 0.28 minutes slower with sevoflurane, 95% CI -0.56 to 1.12, four studies, low-quality evidence). Pooled analyses for adverse events suggest lower risk of nausea and vomiting with propofol than with sevoflurane (risk ratio (RR) 0.68, 95% CI 0.51 to 0.91, low-quality evidence) or isoflurane (RR 0.45, 95% CI 0.26 to 0.78) and greater risk of haemodynamic changes with propofol than with sevoflurane (RR 1.85, 95% CI 1.07 to 3.17), but no differences in the risk of shivering or pain. Pooled analyses for brain relaxation suggest lower risk of tense brain with propofol than with isoflurane (RR 0.88, 95% CI 0.67 to 1.17, low-quality evidence), but no difference when propofol is compared with sevoflurane.

**AUTHORS’ CONCLUSIONS:**
The finding of our review is that the intravenous technique is comparable with the inhalational technique of using sevoflurane to provide early emergence from anaesthesia. Adverse events with both techniques are also comparable. However, we derived evidence of low quality from a limited number of studies. Use of isoflurane delays emergence from anaesthesia. These results should be interpreted with caution. Randomized controlled trials based on uniform and standard methods are needed. Researchers should follow proper methods of randomization and blinding, and trials should be adequately powered.


Background Understanding the patient perspective on healthcare is central to the evaluation of quality. This study measured selected patient-reported outcomes after anaesthesia in order to identify targets for research and quality improvement. Methods This cross-sectional observational study in UK National Health Service hospitals, recruited adults undergoing non-obstetric surgery requiring anaesthesia care over a 48 h period. Within 24 h of surgery, patients completed the Bauer questionnaire (measuring postoperative discomfort and satisfaction with anaesthesia care), and a modified Brice questionnaire to elicit symptoms suggestive of accidental awareness during general anaesthesia (AAGA). Patient, procedural and pharmacological data were recorded to enable exploration of risk factors for these poor outcomes. Results 257 hospitals in 171 NHS Trusts participated (97% of eligible organisations). Baseline characteristics were collected on 16,222 patients; 15,040 (93%) completed postoperative questionnaires. Anxiety was most frequently cited as the worst aspect of the perioperative experience. Thirty-five per cent of patients reported severe discomfort in at least one domain: thirst (18.5%; 95% CI 17.8-19.1), surgical pain (11.0%; 10.5-11.5) and drowsiness (10.1%; 9.6-10.5) were most common. Despite this, only 5% reported dissatisfaction with any aspect of anaesthesia-related care. Regional anaesthesia was associated with a reduced burden of side-effects. The incidence of reported AAGA was one in 800 general anaesthetics (0.12%). Conclusions Anxiety and discomfort after surgery are common; despite this, satisfaction with anaesthesia care in the UK is high. The inconsistent relationship between patient-reported outcome, patient experience and patient satisfaction supports using all three of these domains to provide a comprehensive assessment of the quality of anaesthesia care.
**BACKGROUND:**
Models of propofol pharmacokinetics and pharmacodynamics developed in patients without brain pathology are widely used for target-controlled infusion (TCI) during brain tumour excision operations. The goal of this study was to determine if the presence of a frontal brain tumour influences propofol pharmacokinetics and pharmacodynamics and existing PK-PD model performance.

**METHODS:**
Twenty patients with a frontal brain tumour and 20 control patients received a propofol infusion to achieve an induction-emergence-induction anaesthetic sequence. Propofol plasma concentration was measured every 4 min and at each transition of the conscious state. Bispectral index (BIS) values were continuously recorded. We used non-linear mixed-effects modelling to analyse the effects of the presence of a brain tumour on the pharmacokinetics and pharmacodynamics of propofol. Subsequently we calculated the predictive performance of Marsh, Schnider, and Eleveld models in terms of median prediction error (MdPE) and median absolute prediction error (MdAPE).

**RESULTS:**
Patients with brain tumours showed 40% higher propofol clearance than control patients. Performance of the Schnider model (MdPEpk -20.0%, MdAPEpk 23.4%) and Eleveld volunteer model (MdPEpk -8.58%, MdAPEpk 21.6%) were good. The Marsh model performed less well (MdPEpk -14.3%, MdAPEpk 41.4%), as did the Eleveld patient model (MdPEpk -30.8%, MdAPEpk 32.1%).

**CONCLUSIONS:**
Brain tumours might alter the pharmacokinetics of propofol. Caution should be exerted when using propofol TCI in patients with frontal brain tumours due to higher clearance.