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Effects of sevoflurane and isoflurane on acute myocardial infarction model establishment in mice

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ABSTRACT

The selection of anesthetic drugs in the preparation of an acute myocardial infarction (AMI) model is very important. We specifically focus on various effects of sevoflurane and isoflurane in a murine AMI model, which have not been previously compared. Furthermore, we evaluated success of our AMI model using following methods: echocardiography, TTC staining, and PCR testing. The results show that compared to the isoflurane group, the sevoflurane group mice had shorter anesthetic induction(66.40 ± 2.90S vs. 125.10 ± 6.30S $P < 0.0001$) and recovery times(28.00 ± 1.07S vs. 56.88 ± 4.14S, $P < 0.0001$), lower incidence of respiratory depression (0 % vs. 50.00 %, $P = 0.0325$), and more successful models (93.33 % vs. 60.00 %, $P = 0.0801$). There were no significant differences in cardiac function, infarction area(49.41 ± 4.18 % vs. 48.66 ± 3.79 %, $P = 0.5266$), or inflammatory factors in the myocardial infarction area between the two groups. Sevoflurane may therefore be a better choice for the establishment of AMI models in mice.

1. Introduction

Acute myocardial infarction (AMI) is one of the leading causes of death and disability worldwide [1,2]. Researchers typically use left anterior descending coronary artery (LAD) ligation to create myocardial infarction in animals in order to investigate its underlying molecular mechanisms and explore potential therapeutic interventions. Using a mouse model of AMI is the most common experimental animal model for studying cardiac injury and remodeling in the literature [3–5] (see Figs. 1–6).

A new rapid method of AMI wherein the animals are anesthetized with inhalation anesthetics but not ventilated is both less invasive and more efficient than holder methods, and it also improves surgical mortality [6]. Despite inhalation anesthetics sharing common characteristics [7], different inhalation anesthetics such as isoflurane and sevoflurane have many diverse physiological and pharmacological properties [8,9] that are critical for establishing a rapid AMI model.

Isoflurane is now the most common choice of inhaled anesthetic for laboratory animal experimentation [10,11]. Recently, however, sevoflurane has become the most widely used anesthetic in human cardiac surgery, since induction and recovery with sevoflurane are faster and more stable than those with other inhaled anesthetics [12]. However, the effects of different inhalation anesthetics in mouse AMI models and the evaluation of model prognoses have yet to be reported in the literature. In this study, we therefore aimed to compare the effects of sevoflurane and isoflurane in a murine AMI model in order to evaluate them against one another directly.

2. Materials and methods

2.1. Animals

Male C57BL/6J mice: *Mus musculus musculus* (8–10 weeks old) weighing 19–25 g were purchased from Hunan Slake Jingda Experimental Co., Ltd. and were kept in the Animal Experiment Center of Hunan Provincial People's Hospital (temperature: 22–24 °C, relative humidity: 40–60 %, light/dark cycle of 12 h) with free access to food and water throughout the experiment. This animal experiment was approved by the ethics committee of Hunan Provincial People's Hospital (Approval No. 2024143), and all efforts were made to minimize the number of mice used in experiments. The present study was reported in accordance with the ARRIVE 2.0 Essential 10 guidelines (https://arriveguidelines.org).

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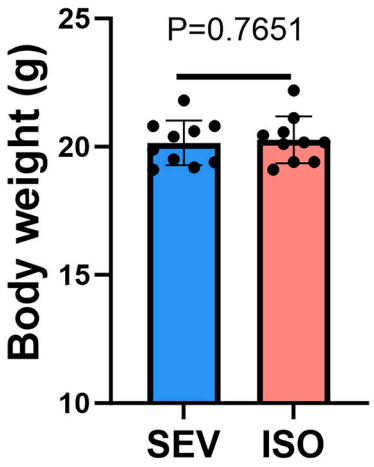

Fig. 1. No differences were observed in the weight of the mice before surgery between the SEV and ISO groups. Data (mean ± SEM) were analyzed using an unpaired *t*-test. n = 10/group.

2.2. Experimental protocol

The mice were divided into two groups (SEV and ISO). To induce AMI, the mice were subjected to left anterior descending (LAD) coronary artery ligation during anesthesia as described previously [6] and placed individually in an anesthesia induction chamber until loss of righting reflex after which time they were removed and fitted with a mask to maintain anesthesia. The SEV group was induced with 5 % sevoflurane and later maintained with 3 % sevoflurane [13] with a specific sevoflurane delivery system (RWD, Shenzhen, China), and the ISO group was induced with 5 % isoflurane and later maintained with 2 % isoflurane [7] with a specific isoflurane delivery system (Viking Medical, Medford, NJ). Air flow was 2 mL/min. Induction time, recovery time, and respiratory depression were recorded, where the induction time was defined as the time between placing them in the induction chamber with inhalation anesthetics until they lose the righting reflex [14] and the recovery time of anesthesia was defined as the time between the cessation of administration of the anesthetic and the recovery of the righting reflex [15]. Anesthesia machine calibration is completed by professional and technical personnel using specific instruments, and relevant training is carried out before the use of anesthesia machines.

2.3. AMI model establishment

As previously described [6], after being fully anesthetized as described above and receiving maintenance anesthesia in a supine position by means of a mask, each mouse's heart was promptly repositioned into the intra-thoracic compartment subsequent to ligation, and a purse-string suture was performed within a time frame of 3–5 min.

2.4. Echocardiography

Echocardiography was performed before surgery or at 1, 3, or 7 days after AMI in M-mode with an echocardiography system (VINNO6, China) that utilized an X9-22L imaging transducer as previously described [13]. The mice were induced with 2 % sevoflurane and later maintained with 1–2 % sevoflurane at a flow rate of 2 L/min, while maintaining a heart rate of 400–600 beats per min during this time. Two-dimensional images were recorded in parasternal long-axis projections with guided M-mode recordings at the midventricular level. The following variables were measured: left ventricular ejection fraction

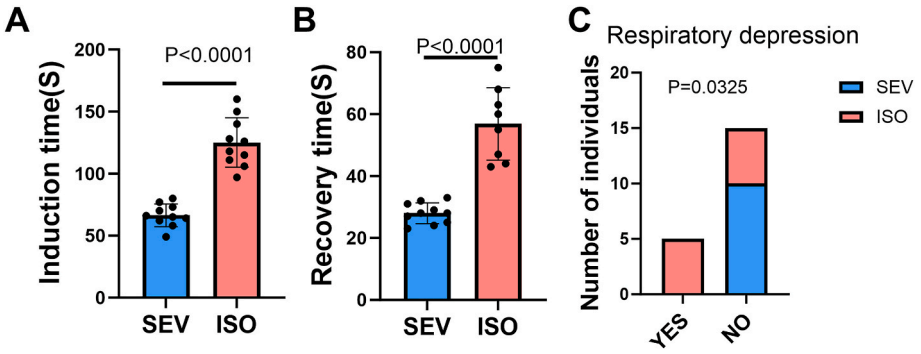

Fig. 2. Comparison of anesthesia effects between the SEV and ISO groups. **A.** comparison of induction time between the SEV (n = 10)and ISO group (n = 10). **B.** Comparison of recovery time between the SEV (n = 10) and ISO group (n = 8). **C.** Comparison of respiratory depression between the SEV (n = 10)and ISO group (n = 10). Data (mean ± SEM) in **A** and **B** were analyzed using an unpaired *t*-test. Data are presented as the mean ± SEM. Data (absolute count) in **C** were analyzed using a chi-squared test.

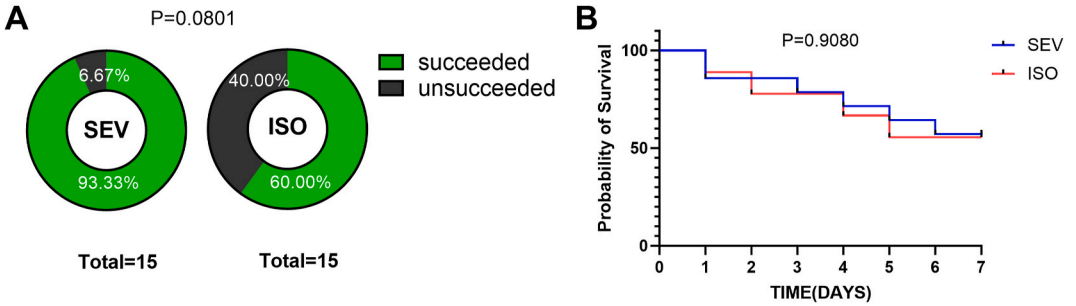

Fig. 3. Comparison of Success rate of AMI modeling and survival rate between the SEV and ISO groups. **A.** Comparison of the success rate of the AMI model between the SEV (n = 15) and ISO group (n = 15). **B.** Comparison of the survival rate between the SEV (n = 15) and ISO group (n = 15). Data (absolute count) in **A** were analyzed using a chi-squared test. Data in **B** were evaluated using a log-rank (Mantel-Cox) test.

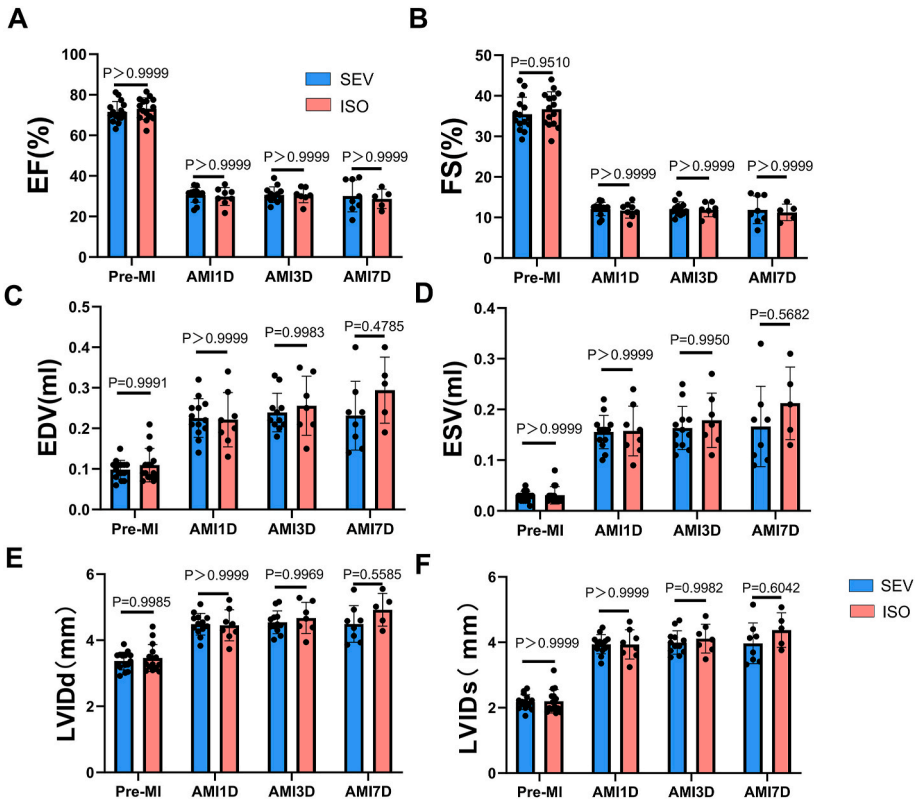

Fig. 4. Comparison of cardiac functions during the days before surgery and at 1, 3, and 7 days after AMI between the SEV and ISO group. **A.** Echocardiographic parameters of left ventricular ejection fraction (LVEF), **B.** left ventricular fractional shortening (LVFS), **C.** end-diastolic volume (EDV), **D.** end-systolic volume (ESV), **E.** left ventricular end-diastole internal diameter (LVIDd), and **F.** left ventricular end-systolic internal diameter (LVIDs) were measured on the days before surgery and 1, 3, and 7 after AMI. Sample sizes were n = 8–15 for SEV mice and n = 5–15 for ISO mice. Data are presented as the mean ± SEM, and two-way ANOVA was used to analyze repeated measurements, followed by the Bonferroni post hoc test.

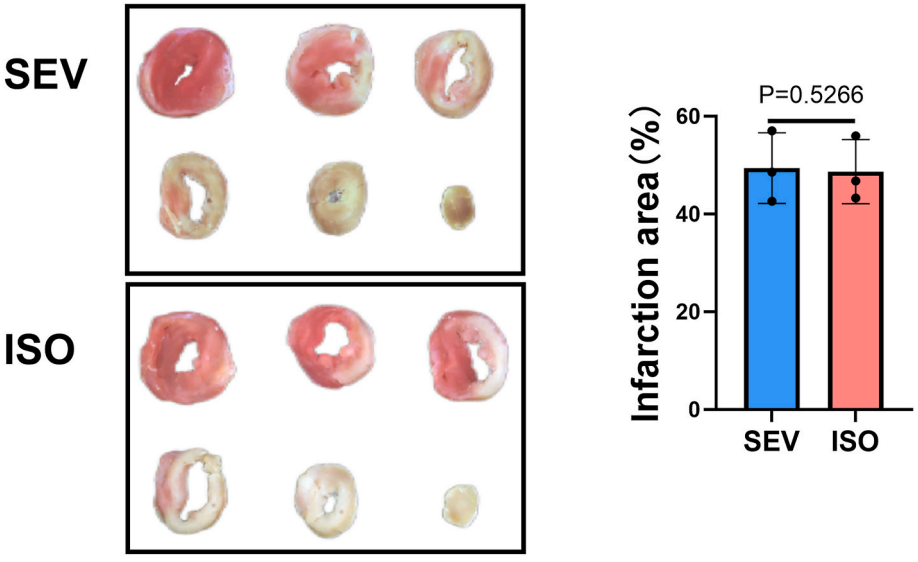

Fig. 5. Comparison of infarct areas between the SEV and ISO groups. **A.** Representative TTC image of the SEV and ISO groups. **B.** The infarction area of the SEV and ISO groups (n = 3 for SEV and n = 3 for ISO). Data (mean ± SEM) in B were analyzed using an unpaired *t*-test.

(LVEF); left ventricular fractional shortening (LVFS); left ventricular end-diastole internal diameter (LVIDd); left ventricular end-systolic internal diameter (LVIDs); end-diastolic volume (EDV); and end-systolic volume (ESV).

2.5. 2,3,5-Triphenyltetrazolium chloride (TTC) staining

To evaluate the extent of myocardial infarction, TTC staining was conducted after the mice were euthanized by a lethal intraperitoneal injection of sodium pentobarbital (50 mg/kg). Subsequently, their hearts were harvested and frozen for 10 min at −80 °C in a freezer. Slices

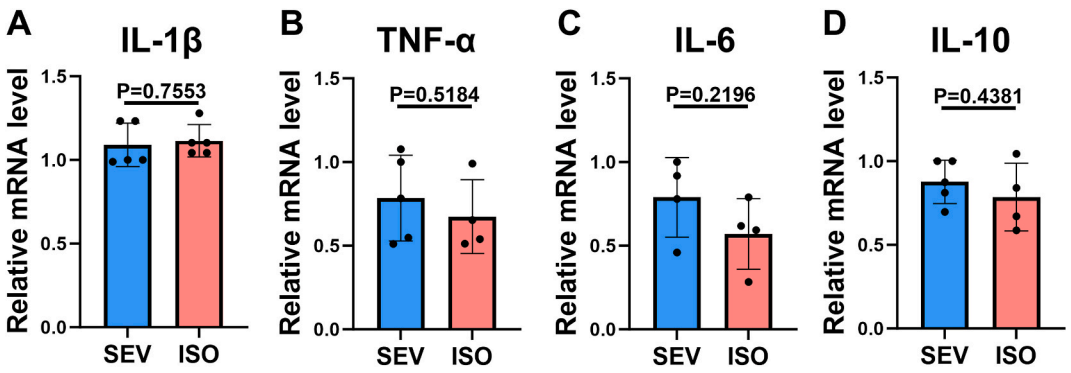

Fig. 6. Comparison of inflammatory levels between the SEV and ISO groups. **A.** The mRNA level of IL-1β **B.** TNF-α, **C.** IL-6, and **D.** IL-10 in infarct areas three days post AMI were comparable between groups. All data were analyzed using an unpaired *t*-test. Sample sizes were n = 4–5 for SEV mice and n = 4–5 for ISO mice. Data are presented as the mean ± SEM.

of frozen hearts, each 1.0 mm thick, were subjected to a 10-min staining process at 37 °C with 2 % TTC. Gross microscopy (Stemi508, Zeiss, Germany) was used to take pictures of each heart slice after it had been fixed for 6 h in 4 % paraformaldehyde. Image-Pro Plus 6.0 was used to measure the infarct area. After TTC staining, the infarcted areas appear pale and viable myocardium stains red. The percentage of the TTC-negative staining area (infarct myocardium) to the total cardiac area was used to compute the infarct size.

2.6. RT-PCR

For RT-PCR testing the infarcted heart tissue was harvested from each mouse. The total RNA of the cardiac tissues was then extracted using the TRIzol reagent. 2 μg RNA was reversely transcribed into cDNA using a Reverted Aid First Strand cDNA Synthesis Kit (Thermo Fisher Scientific, USA), and SYBR Green (BioRad, USA) was used for RT-PCR analysis using the CFX96 Touch™ Deep Well Real-Time PCR Detection System (BioRad Laboratories, Inc., USA). The expression levels of IL-1β, IL-6, IL10, and TNF-α in the infarcted heart were measured. Supplementary Table 3 lists all the primer sequences employed in the experiments.

2.7. Statistical analysis

All measurement data were expressed as mean (SEM), and counting data were expressed as absolute count or percentage prior to analysis. An unpaired two-tailed Student's t-test was applied to compare variables between groups, and two-way ANOVA was used to analyze repeated measurements, followed by the Bonferroni post hoc test. A chi-squared test was used to compare count data, and survival curves of mice were evaluated using the log-rank (Mantel-Cox) test. Statistical values were defined as significant at a P-value <0.05. Finally, all diagrams and results were generated using GraphPad 8 software (GraphPad Software, USA).

3. Results

3.1. Baseline weight of the mice before surgery

Pre-operative weights in the SEV and ISO groups were 20.15 ± 0.28g and 20.27 ± 0.29g, which were not statistically different from each other(see Fig. 1)

3.2. Induction time, recovery time, and occurrence of respiratory depression under the application of sevoflurane and isoflurane

Mice in the SEV group had a significantly shorter induction time compared to those in the ISO group, approximately half as long (66.40 ± 2.90S vs. 125.10 ± 6.30S P < 0.0001)(see Fig. 2A). Likewise, the same trend was observed for recovery time. The SEV mice recovered from anesthesia approximately twice as fast as those in the ISO group (28.00 ± 1.07S vs. 56.88 ± 4.14S, P < 0.0001)(see Fig. 2B). The occurrence of respiratory depression in SEV mice was also lower during the establishment of the AMI model: A total of 5/10 mice developed respiratory depression in the ISO group compared to 0/10 in the SEV group (P = 0.0325)(see Fig. 2C).

3.3. Success rate of AMI modeling and survival rates under sevoflurane and isoflurane

AMI model was successfully established in 14/15 of the mice in the SEV group(see Fig. 3A); 1 mouse died from undetermined causes IN the ISO group. The AMI model was successfully established in 9/15 mice (see Fig. 3A); 5 mice died from respiratory depression, and one mouse died from complications from receiving stitches in the wrong location due to inadvertent movement. Thus, the success rate of the SEV group was substantially higher than that of the ISO group. However, the sample sizes were small, and the difference was not statistically significant (93.33 % vs. 60.00 %, P = 0.0801)(see Fig. 3A). During the 7-day observation period, there were no differences observed between the two groups in terms of survival (P = 0.9080)(see Fig. 3B).

3.4. The cardiac functions of the two groups

Echocardiographic parameters, including LVEF, LVFS, LVIDd, LVIDs, EDV, and ESV, were not different between SEV and ISO groups during the days before surgery or at 1, 3, or 7 after AMI(see Fig. 4). Nevertheless, both groups displayed significantly decreased systolic function after AMI, as indicated by lower FS and EF values (Supplementary Table 1). Furthermore, EDV, ESV, LVIDd, and LVIDs increased after AMI in both groups (SEV and ISO) (Supplementary Table 1).

3.5. The infarct areas of the two groups

There were no differences between the infarct areas in the SEV and ISO groups (49.41 ± 4.18 % vs. 48.66 ± 3.79S P = 0.5266) 3 days post AMI(see Fig. 5).

3.6. The inflammatory levels of the infarct areas in the two groups

There were no significant differences in IL-1β (see Fig. 6A), TNF-α ((see Fig. 6B), IL-6 ((see Fig. 6C), or IL-10 ((see Fig. 6D) in the two groups of mice 3 days post AMI.

4. Discussion

With the advent of next-generation sequencing (NGS) technology, combining multiple databases to analyze the pathological mechanisms and key targets of diseases has become a hotspot for solving clinical problems, and multi-omics has been gradually applied to the study of the molecular mechanisms of AMI [16–18]. Research of pathological mechanisms and therapeutic targets of AMI has mainly relied on animal models due to the limited access to human cardiac tissue. As an experimental basis for AMI in humans, it is very important to develop repeatable procedures for the successful preparation of AMI animal models, and selecting appropriate anesthetic drugs is of great importance in this task. In this study, we compared the application of isoflurane and sevoflurane, and found that sevoflurane was associated with shorter induction and recovery times and fewer adverse reactions, but mortality rate, infarct areas, and inflammatory levels of infarct areas did not differ between the SEV and ISO groups. Therefore, sevoflurane may be a more suitable anesthetic for AMI model establishment in mice compared to isoflurane.

Good controllability of anesthetic depth is critical for the successful establishment of AMI animal models. Due to its low blood gas partition coefficient [19], sevoflurane has the advantages of easier control of anesthetic depth and more rapid recovery from anesthesia than other anesthetic agents [20]. In this study, we found that the induction time of sevoflurane was about half that of isoflurane, and similarly the recovery time was also about half that of the isoflurane group, consistent with previous studies. Adverse reactions to sevoflurane anesthesia were fewer as well, and the isoflurane group was typically too shallow or too deep in terms of anesthesia. For example, a model failure due to inadvertent movement occurred in the isoflurane group but not in the sevoflurane group. Furthermore, in the isoflurane group, respiratory depression, including slower and reduced respiratory rates, caused the death of 4 mice, but similar changes did not occur in the sevoflurane group. This shows that sevoflurane has some advantages in AMI modeling.

Previous studies have shown that prolonged or multiple inhalation anesthetic exposure causes neuronal apoptosis (isoflurane or sevoflurane, 4h, mice [21]), memory and cognitive dysfunction (e. g. sevoflurane 60min–240min, mice; [22]; sevoflurane, once daily for three days, mice [23], genetic damage (Sevoflurane, 120min, rats [24], and intestinal flora imbalance (Sevoflurane, 4h, mice [25]). A Redel et al. [26] found in a 45-min of coronary artery occlusion (CAO) and 180-min perfusion mouse model than when 1.0 MAC isoflurane (ISO), sevoflurane (SEV), or desflurane (DES) were administered 30 min prior to CAO for 15 min, the potency of volatile anesthetics to reduce myocardial infarct size in mice significantly increased, the most from ISO, then less from SEV, and the least from DES. However, when 1.0 MAC ISO, SEV, or DES was administered for 18 min starting 3 min prior to the end of CAO, the potency of these volatile anesthetics to reduce myocardial infarct size in mice was similar. Considerable studies have reported that volatile anesthetics exert a protective effect against myocardial ischemia-reperfusion injury. The mechanism of this protective action has not been fully elucidated, and involves a series of intracellular signal transduction pathways, including the opening of ATP sensitive potassium ion channels, the autokinetic regulation of calcium ions in cells and mitochondria, the synthesis of nitric oxide and the release of reactive oxygen species [27]. More likely, different signal transduction cascades are responsible for preconditioning and postconditioning, and these cascades may be activated differently by different volatile anesthetics. In addition, data from the rabbit model suggest that anesthetic-induced preconditioning is a threshold phenomenon [28]. Above the threshold, infarct size reduction could not be increased neither by prolongation of desflurane administration nor by increase of MAC. Inhalation anesthesia requires a certain dose and time to exert its protective effect. Our study showed that with isoflurane and sevoflurane use during modeling, there were no significant changes in mortality, myocardial infarction area, cardiac function, or cardiac inflammatory factors in either group. This may be because the modeling time was completed within 3–5min, the exposure of the mice to the anesthetic drugs was relatively short and not long enough to produce serious effects. Enomics might identify genetic variants that predispose to higher inflammation or larger infarct size. Transcriptomics could show which genes are upregulated in response to infarction, leading to inflammation. Proteomics would look at the actual proteins involved, like cytokines, and metabolomics might show the metabolic changes due to ischemia. Network analysis in systems biology could map interactions between genes, proteins, and metabolites to identify key nodes that influence both inflammation and tissue repair. In our study, there was no significant difference between pro-inflammatory (IL-1β and TNF-α) and anti-inflammatory factors (IL-6 and IL-10) at the transcriptional level, nor was there a significant difference in tissue necrosis at the cardiac spatial level. With the continuous progress of science and technology, multi-dimensional mechanism mining combining proteomics, transcriptome and non-coding RNA is the most effective and accurate method. In future studies, the combination of proteomics and spatial transcriptomics will better explain the difference in the action of the two drugs. Studies have shown that sevoflurane can play a cardioprotective role by improving oxygen supply and demand balance. Compared with isoflurane, sevoflurane has less effect on respiration [29]. According to our results that SEV group has less respiratory depression, which may improve oxygen supply (and favourable oxygen supply-demand balance) to make mice more likely to survive, this needs to be clarified by further study.

The prediction of a computational model based on finite element method has shown some advantages as lower cost and faster results in evaluating the effect of hip replacement on stability, stress and deformation of total hip prosthesis [30–33], compair to clinical and laboratory study. Similarly, incorporating computational simulation may make the evaluation of the effects of sevoflurane and isoflurane faster, cheapter and easier to understand, this is a potential avenue for future research.

Notable limitations of this study is that it did not include female mice and did not compare other concentrations and anesthesia protocols for either of the two inhaled anesthetics. Only male mice were used in this study to eliminate confounding factors such as reproductive cycles and hormone fluctuations. These shortcomings can be remedied in future work.

5. Conclusion

Sevoflurane anesthesia may be a better choice for AMI modeling in mice, with its shorter induction time, more rapid recovery, and lower risk of adverse reactions compared to isoflurane. Importantly, there was no significant difference in observed cardiac function, inflammatory factors, or myocardial infarction area between sevoflurane and isoflurane.

CRediT authorship contribution statement

Jia-Nan Li: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Liu-Hao-Nan Zeng:** Writing – review & editing, Investigation. **Lu Jin:** Writing – review & editing, Investigation. **Ji-Tong Liu:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bbrep.2025.102000.

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