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Contents

Comparative study on incidence of airway related complications while using polyvinyl chloride tubes with a strategic cuff release maneuver *versus* armoured flexometallic tubes endotracheal tubes in neurosurgical patients undergoing surgery in lateral position

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69

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INTRODUCTION

Prolonged surgical duration and extreme neck positions increase the complexity, raising the risk of airway compromise[1] in neurosurgical patients. Endotracheal tube (ETT) choice is pivotal, with conventional polyvinyl chloride (PVC) tubes prone to kinking[2] and flexometallic tubes (FMT) introducing their own challenges.[3] Tube biting, especially during motor evoked potential monitoring, and the need for postoperative tube exchanges from flexometallic to PVC tubes in patients requiring postoperative mechanical ventilation, brings its own set of complications. Recognizing these issues, a strategic cuff release maneuver was proposed for PVC tubes to mitigate kinking. The hypothesis posited that this maneuver would prevent kinking and twisting, with a noninferiority clinical trial conducted to validate this approach. We compared incidence of intraoperative kinking and related complications. Other measurables were airway, respiratory and hemodynamic complications in post-operative period and length of stay in the intensive care unit (ICU) and hospital.

METHODS

A single-centre prospective randomized controlled trial, approved by institute ethics committee, and registered in CTRI, recruited American Society of Anesthesiologists Physical Status I/II patients (aged 18–65) undergoing elective neurosurgeries in lateral position. Patients were randomized into Group PVC (PVC tubes) or Group FMT (armoured FMT), both using STERIMED® high-volume, low-pressure cuffs for intubation. Group PVC incorporated a strategic cuff release prior to the final neck positioning (neck flexion, lateral tilt, and lateral rotation) following the 3-pin application on the patient's head. The ET cuff was temporarily deflated to facilitate free movement of the distal end of the ET. Head was secured in the Mayfield skull clamp upon achieving the optimum head position for surgery. The ET cuff was reinflated to targeted pressure of 25–30 cm water.

During the intraoperative period, patients were monitored for any signs of ET kinking, which included an abrupt rise in peak airway pressure, expiratory tidal volume changes,

desaturation, or the appearance of a characteristic end-tidal carbon dioxide ($EtCO_2$) graph. A backup action plan was kept ready for kinking events.

Postoperative care and extubation were based on patient condition and protocol, documenting hemodynamic and respiratory complications during tube exchange and postoperative airway issues and other complications during ICU stay.

RESULTS

A total of 70 patients recruited with 35 patients in each group. Both groups had comparable baseline characteristics. No tube kinking signs were observed during surgery in any of the groups. Postoperative ventilation need matched at 71% for both groups. Flexometallic tubes patients underwent exchange with PVC tube after completion of surgery inside OT. This was associated with desaturation in three patients, airway injury in one patient. Tube exchange was also associated with hypertension (72%), hypotension (4%), and tachycardia in 80% of cases in FMT group requiring postoperative ventilation. Patients in FMT group had higher postextubation sore throat (66% *vs* 34%; *P* = 0.009) and hoarseness (57% *vs* 29%; *P* = 0.016) than the PVC group. Intensive care unit stay, and hospital stay was comparable in both the groups.

DISCUSSION

Polyvinyl chloride tubes with a strategic cuff release manoeuvre displayed remarkable safety, lacking any kinking issues during challenging neck positions for neurosurgical patients. We conclude that PVC tube with this maneuver are not inferior but match and even excel beyond flexometallic tubes, potentially reducing postoperative airway complications in neurosurgical patients undergoing surgery in lateral position.

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Disruption of somatosensory network connectivity in patients with supratentorial gliomas during mild sedation with midazolam

Submission ID

77

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INTRODUCTION

Midazolam can induce transient motor deficits in patients with supratentorial gliomas. This effect can be reversed by the specific antagonist flumazenil.[1] Brain imaging studies indicate that midazolam induced light sedation preserves or increases lower-level functional networks connectivity, such as the somatosensory network.[2,3] Nevertheless, these findings were in healthy brains. Patients with gliomas may have brain network reorganization to adapt and compensate for the pathological state.[4] Therefore, it is unclear how the brain networks of patients with gliomas reacts during mild sedation considering the effects of both the tumor and drug. The object of this study was to examine the changes in sensorimotor within-networks and internetwork functional connectivity before and after midazolam mild sedation in patients with motor cortex gliomas.

METHODS

Thirty-six subjects, 20 glioma patients, 16 healthy volunteers, were enrolled. All met the inclusion criteria and signed informed consent. The study was approved by IRB at Beijing Tiantan Hospital (No. KY2018-050-02) and ClinicalTrials.gov (NCT81701038).

Upon entry into the MRI operating room, peripheral intravenous access was established, along with routine monitoring (ECG, BP, SpO_2 end-tidal CO_2) and oxygen supplementation. Initial structural and resting state functional magnetic resonance imaging (rs-fMRI) scans were performed. Then, participants were given intravenous midazolam starting at 0.03 $mg \cdot kg^{-1}$ and titrated to mild sedation, OAA/S = 4. A rs-fMRI scan was repeated during sedation.

MRI data acquisition was performed on a Siemens 3.0-Tesla Verio scanner. Independent Component Analysis[5] was used to identify functional connectivity within the sensorimotor network. Regions of interest were extracted for internetwork functional connectivity comparison.

Continuous variables were analyzed by independent sample *t* tests. Categorical variables were subjected to Chi square tests. The General Linear Model was applied for image comparison, with reported functional connectivity results based on an uncorrected voxel-wise height threshold of $P < 0.001$, complemented by a false discovery rate-corrected cluster-wise threshold of $P < 0.05$.

RESULTS

Two glioma patients were excluded due to over-sized tumors and excessive head movement. Therefore 18 glioma patients and 16 healthy volunteers were used for data analysis.

Compared to the healthy controls, the sensorimotor network (SMN) connectivity was reduced in glioma group before sedation. After mild sedation, the SMN intra-network connectivity was intact in healthy controls, by contrast, it was disrupted in the glioma group as there was increased connectivity in precentral and postcentral gyrus regions within SMN (Voxel, P[unexpected] < 0.005; Cluster, P[FDR] < 0.05).

For the internetwork connectivity, the SMN-right supramarginal gyrus connection in the glioma group was increased during awake state but was replaced by SMN-precuneous and SMN-anterior cingulate increases during mild sedation (Voxel, P[unexpected] < 0.001; Cluster size > 20). These changes were not seen in healthy control.

DISCUSSION

Glioma patients' brain network alterations are complex and extensive, involving reduced long-distance connections and changes in cross-hemispheric connections. Brain reorganization may allow compensation for some of these impairments. The glioma patients exhibited disruptions within intra- and inter- network connectivities. Surgery and sedative drugs may disrupt network compensation, leading to the re-emergence of (pre-existing) neurofunctional impairments. During the “double attack” from midazolam and the glioma, the pattern of intra- and inter-network connectivity were generally disrupted, which indicated that some of the network compensation became maladaptive. This may contribute to the reversible clinically unmasked focal neurological deficit phenomenon with sedation.

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Effect of hypotension on adverse outcome(s) in patients with moderate to severe traumatic brain injury: systematic review and meta-analysis

Submission ID

108

AUTHORS

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INTRODUCTION

Traumatic brain injury (TBI) is a leading cause of death and disability worldwide. The estimated incidence of TBI is approximately 69 million per year with a disability rate of 111 per 100,000 individuals.[1,2] Using a systolic blood pressure (SBP) of ≤ 90 mm Hg threshold, studies have shown that hypotension as a secondary injury in TBI patients is associated with poorer outcomes.[3] To reflect this, management guidelines from the Brain Trauma Foundation advises maintaining a SBP of ≤ 100 mm Hg for TBI patients 50 to 69 yr of age or ≤ 110 mm Hg for other age groups.[4] Currently, there lacks a comprehensive review examining the effect of hypotension on TBI patient outcomes. Thus, this systematic review aims to present pooled results of available literature and provide insight into the impact of hypotension on adverse outcomes in the moderate to severe TBI setting.

METHODS

This study protocol has been registered in the PROSPERO registry. A literature search of studies examining the outcomes of moderate to severe TBI patients with hypotension was conducted using MEDLINE, Embase, Cochrane Central Register of Controlled Trials, Cochrane Database of Systematic Reviews, CINAHL, and Web of Science, and Scopus. Eligibility criteria included: moderate to severe TBI patients based on Glasgow Coma Scale or abbreviated injury scale scale, reported adverse outcomes, and age ≥ 10. Exclusion criteria include studies examining only mild TBI, case series, case reports, and reviews. Independent reviewers will conduct initial title and abstract screening followed by full-text review for eligibility using Covidence software. Data extraction of study characteristics, patient characteristics, outcomes, and quality of study

will be conducted using an independent form. The primary outcome of interest was adverse outcomes (death and/or vegetative state) following hypotension in TBI patients within six months. Secondary outcomes included incidence of hypotension, and adverse outcomes in association with patient characteristics such as age, TBI severity, etc. Forest plots, odds ratios, and incidence with 95% confidence intervals were generated. Subgroup analyses, meta-regression, and sensitivity analysis were performed to assess publication bias and heterogeneity.

RESULTS

The search strategy identified 16,720 records with 54 studies meeting eligibility criteria consisting of 363,820 patients. Pooled analysis demonstrated an increased risk of mortality in hypotensive TBI patients (odds ratio [OR], 2.17; 95% confidence interval [CI], 1.93 to 2.43). When only assessing studies with a hypotension threshold of SBP ≤ 90 mm Hg (35 studies), the risk of mortality was higher (OR, 2.49; 95% CI, 2.07 to 3.00). Further subgroup analysis was performed to assess risk of mortality in relation to TBI severity, hypoxia adjustment, location of blood pressure measurement, TBI classification, and various hypotension blood pressure thresholds. For secondary outcome, pooled analysis showed an overall incidence of hypotension of 17.95% (95% CI, 16.52 to 19.37). Incidence was further subcategorized and assessed based on various hypotension blood pressure thresholds, where the incidence of hypotension was 15.24% (95% CI, 14.04 to 16.43) in studies utilizing SBP ≤ 90 mm Hg as threshold.

DISCUSSION

This systematic review presents data of nearly 370,000 patients which empirically demonstrate the increased risk of mortality in TBI patients that develop hypotension. Our results show that the risk of adverse outcomes is true regardless of the TBI scale used (AIS *vs* GCS), and at what location the hypotension was determined (ED *vs* EMS). Furthermore, our results demonstrate the high incidence of hypotension in moderate to severe TBI patients. This systematic review can be served to further support current guidelines and recommendations to maintain blood pressure control above 90 mm Hg when providing care to patients with TBI.

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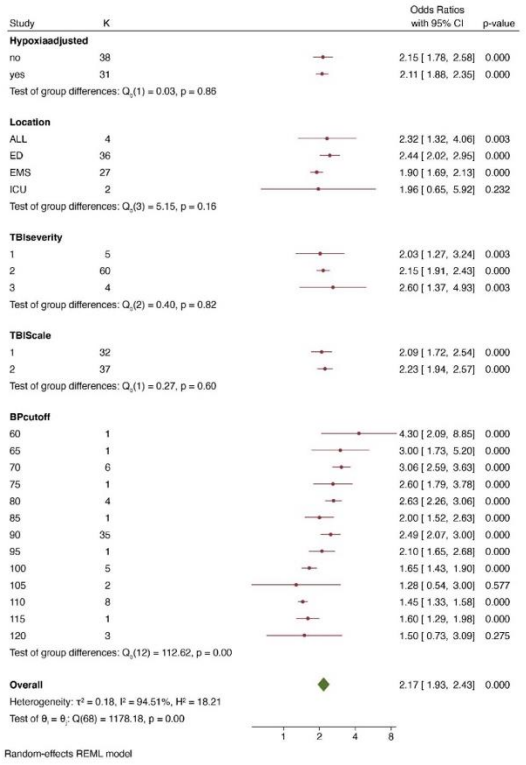
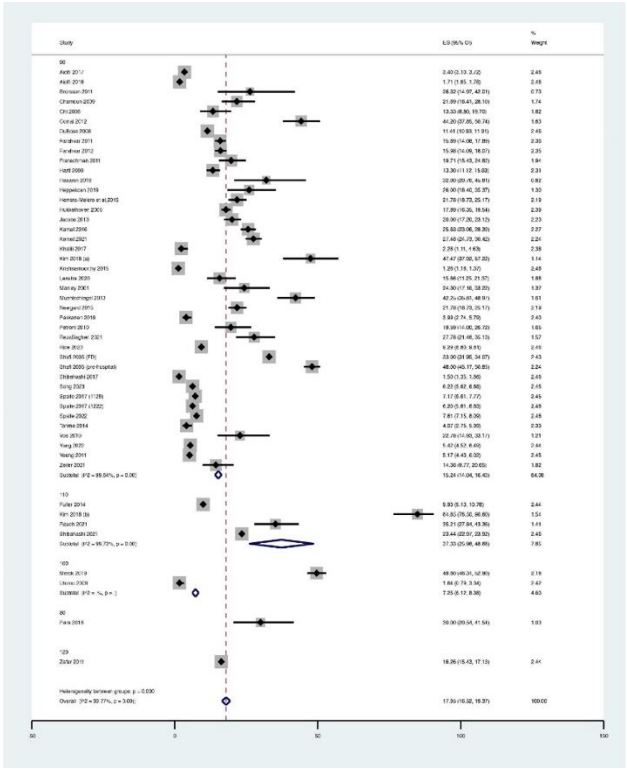
Figure

a)



b)



Management of idiopathic intracranial hypertension in a young patient with severe class 3 obesity

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23

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INTRODUCTION

Idiopathic intracranial hypertension (IIH) is a condition of increased intracranial pressure (ICP) without structural cause seen on conventional imaging.[1,2] Patients with IIH can present with headache, papilledema, visual disturbance, and sixth cranial nerve palsy.[1,2] IIH has been strongly associated with obesity (World Health Organization [WHO] classification body mass index [BMI] > 30),[1] which is correlated with increased morbidity and mortality.[3,4] Initial treatment in IIH includes weight loss and medical management with carbonic anhydrase inhibitors such as acetazolamide, which work by decreasing cerebrospinal fluid (CSF) production. Cerebrospinal fluid shunting procedures are another option for treatment.[2] Venous sinus stenting is usually reserved for patients with refractory IIH and papilledema despite maximal medical treatment.[2]

We report a young male patient with severe class 3 obesity (BMI, 107) with severe bilateral transverse sinus stenosis for stenting under general anesthesia. Patient was seen in preoperative clinic for better planning and arrangement of extra personnel and equipment.

CASE PRESENTATION

A 23-yr-old male patient (weight, 286 kg; height, 163 cm; BMI, 107) presented with history of refractory IIH for five years. Airway examination showed a good mouth opening (Mallampati score 3), good thyromental distance (> 6 cm), normal upper lip bite test (class 1), and normal neck movement. After written informed consent was obtained, the patient positioned himself on the interventional radiology table to minimize risk to patient and staff.[4] A troop pillow with extra blankets to augment ramped position[5] were used, with extra arm boards to support the pannus (Figure A). Standard monitors, a pre-induction radial arterial line, and an 18G *iv* cannula were placed using ultrasound guidance.

Patient's airway was topicalized using lidocaine nebulization and 4% lidocaine spray. Loading dose of 1 $\mu g \cdot kg^{-1}$ dexmedetomidine was infused over ten minutes. Patient was intubated using awake fibreoptic bronchoscopic technique with videolaryngoscope assistance. After endotracheal tube was secured, general anesthesia was induced using propofol 200 mg

and rocuronium 100 mg, and maintained using Desflurane (Et 3–6%) and Dexmedetomidine 0.3–0.6 $\mu g \cdot kg^{-1} \cdot hr^{-1}$, avoiding opioids altogether. Intraoperative hemodynamics were stable. Venous sinus pressures were high (Figure B). Unfortunately, only left transverse sinus could be stented.

At the end, Sugammadex 500 mg *iv* was given and Desflurane was discontinued. Patient was extubated fully awake, and he remained stable and pain-free postoperatively. Nevertheless, we needed 15 personnel to transfer the patient onto the special expandable bed (weight rating, 454 kg) (Figure C). At three months, he reported much improvement in headache and vision.

CONCLUSION

Super-morbid obesity poses multifaceted challenges for the anesthesiologist. Our patient's body habitus and high BMI (107) precluded simple procedures like computed tomography scanning, and also dictated management as a difficult airway. Coordinated planning through preoperative clinic visit was crucial to successful management, with two anesthesiologists, and extra personnel and equipment transfers involved in out-of-operating-room procedures, and anticipation of potential complications. Weight rating of beds must be checked before perioperative use. Ultrasound guidance proved extremely helpful for vascular access. Dexmedetomidine appears to be a safe choice as an anesthetic adjuvant in such patients, providing anxiolysis for awake intubation, and reducing opioid and anesthetic consumption perioperatively.

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Figure A, B, C

Venous sinus pressures	Baseline	Post-stent
Right lateral transverse sinus	57	55
Right mid-transverse sinus	53	56
Right medial transverse sinus	56	56
Torcula	53	57
Left lateral transverse sinus	52	57
Left mid-transverse sinus	52	58
Left medial transverse sinus	52	58
Left superior sigmoid sinus	51	56
Left inferior sigmoid sinus	35	38
Left internal jugular vein	33	38

(B)

(C)

(A)