Article

Effects of Dexmedetomidine on Cognitive Function, Oxidative Stress and Brain Protection in Patients Undergoing Craniocerebral Surgery

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Abstract

Background: The protective mechanism of dexmedetomidine on the brains of patients undergoing craniocerebral surgery remains unclear. The aim of this study was to examine the impact of dexmedetomidine on cognitive function, oxidative stress, and brain protection in such patients.

Methods: Fifty-four patients who underwent craniocerebral surgery at our hospital from January 2020 to June 2023 were retrospectively selected as study subjects. They were divided into two groups: the control group (n = 27) and the study group (n = 27), based on different auxiliary anesthesia protocols. Patients in the study group received dexmedetomidine before anesthesia induction, using a midline intravenous pump to assist anesthesia, while the control group received an equivalent amount of normal saline. The remaining anesthesia induction and maintenance protocols were consistent for both groups. Cognitive function was assessed using the Mini Mental State Examination (MMSE) before and 1 day after surgery for both groups. Oxidative stress indicators, including malondialdehyde (MDA), glutathione peroxidase (GSH-Px), and superoxide dismutase (SOD) levels in the serum of both groups, were measured using enzyme-linked immunosorbent assay (ELISA). Additionally, changes in postoperative brain injury indicators, namely neuron-specific enolase (NSE) and central nervous system-specific protein (S100β), were detected and compared in the serum of both groups. Concurrently, postoperative adverse reactions were recorded for both groups.

Results: The MMSE scale scores of both groups of patients 24 hours after surgery were significantly lower than those before surgery. However, the MMSE scale scores of the study group patients were notably higher than those in the control group, with a statistically significant difference ($p < 0.05$). One hour after surgery, the serum levels of MDA, GSH-Px, and SOD in both groups of patients were significantly elevated compared to pre-surgery levels. Yet, the study group exhibited significantly lower levels of MDA, GSH-Px, and SOD in comparison to the control group, and these differences were statistically significant ($p < 0.05$). The serum levels of NSE and S100β in both groups were markedly higher than preoperative levels 24 hours after surgery. However, the study group demonstrated significantly lower levels of serum NSE and S100β compared to the control group, with a statistically significant difference ($p < 0.05$). The incidence of postoperative complications in the study group was 7.41% (2/27), indicating a decreasing trend compared to 18.52% (5/27) in the control group. However, this difference did not reach statistical significance ($\chi^2 = 1.477$, $p = 0.224$).

Conclusion: Dexmedetomidine-assisted anesthesia in craniocerebral surgery can effectively enhance postoperative cognitive function, mitigate oxidative stress, and facilitate overall postoperative recovery for patients. The intervention exhibits a favorable safety profile with no reported serious adverse reactions, establishing it as a relatively safe and reliable approach.

Keywords

dexmedetomidine; cranial surgery; cognitive function; oxidative stress; brain protection

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Introduction

Head injury stands as a prevalent critical condition in clinical practice, representing a major contributor to both mortality and disability [1]. Recent advancements in cranial surgery technology have led to continuous development and improvement [2]. Procedures such as intracerebral aneurysm clipping have become routine in neurosurgical practice. However, for patients with craniocerebral injuries undergoing surgical interventions, the associated stimulation and interference may precipitate complications such as brain edema, ischemia, cell death, and intracranial hematoma [3,4]. Additionally, cranial surgery can elevate intracranial pressure, thereby impacting tissue perfusion and diminishing the brain's compliance [5].

Therefore, prioritizing brain protection during surgical anesthesia holds the potential to ameliorate intraoperative ischemia and prevent damage to hypoxic brain tissue. Preserving the normal metabolic function of brain tissue has become a focal point in craniocerebral surgery. Since the 1940s, numerous studies have affirmed the brain-protective effects of both intravenous and inhaled anesthetics. Presently, within the realm of neurosurgical anesthesia, efforts are underway to identify drugs with neuroprotective properties to mitigate the development of ischemic-hypoxic brain injury.

Dexmedetomidine, a relatively recent sedative drug utilized in clinical practice, emerges as a promising candidate. By activating α_2 adrenergic receptors, dexmedetomidine demonstrates anti-inflammatory and antioxidant effects [6,7]. Several studies have indicated that dexmedetomidine contributes to stabilizing blood circulation, safeguarding brain function, maintaining intracranial pressure stability, reducing cerebral oxygen metabolism, and providing analgesia and sedation. This renders it suitable for anesthesia during craniocerebral surgery [8–10]. Nevertheless, the precise mechanism underlying dexmedetomidine's brain-protective effects in patients undergoing craniocerebral surgery remains unclear, and there is insufficient evidence supporting its application in brain protection during such procedures.

Building upon prior research, this study selected 54 patients with craniocerebral injuries as subjects to analyze the impact of dexmedetomidine on cognitive function, oxidative stress response, and neurological function in craniocerebral surgery patients. The aim is to explore the brain-protective function of dexmedetomidine and provide valuable insights for the selection of clinical neurosurgical adjuvant drugs.

Materials and Methods

Research Subjects

Patients who underwent craniocerebral surgery at our hospital between January 2020 and June 2023 were retrospectively selected for analysis. After comprehensive clinical data assessment, 54 cases were ultimately included in the study. These participants were then categorized into a control group and a study group based on different adjuvant drug anesthesia protocols.

In the study group, patients received intravenous pump infusion of dexmedetomidine to assist anesthesia before the induction of anesthesia, while the control group received an equivalent amount of normal saline. The remaining components of the anesthesia induction procedure were kept consistent. Ethical approval for this study was obtained from the Medical Ethics Committee of the First People's Hospital of Daishan (2023-14). The entire experimental process adhered to the principles of informed consent, with patients or their family members receiving comprehensive information about the study. The study was conducted in accordance with the Declaration of Helsinki.

Inclusion Criteria: ① Traumatic craniocerebral hematoma resulting from accidents such as sharp injuries, falls from heights, traffic accidents, etc. ② Meeting the indications for craniocerebral surgery. ③ American Society of Anesthesiologists (ASA) classification score of level I–II [11]. ④ Age between 18 and 60 years, with no gender restrictions. ⑤ Glasgow Coma Score (GCS) ranging from 9 to 12 points [12]. ⑥ Absence of brain herniation. ⑦ Duration of coma after injury within the range of 30 minutes to 6 hours.

Exclusion Criteria: ① Individuals with a history of prior brain injury or craniocerebral surgery. ② Patients who have engaged in prolonged smoking, alcohol consumption, or drug use before the operation. ③ Individuals with pre-existing coronary heart disease, blood system disorders, or infectious diseases prior to surgery. ④ Patients with severe dysfunction in other organs. ⑤ Individuals undergoing long-term use of hormones or immunomodulators.

Methods

Anesthesia Method

The patient observed an 8-hour fasting period and refrained from drinking for 6 hours before surgery. Upon entering the operating room, a consistent team of surgeons and

anesthetists conducted routine preoperative preparations. Vital signs, non-invasive blood pressure, pulse oximetry, electrocardiogram, and electroencephalogram (EEG) bispectral index (BIS) were monitored, and peripheral venous access was established.

Based on the review of clinical data, it was noted that the adjuvant drug anesthesia protocol differed. Ten minutes before tracheal intubation, the study group received an infusion of 1 μg/kg of dexmedetomidine diluted in 100 mL of 0.9% sodium chloride injection (H20183220, Yangzijiang Pharmaceutical Group Co., Ltd., Taizhou, China). The infusion continued at a rate of 0.5 μg/kg/h until skin is sutured. In contrast, the control group received an equivalent volume of normal saline without the addition of any other drugs.

The administration of anesthesia induction drugs follows with the induction protocol as outlined below:

Anesthesia Induction: (1) Etomidate (H20180511, Jiangsu Enhua Pharmaceutical, Xuzhou, China); (2) Sufentanil (H20184171, Yichang Renfu Pharmaceutical, Yichang, China); (3) Cisatracurium (H20180869, Shanghai Hengrui Pharmaceutical Co., Ltd., Shanghai, China).

The dosage sequence for induction is 0.3 mg/kg/h, 0.4 μg/kg/h, and 0.15 mg/kg/h, respectively. Once the eyelash reflex disappeared, a laryngeal mask was inserted for mechanical ventilation and tracheal intubation. Mechanical ventilation parameters were set as follows: tidal volume (VT) = 8~10 mL/kg, ventilation frequency (RR) = 12~14 times/min, maintaining end-tidal carbon dioxide partial pressure (PETCO2) at 35~40 mmHg.

For anesthesia maintenance, the following drugs were utilized: (1) Propofol (H20183360, AstraZeneca Pharmaceutical Co., Ltd., Wuxi, China); (2) Remifentanil (H20180197, Yichang Renfu Pharmaceutical Co., Ltd., Yichang, China).

Continuous intravenous pump infusion rates were set at 6 mg/kg/h and 0.3 mg/kg/h, respectively. Additionally, intermittent intravenous bolus injections of 0.25 mg/kg/h cisatracurium (H20180869, Shanghai Hengrui Pharmaceutical Co., Ltd., Shanghai, China) were administered. Mechanical ventilation parameters for this phase were maintained at $P_{ET}CO_2$ = 35 mmHg, inspired oxygen concentration (FiO_2) = 1.0, and SPO_2 >98%.

Observation Indicators

Cognitive Function Level Testing

The cognitive level of both groups was evaluated using the Mini Mental State Examination (MMSE) before surgery and 1 day after the surgical procedure. MMSE serves as a tool for assessing cognitive function [13]. The scoring content encompasses various cognitive domains, including memory, orientation, attention, language, and visual-spatial abilities. The total score ranges from 0 to 30 points, with a lower score indicating better cognitive function, and a higher score suggesting more severe cognitive dysfunction.

Oxidative Stress Indicator Level Detection

A fasting peripheral venous blood sample of 5 mL was collected from all study subjects before surgery and 1 hour after surgery. The samples were centrifuged at 3000 r/min, with a centrifugal radius of 10 cm, for 15 minutes. The upper serum was then collected and stored for testing. The enzyme-linked immunosorbent assay (ELISA) method, primarily utilized for detecting soluble antigens or antibodies, involves binding these components to the surface of a specific solid-phase carrier. This method employs the specific interaction between antigens and antibodies to initiate immune reactions, enabling qualitative or quantitative analysis based on color intensity.

For the experiment, the separated serum and ELISA kit were utilized to measure the concentrations of malondialdehyde (MDA) (EEA015, Invitrogen, Carlsbad, CA, USA), glutathione peroxidase (GSH-Px) (EEA010, Invitrogen, Carlsbad, CA, USA), and superoxide dismutase (SOD) (EIASODC, Invitrogen, Carlsbad, CA, USA), following the kit instructions. After setting up each group of plate wells, 25 μL of sample and calibrator were added to each well, covered with film, and incubated for 30 minutes. Subsequently, 250 μL of enzyme-labeled antibody working solution and substrate were added to each well, mixed by pipetting, and incubated at 37 °C for 60 minutes. The reaction was stopped after sufficient incubation, and the microplate reader's wavelength was set to 510 nm for detection, measuring the absorbance (OD) value. A standard curve was established, and sample concentrations were calculated accordingly.

Brain Damage Indicator Level Detection

Five mL of fasting peripheral venous blood was collected from all study subjects before surgery and 1 day after surgery. The collected blood samples were then centrifuged at 3000 r/min with a 10 cm radius for 15 minutes. The upper serum was collected and stored for testing. To ensure accuracy, the ELISA kit and serum were equilibrated to room temperature before measurement. ELISA was employed to measure the expression levels of neuron-specific enolase (NSE) and central nervous system-specific protein (S100β) in the serum. The levels of NSE and S100β in the serum were determined following the instructions of the ELISA kit for NSE (EEL040, Invitrogen, Carlsbad, CA, USA) and S100β (EEL109, Invitrogen, Carlsbad, CA, USA). All procedures were strictly conducted in accordance with the instructions provided with the ELISA kit.

Occurrence of Adverse Reactions

The occurrence of postoperative adverse reactions in both groups was diligently documented, and appropriate treatment measures were promptly administered. Common adverse reactions included nausea, vomiting, skin itching, choking, agitation, and convulsions. The frequency of these adverse reactions was calculated and subsequently compared between the two groups.

Statistical Analysis

Statistical analysis of the collected data was conducted using SPSS 23.0 software (IBM, Armonk, NY, USA). Normally distributed measurement data were presented as mean $\pm$ standard deviation ($\bar{x} \pm$ s), the independent sample *t*-test was employed for data comparison. Count data were expressed as [n (%)], and the χ^2 test was utilized for data comparison. A significance level of $p < 0.05$ was considered to indicate statistical significance.

Results

Comparison of General Information between the Two Groups of Patients

No statistically significant differences were observed in general information, including gender, age, body mass index, time to hospital after injury, ASA classification, and GCS score between the two groups of patients ($p > 0.05$). Refer to Table 1 for details.

Comparison of Cognitive Function Scores between the Two Groups of Patients

The results revealed no statistically significant difference in the MMSE scale scores between the two groups before surgery ($p > 0.05$). However, one day after surgery, the MMSE scale scores for both groups were significantly lower than those before surgery. Notably, the MMSE scale scores of the patients in the study group were significantly higher than those in the control group, with a statistically significant difference ($p < 0.05$). Refer to Table 2 for detailed information.

Comparison of Oxidative Stress Indicators between the Two Groups of Patients

The test results of the emergency index levels for both groups indicated no statistically significant difference in the serum levels of MDA, GSH-Px, and SOD before surgery ($p > 0.05$). However, one hour after surgery, the serum levels of MDA, GSH-Px, and SOD in both groups of patients were significantly higher than those before surgery. Notably, the serum levels of MDA, GSH-Px, and SOD in the study group were significantly lower than those in the control group, and these differences were statistically significant ($p < 0.05$). Refer to Table 3 for detailed results.

Comparison of Brain Injury Indicators between Two Groups of Patients

The results demonstrated that before surgery, there was no statistically significant difference in the serum levels of NSE and S100β between the two groups of patients ($p > 0.05$). However, one day after surgery, the serum levels of NSE and S100β for both groups were significantly higher than those before surgery. Importantly, the serum levels of NSE and S100β in the study group were significantly lower than those in the control group, and this difference was statistically significant ($p < 0.05$). Refer to Table 4 for detailed information.

Comparison of Postoperative Complication Rates between the Two Groups of Patients

After surgery, patients in both groups encountered diverse adverse reactions, including nausea and vomiting, skin itching, coughing, agitation, and convulsions. However, these reactions were effectively alleviated or resolved with symptomatic treatment. The incidence of postoperative complications in the study group was 7.41% (2/27), indicating a declining trend compared to 18.52% (5/27) in the

Table 1. Comparison of general information and clinical characteristics of the two groups of patients [$\bar{x} \pm$ s, *n* (%)].

Items		Control group ($n = 27$)	Study group ($n = 27$)	χ^2/t-value	p-value
Gender	Male	15 (55.56)	17 (62.96)	0.307	0.580
	Female	12 (44.44)	10 (37.04)		
Age (years)		44.59 ± 4.54	45.42 ± 5.18	0.626	0.534
Body mass index (kg/m^2)		24.85 ± 2.68	24.78 ± 2.56	0.098	0.922
Time to hospital after injury (h)		3.24 ± 0.53	3.42 ± 0.62	1.147	0.257
ASA classification	I	8 (29.63)	9 (33.33)	0.086	0.770
	II	19 (70.37)	18 (66.67)		
GCS score		10.12 ± 1.03	10.14 ± 1.01	0.072	0.943

Note: ASA, American Society of Anesthesiologists classification score; GCS, Glasgow Coma Score.

Table 2. Comparison of MMSE scale scores between the two groups of patients ($\bar{x} \pm$ s, score).

Groups	Patients	Preoperative	1 d after surgery
Control group	27	28.26 ± 2.95	23.16 ± 2.25*
Study group	27	28.18 ± 2.87	25.49 ± 2.34*
t-value		0.101	3.730
p-value		0.920	<0.001

Note: MMSE, Mini Mental State Examination; Compared with the same group of patients before operation, *$p < 0.05$.

control group. Nonetheless, the difference was not statistically significant ($\chi^2 = 1.477$, $p = 0.224$). Refer to Table 5 for detailed information.

Discussion

Under normal circumstances, the brain maintains the stability of cerebral blood flow through its inherent vascular resistance mechanism, ensuring a balance between cerebral oxygen supply and demand under typical physiological conditions [14]. However, head injury can disrupt this cerebrovascular resistance mechanism, leading to an imbalance in cerebral oxygen supply and demand [15].

Craniocerebral injury represents a common acute and critical condition in neurosurgery, characterized by sudden onset, rapid disease progression, and high mortality. Emergency surgery is often required to save the patient's life. Craniocerebral surgery carries the inherent risk of cerebral ischemia. Throughout the procedure, hypoxic-ischemic damage to the brain can significantly impact surgical outcomes and recovery [16]. Such damage may result in cerebral ischemia-hypoxic injury, severely impairing the patient's neurological and motor functions. This, in turn, affects the overall quality of life for patients and places an additional burden on both families and society [17].

The challenge of ensuring brain protection during craniocerebral surgery is a critical issue that demands attention and resolution. Surgical procedures, intraoperative bleeding, and intracranial pressure variations can significantly impact perioperative hemodynamics and oxidative stress responses during craniocerebral surgery, thus influencing patient prognosis [18].

Numerous studies have affirmed the brain-protective effects of dexmedetomidine. As a highly selective α_2-adrenergic receptor agonist, dexmedetomidine has emerged for its diverse potentials and is widely applied in cranial surgery [19]. In patients undergoing cranial surgery, dexmedetomidine demonstrates the ability to mitigate anesthetic drug-related neurotoxicity, reduce intracranial pressure, enhance brain metabolism, minimize brain cell apoptosis and necrosis, inhibit the expression of excitatory neurotransmitters, alleviate postoperative pain, and promote overall postoperative recovery [20,21].

This study involved 54 patients with craniocerebral injury in our hospital as study subjects. Different adjuvant anesthetic drugs were administered to the two groups of patients during the operation to observe and analyze the effects of dexmedetomidine on cognitive function and oxidative stress, with a focus on discussing its brain-protective effect.

The results demonstrated that the MMSE scale scores in both groups were significantly lower than preoperative levels, and the MMSE scale scores in the study group were significantly higher than those in the control group. This suggests that cranial surgery does have an impact on the patient's cognitive level to some extent, but the intervention of dexmedetomidine can mitigate the impact of cranial surgery on cognitive function.

The study further found that the serum levels of MDA, GSH-Px, and SOD in both groups of patients after surgery

Table 3. Comparison of oxidative stress indicators between the two groups of patients ($\bar{x} \pm s$).

Groups	Patients	MDA (mmol/L)		GSH-Px (ng/L)		SOD (ng/L)	
		Preoperative	1 h after surgery	Preoperative	1 h after surgery	Preoperative	1 h after surgery
Control group	27	4.34 ± 0.47	7.42 ± 0.71*	43.24 ± 4.86	86.57 ± 8.73*	147.12 ± 14.73	242.59 ± 21.37*
Study group	27	4.41 ± 0.54	6.57 ± 0.62*	42.78 ± 4.79	67.36 ± 6.65*	146.51 ± 13.56	204.67 ± 19.48*
t-value		0.508	4.686	0.350	9.096	0.158	6.814
p-value		0.614	<0.001	0.728	<0.001	0.875	<0.001

Note: MDA, malondialdehyde; GSH-Px, glutathione peroxidase; SOD, superoxide dismutase; Compared with the same group of patients before operation, $^*p < 0.05$.

Table 4. Comparison of brain injury indicators between the two groups of patients ($\bar{x} \pm s$).

Groups	Patients	NSE (μg/L)		S100β (ng/mL)	
		Preoperative	1 d after surgery	Preoperative	1 d after surgery
Control group	27	16.85 ± 1.55	34.17 ± 3.34*	0.39 ± 0.04	1.03 ± 0.11*
Study group	27	16.78 ± 1.46	27.28 ± 2.41*	0.41 ± 0.05	0.81 ± 0.07*
t-value		0.171	8.692	1.623	8.768
p-value		0.865	<0.001	0.111	<0.001

Note: NSE, neuron-specific enolase; S100β, central nervous system-specific protein; Compared with the same group of patients before operation, $^*p < 0.05$.

were significantly higher than before surgery, and the serum levels of MDA, GSH-Px, and SOD in the study group were significantly lower than those in the control group. This indicates that patients experience severe oxidative stress in their bodies after craniocerebral surgery. However, with dexmedetomidine-assisted anesthesia, the abnormal increase in oxidative stress factors was suppressed to a certain extent.

Craniocerebral surgery, being an invasive procedure, stimulates the body and induces oxidative stress reactions that affect brain function. Patients with craniocerebral injury exhibit abnormal increases in intracranial oxidative stress response and reduced release of antioxidant factors, resulting in an imbalance in the overall oxidative stress response in the patient's body. Simultaneously, factors contributing to brain damage exacerbate neuron damage in the secondary injury cascade process, intensifying overall brain damage.

In the application of dexmedetomidine, the α_2 receptor subtype plays a crucial role. The α_2 receptor subtype, primarily located in presynaptic and postsynaptic areas, regulates neuronal excitability and norepinephrine release, contributing to the observed neuroprotective effects of dexmedetomidine.

Dexmedetomidine can effectively bind to α_2 receptors located in the locus coeruleus of the brainstem, leading to sedative and hypnotic effects. Furthermore, its binding to α_2 receptors in the dorsal horn of the spinal cord inhibits neurotransmitter release and the transmission of pain signals [22].

The study observed varying degrees of adverse reactions such as nausea and vomiting, skin itching, coughing, agitation, and convulsions in both groups of patients after surgery. However, these reactions were effectively mitigated or resolved with symptomatic treatment. It has been identified in studies that dexmedetomidine, during its action, activates postsynaptic membrane receptors, leading to the inhibition of sympathetic nerve activity. This results in a reduction in heart rate and blood pressure, alleviating patient anxiety and achieving sedative effects [23].

Currently, dexmedetomidine is extensively utilized in neurosurgery and has evolved into a pivotal sedative drug in this field [24,25]. Dexmedetomidine acts on the subcortex, reducing the release of histamine from cortical and subcortical projection systems, without compromising neurological function, thus exhibiting neuroprotective effects [26].

The study revealed that the serum S100β levels in both groups of patients after surgery were significantly higher than before surgery. Notably, compared with the control group, patients in the study group exhibited a significant reduction in serum S100β levels. This suggests that dexmedetomidine has a protective effect on brain tissue damage. S100β, a calcium-sensitive protein, can be released into cerebrospinal fluid or peripheral blood during

Table 5. Comparison of the incidence of postoperative adverse reactions between the two groups of patients [*n* (%)].

Groups	Patients	Feel sick and vomit	Itchy skin	Choking	Restless	Convulsion	Incidence of adverse reactions
Control group	27	1 (3.70%)	0 (0%)	2 (7.41%)	1 (3.70%)	1 (3.70%)	5 (18.52%)
Study group	27	0 (0%)	0 (0%)	1 (3.70%)	1 (3.70%)	0 (0%)	2 (7.41%)
χ^2-value							1.477
p-value							0.224

the acute phase of brain injury, increasing the expression of S100β, which is a key factor in the nerve injury process [27]. Currently, S100β levels in cerebrospinal fluid and blood are considered reliable biomarkers for acute brain injury caused by cardiovascular disease or traumatic injury [28,29].

NSE is abundant in neurons, and during brain injury, the functional structure of neuronal cell membranes is damaged, leading to the release of NSE into the intercellular space and cerebrospinal fluid [30]. As NSE passes through the damaged blood-brain barrier and enters the bloodstream, its expression in the blood increases. Therefore, serum NSE levels serve as an important indicator for evaluating brain damage and are positively correlated with neuronal damage [31]. Studies on traumatic brain injury indicate that patients with traumatic brain injury exhibit higher serum NSE levels than healthy individuals, with the severity of traumatic brain injury corresponding to elevated serum NSE levels and a poorer prognosis [32]. Further analysis through logistic regression in this study showed that early elevated serum NSE levels were an independent risk factor for a poor patient prognosis. The findings of this study align with these results, indicating that serum NSE levels in both groups of patients significantly increased after surgery, suggesting a relatively serious degree of brain injury. However, compared with the control group, patients in the study group demonstrated a significant reduction in serum NSE levels, indicating that dexmedetomidine can mitigate postoperative craniocerebral injury in patients undergoing craniocerebral surgery.

Conclusion

In conclusion, the use of dexmedetomidine as an adjunctive anesthetic in craniocerebral surgery for patients with craniocerebral injury proves effective in enhancing postoperative cognitive function, reducing oxidative stress reactions in the patient's body, and facilitating postoperative recovery. Dexmedetomidine demonstrates a brain-protective effect, effectively mitigating craniocerebral injury, and is associated with minimal serious adverse reactions. The overall safety and reliability of dexmedetomidine make it a valuable and secure option in the management of craniocerebral surgery.

Availability of Data and Materials

The data used to support the findings of this study are available from the corresponding author upon request.

Author Contributions

YF and ZJ designed the research study. YF performed the research. ZJ analyzed the data. Both authors contributed to editorial changes in the manuscript. Both authors read and approved the final manuscript. Both authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

This study was approved by the Medical Ethics Committee of the First People's Hospital of Daishan (2023-14). The entire experimental procedure adhered to the principles of informed consent, with patients or their family members being provided with information about the study. The study was carried out in compliance with the Declaration of Helsinki.

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Conflict of Interest

The authors declare no conflict of interest.

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