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ORIGINAL ARTICLE

Impact of Oxidative Stress on Patients Before and After Exposure to Sevoflurane and Desflurane Inhalational Anaesthesia

Kareem Salim Abod,[1] Montadher Ali Mahdi,[2] Abdul Hussien Jaber Luaibi Al-Massoodi,[1] Karrar Jabbar Mansoor,[3] Jaafar Sadiq Jaafar[1]

Abstract

Background/Aim: Oxidative stress, which is characterised by an imbalance between reactive oxygen species (ROS) production and antioxidant defences, is a critical factor influencing surgical outcomes. Inhalational anaesthetics such as sevoflurane and desflurane are widely used, yet their differential effects on oxidative stress remain a subject of investigation. This study aimed to evaluate the impact of these anaesthetic agents on oxidative stress biomarkers in surgical patients.

Methods: The study included a total of 60 patients who underwent laparoscopic cholecystectomy. The patients were categorised into two categories based on the type of anaesthetic they received: sevoflurane (n = 30) and desflurane (n = 30). Blood samples were collected pre- and post-anaesthesia to assess oxidative stress markers, including glutathione (GSH), total antioxidant capacity (TAC), total oxidative status (TOS) and malondialdehyde (MDA). Statistical analyses were performed using an independent t-test, with a significance threshold of $p < 0.05$.

Results: Sevoflurane and desflurane both induced significant oxidative stress, but desflurane exhibited a more pronounced effect. Post-anaesthesia, the desflurane group showed a greater decrease in GSH (ΔGSH: -493.48 ± 153.85 vs -245.77 ± 201.89; $p < 0.0001$) and TAC (ΔTAC: -0.75 ± 0.13 vs -0.35 ± 0.27; $p < 0.0001$), along with a higher increase in MDA (ΔMDA: 2.06 ± 0.74 vs 0.77 ± 0.47; $p < 0.0001$). Although both anaesthetics elevated TOS, the increase was more substantial with desflurane.

Conclusion: Desflurane induced significantly higher oxidative stress compared to sevoflurane, likely due to differences in their metabolic pathways and mitochondrial effects. These findings highlight the need for tailored anaesthetic strategies, particularly for patients with pre-existing oxidative stress conditions. Future research should explore potential mitigation strategies, including antioxidant supplementation and multimodal anaesthesia approaches, to optimise perioperative care.

Key words: Sevoflurane; Desflurane; Antioxidants; Oxidative stress; Anaesthesia, endotracheal.

1. Al-Nukhba University College, Baghdad, Iraq.
2. National Centre for Cancer Research, University of Baghdad, Baghdad, Iraq.
3. Mustansiriyah University, Baghdad, Iraq.

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Corresponding author:
MONTADHER ALI MAHDI
E: montadhermalky@yahoo.com
E: Montadher.a@bccru.uobaghdad.edu.iq

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Introduction

An imbalance between the body's antioxidant defence systems and the generation of reactive oxygen species (ROS) leads to oxidative stress, a crucial biological phenomenon.[1] This imbalance

can accelerate the ageing process and cause cellular and tissue damage, which can contribute to the development of a number of chronic diseases, including diabetes, cardiovascular disorders, neurodegenerative ailments and some types of cancer.[2] Highly reactive chemicals known as free radicals have the ability to oxidase proteins, lipids and nucleic acids, which can impede cellular function and cause organ damage.[3]

In recent years, oxidative stress has gained significant attention in perioperative medicine, particularly concerning the role of inhalational anaesthetics in modulating oxidative balance.[4] Agents such as sevoflurane and desflurane are widely used for inducing and maintaining general anaesthesia.[5] While these anaesthetics are essential for surgical procedures, growing evidence suggests that they may differentially affect oxidative stress levels by altering ROS generation and antioxidant capacity.[6] This is particularly concerning, as oxidative stress during and after surgery can lead to complications such as tissue injury, delayed recovery and increased postoperative morbidity.[7]

The discrepancy in oxidative stress induction between sevoflurane and desflurane is hypothesised to arise from their distinct chemical properties and metabolic pathways, which influence mitochondrial function and the overall redox state within cells.[8] Sevoflurane, known for its relatively lower toxicity, has been reported to increase free radical levels but may also enhance antioxidant defences in certain patient populations.[9] In contrast, desflurane has been associated with a more pronounced increase in oxidative stress, potentially due to its adverse effects on mitochondrial function, leading to elevated ROS production and cellular damage.[10] Mitochondria are central to cellular energy metabolism and their impairment is a key source of ROS generation during anaesthesia. Sevoflurane has been shown to exert a relatively mild inhibitory effect on mitochondrial oxidative phosphorylation, partly preserving ATP production and cellular energy homeostasis. In contrast, desflurane can disrupt electron transport chain activity, leading to increased electron leakage, excessive ROS formation and pronounced mitochondrial dysfunction. These differential effects on mitochondrial dynamics may underlie the distinct oxidative stress profiles observed between the two agents.[11, 12]

The assessment of oxidative stress in clinical research is commonly conducted through the measurement of key biomarkers. Among these, glutathione (GSH), total antioxidant capacity (TAC), total oxidative status (TOS) and malondialdehyde (MDA) are widely used to evaluate the balance between oxidant production and antioxidant defences.[13] Elevated levels of MDA serve as an indicator of lipid peroxidation and cellular membrane damage, while fluctuations in GSH and TAC provide insights into the efficacy of endogenous antioxidant mechanisms.[14] These biomarkers not only facilitate the quantification of oxidative damage but also help delineate the clinical implications of anaesthetic-induced oxidative stress.

Given the potential impact of oxidative stress on surgical outcomes, it is crucial to understand how different inhalational anaesthetics influence oxidative balance. Therefore, the present study was designed with the primary objective of assessing the differential effects of sevoflurane and desflurane on oxidative stress biomarkers, including GSH, TAC, TOS and MDA, in patients undergoing laparoscopic cholecystectomy. A secondary aim was to evaluate pre- and post-anaesthesia changes in these biomarkers within each group, thereby clarifying the relative contribution of each anaesthetic agent to perioperative oxidative stress.

Methods

Study design and participants

This study was conducted at Baghdad Teaching Hospital in Medical City, Iraq and included 60 patients who underwent laparoscopic cholecystectomy. Patients were randomly allocated into two groups according to the anaesthetic agent administered: sevoflurane group (n = 30) and desflurane group (n = 30). Randomisation was performed using a simple random allocation method to ensure unbiased distribution. All enrolled patients were classified as ASA physical status I–II and had no significant comorbidities. For each patient, venous blood samples were collected at two time points (before induction of anaesthesia and after completion of anaesthesia) to assess oxidative stress biomarkers, including GSH, TAC, TOS and MDA.

Inclusion criteria: age: 20 to 50 years; patients undergoing laparoscopic cholecystectomy under inhalational anaesthesia using sevoflurane or desflurane; no history of chronic diseases such as diabetes, hypertension, or autoimmune disorders.

Exclusion criteria: use of antioxidant supplements or specific medications that could interfere with oxidative stress markers.

Sample collection and preparation

Participants' venous blood samples were taken both before and after they were exposed to inhalational anaesthesia. The samples were collected in the period from February to April 2025. To extract the serum, the blood was moved to gel tubes and centrifuged at 1500 g for 10 minutes at room temperature. Before analysis, the serum was kept at -20 °C after being aliquoted into three Eppendorf tubes.

The Ellman method, in which thiol groups react with DTNB to form a yellow-coloured product, was used to measure GSH levels. At 420 nm, absorbance was measured and the extinction coefficient was used to determine the GSH content ($\varepsilon = 13600\ M^{-1}cm^{-1}$).[15]

The Erel technique, which uses antioxidants to prevent O-dianisidine from oxidising, was used to detect TAC. TAC levels were calculated using a standard curve of vitamin C and absorbance was measured at 444 nm.[16]

By oxidising ferrous ions to ferric ions and creating a coloured complex with xylenol orange, the Erel technique was used to determine TOS. TOS levels were computed using a conventional hydrogen peroxide curve and absorbance was measured at 560 nm.[16]

The thiobarbituric acid (TBA) method was used to quantify MDA, a marker of lipid peroxidation. At 535 nm, the absorbance of the MDA-TBA complex was measured and the extinction coefficient was used to determine the MDA concentration ($\varepsilon = 1.56 \times 10^5\ M^{-1}cm^{-1}$).[17]

Statistical analysis

Mean ± standard deviation (SD) was used to illustrate the data. Excel 2010 and SPSS version 20 were used to conduct statistical comparisons. To examine the differences between the pre-exposure and post-exposure groups, the independent t-test was employed. Statistical significance was obtained when the p-value was less than 0.05.

Results

Demographic characteristics of patients (age, body mass index - BMI) and duration of surgery are presented in Table 1.

Table 1: Demographic and clinical characteristics of participants

Variables	Sevoflurane (Mean ± SD)	Desfluran (Mean ± SD)	p-value
Age (years)	40.06 ± 12.43	41.13 ± 12.55	0.742
BMI (kg /m²)	28.52 ± 3.83	27.00 ± 3.09	0.097
Duration of surgery (min)	83.40 ± 18.00	84.00 ± 17.40	0.986

Student t-test; BMI: body mass index;

Oxidative stress parameters

The oxidative stress parameters (GSH, TAC, TOS and MDA) were measured before and after anaesthesia in the sevoflurane group. The results are summarised in Table 2.

Table 2: Oxidative stress parameters in the sevoflurane group

Variables	Before undergoing anaesthesia (Mean ± SD)	After undergoing anaesthesia (Mean ± SD)	p-value
GSH	1306.52 ± 24.26	1066.28 ± 208.93	0.0001
TAC	1.51 ± 0.09	1.17 ± 0.29	0.0001
TOS	45.01 ± 7.04	49.41 ± 8.65	0.0440
MDA	0.75 ± 0.12	1.53 ± 0.44	0.0001

Student t-test; GSH: glutathione; TAC: total antioxidant capacity; TOS: total oxidative status; MDA: malondialdehyde;

The oxidative stress parameters in the desflurane group are presented in Table 3. The results show a more pronounced effect on oxidative stress compared to the sevoflurane group.

Table 3: Oxidative stress parameters in the desflurane group

Variables	Before undergoing anaesthesia (Mean ± SD)	After undergoing anaesthesia (Mean ± SD)	p-value
GSH	1296.93 ± 47.28	803.45 ± 150.47	0.0001
TAC	1.51 ± 0.09	0.76 ± 0.11	0.0001
TOS	48.19 ± 8.05	61.75 ± 7.40	0.0001
MDA	0.77 ± 0.14	2.83 ± 0.77	0.0001

Student t-test; GSH: glutathione; TAC: total antioxidant capacity; TOS: total oxidative status; MDA: malondialdehyde;

Comparison between sevoflurane and desflurane

The changes in oxidative stress parameters (delta values) between the sevoflurane and desflurane groups are presented in Table 4 and illustrated in Figure 1.

Table 4: Oxidative stress parameters in the delta sevoflurane vs desflurane

Variables	Sevoflurane (Mean ± SD)	Desflurane (Mean ± SD)	p-value
Delta-GSH	245.77 ± 201.89	493.48 ± 153.85	0.0001
Delta-TAC	0.35 ± 0.27	0.75 ± 0.13	0.0001
Delta-TOS	9.81 ± 7.12	13.70 ± 10.26	0.0950
Delta-MDA	0.77 ± 0.47	2.06 ± 0.74	0.0001

Student t-test; GSH: glutathione; TAC: total antioxidant capacity; TOS: total oxidative status; MDA: malondialdehyde; Delta: difference before and after undergoing anaesthesia;

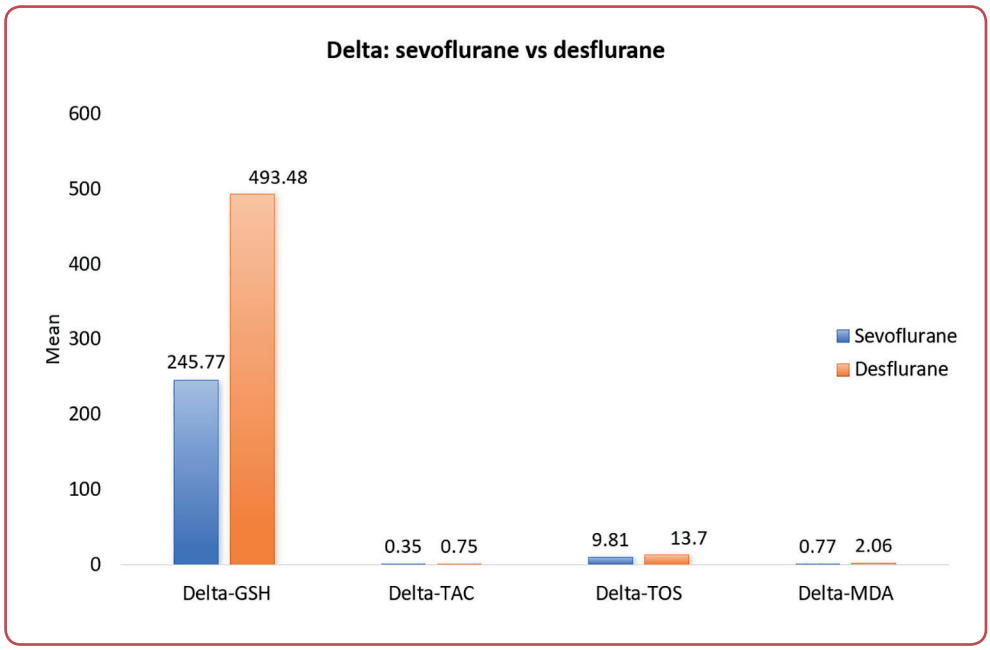

Figure 1: Comparison of delta values (ΔGSH, ΔTAC, ΔTOS, ΔMDA) between sevoflurane and desflurane groups

Delta: difference before and after undergoing anaesthesia; GSH: glutathione; TAC: total antioxidant capacity; TOS: total oxidative status; MDA: malondialdehyde;

Discussion

This study aimed to compare the effects of sevoflurane and desflurane on oxidative stress markers by evaluating reduced GSH, TAC, TOS and MDA levels before and after anaesthesia. The results provide valuable insights into how these anaesthetics influence redox balance through distinct biochemical mechanisms, directly addressing the research objectives. The study confirmed that both anaesthetics induce oxidative stress, albeit to varying degrees, with desflurane exerting a significantly stronger effect.

Initially, the absence of meaningful differences in baseline demographic variables and surgical duration between the study groups supports the validity of attributing the observed alterations

in oxidative stress markers primarily to the anaesthetic agents rather than confounding factors. Additionally, to ensure consistency and eliminate potential confounders, all blood samples were collected from patients undergoing laparoscopic cholecystectomy (gallbladder removal surgery). This standardised approach minimised variability from different surgical procedures, thereby strengthening the reliability of the findings. Sevoflurane exposure was associated with a moderate reduction in antioxidant defences and only a limited rise in oxidative stress indicators, suggesting a comparatively milder redox imbalance. This suggests that sevoflurane, due to its slower metabolic rate, generates lower levels of ROS, leading to a more gradual depletion of antioxidants without causing severe cellular damage.[18] In contrast, desflurane exposure was linked to a pronounced depletion of antioxidant defences and a marked activation of oxidative pathways, consistent with a state of severe oxidative stress. These findings reinforce the growing evidence that desflurane provokes a greater redox imbalance compared to other inhalational agents, underscoring its stronger impact on cellular homeostasis. This imbalance suggests an increase in the formation of ROS, such as superoxide and peroxide, which exceed the body's capacity to neutralise them through its natural antioxidant defences. This accumulation leads to uncontrolled oxidative reactions affecting critical cellular components such as proteins, lipids and DNA, resulting in cellular damage that can directly impair immune function.[19]

At the cellular level, desflurane is associated with disruptions in the electron transport chain within mitochondria, which leads to electron leakage and excessive ROS production. This mitochondrial dysfunction not only affects cellular energy levels but also triggers inflammatory signalling pathways such as NF-κB, leading to increased production of pro-inflammatory cytokines like IL-6, TNF-α and IL-1β. This oxidative-inflammatory environment weakens immune cell function, particularly macrophages and T-cells, thereby diminishing the body's ability to fight infections and impeding proper recovery after surgery.[20] This explanation aligns with the findings of Zivkovic et al, which highlighted that major surgeries lead to elevated oxidative stress and inflammation, negatively impacting recovery quality.[20] Similarly, Pang et al demonstrated that disruptions in the balance between free radicals and antioxidants contribute to neurocognitive and immune complications after surgery.[21] Furthermore, Watt et al showed that laparoscopic surgeries significantly affect gene expression related to oxidative stress and DNA repair, supporting the hypothesis that anaesthesia-induced reductions in antioxidant defences contribute to weakened immune responses and increased postoperative complications.[22] Therefore, presented results suggest that desflurane may increase the risk of postoperative inflammation and complications through a dual mechanism involving oxidative stress and immune suppression. This emphasises the importance of assessing the oxidative state of patients prior to surgery and implementing preventive strategies, such as antioxidant supplementation or modifying anaesthesia protocols, to mitigate these effects.

The differences in oxidative stress responses between sevoflurane and desflurane can be explained by their distinct metabolic and mitochondrial effects. Sevoflurane undergoes hepatic metabolism at a slower rate, producing fewer ROS, which explains the moderate oxidative impact observed.[23] This aligns with previous studies which reported that sevoflurane's lower ROS generation leads to reduced oxidative damage compared to other inhalational anaesthetics.[24, 25] Desflurane, on the other hand, has a much lower solubility in blood and is primarily eliminated through exhalation, which reduces hepatic metabolism but increases mitochondrial oxidative stress. It disrupts the electron transport chain (ETC) in mitochondria, leading to electron leakage and overproduction of superoxide anions, significantly elevating ROS levels.[26] This mechanism has been well-documented in studies, which demonstrated that desflurane is associated with higher mitochondrial dysfunction and oxidative damage.[27, 28] Additionally, Tanaka et al provided evidence that desflurane disrupts mitochondrial respiration more than sevoflurane, explaining its greater oxidative burden.[29]

Beyond mitochondrial dysfunction, recent literature suggests that desflurane triggers pro-inflammatory pathways, exacerbating oxidative stress. Specifically, desflurane has been shown to activate the NF-κB pathway, leading to increased production of pro-inflammatory cytokines such as IL-6, TNF-α and IL-1β, which further amplify ROS generation and cellular damage.[30] Studies demonstrated that desflurane promotes inflammatory signalling more aggressively than sevoflurane, contributing to its higher oxidative impact.[31, 32] Furthermore, oxidative stress and

inflammation are closely linked to postoperative complications, including delayed wound healing, prolonged recovery and even potential neurocognitive impairment due to oxidative stress-induced neuronal damage. Studies suggest that increased MDA and TOS levels after anaesthesia may correlate with transient cognitive dysfunction in older patients, which highlights the need for more targeted perioperative management strategies.[33] The comparative analysis demonstrated that desflurane imposes a greater oxidative burden than sevoflurane, with more pronounced depletion of antioxidants and enhanced lipid peroxidation. Although differences in some parameters did not reach statistical significance, even moderate increases in oxidative stress may hold clinical relevance, particularly in vulnerable patient populations, where subtle shifts can contribute to endothelial dysfunction and systemic inflammation. From a theoretical perspective, these findings reinforce the concept that different anaesthetic agents exert unique oxidative stress profiles based on their pharmacokinetic and metabolic properties. Understanding these distinctions contributes to refining anaesthetic selection, particularly in patients with pre-existing oxidative stress conditions or mitochondrial dysfunction.

Clinically, this study highlights the importance of individualised anaesthetic strategies. Patients with compromised antioxidant defences, such as those with neurodegenerative diseases, diabetes, or cardiovascular disorders, may be more vulnerable to the oxidative effects of desflurane.[34] Implementing protective strategies, such as preoperative antioxidant supplementation (eg vitamin C, N-acetylcysteine), optimising perioperative oxygen delivery and using targeted ventilation techniques, could mitigate oxidative damage. Studies suggest that antioxidant co-administration can attenuate anaesthesia-induced oxidative stress, providing a potential avenue for improving patient outcomes. Additionally, adjusting anaesthetic protocols by reducing desflurane concentration and utilising low-flow anaesthesia techniques may help minimise oxidative stress.[35-37]

Despite its strengths, this study has certain limitations. The sample size, while sufficient to demonstrate significant differences, may limit the generalisability of the findings. Additionally, while key oxidative stress markers were analysed, future research should incorporate additional biomarkers, such as inflammatory cytokines (IL-6, TNF-α) and mitochondrial function indicators, to provide a more comprehensive picture of anaesthetic-induced oxidative stress. Moreover, inter-individual variability in antioxidant defence mechanisms should be explored, as genetic predisposition may influence susceptibility to oxidative damage from inhalational anaesthetics. Future studies could also investigate multimodal anaesthesia approaches that reduce reliance on inhalational anaesthetics, potentially lowering oxidative stress levels.

Moreover, this study did not evaluate postoperative complications such as infections or delirium, nor did it include comparisons with alternative anaesthetic modalities such as total intravenous anaesthesia (TIVA) or target-controlled infusion (TCI). These aspects should be addressed in future research to provide a broader perspective on perioperative oxidative stress. However, the primary objective of the present study was specifically to compare two widely used inhalational anaesthetic agents—sevoflurane and desflurane—that are exclusively administered via the inhalational route and therefore inclusion of TIVA or TCI was beyond the scope of this work.

Age stratification into smaller subgroups was not feasible due to the limited sample size, which would reduce statistical power. Future studies with larger cohorts should address this aspect. In addition, all patients included in this study were ASA I–II without significant comorbidities, which minimised potential confounding; however, this also prevented assessment of possible associations between concomitant diseases and oxidative stress biomarkers and this should be considered in future research.

This study provides clear evidence that sevoflurane and desflurane differentially impact oxidative stress levels, with desflurane inducing significantly greater mitochondrial dysfunction, lipid peroxidation and inflammatory signalling. These findings are consistent with previous literature and enhance our understanding of how inhalational anaesthetics influence redox homeostasis. Given these differences, anaesthetic choice should be carefully considered, particularly in high-risk patients. Future research should focus on optimising perioperative management strategies to minimise oxidative damage, thereby improving patient safety and long-term outcomes in anaesthesia practice.

Conclusion

This study demonstrates that sevoflurane and desflurane exert differential effects on oxidative stress, with desflurane inducing significantly greater redox imbalance, mitochondrial dysfunction and lipid peroxidation. The observed alterations in oxidative stress biomarkers, including GSH, TAC, TOS and MDA, underscore the distinct biochemical impact of these anaesthetics and highlight the importance of careful anaesthetic selection in clinical practice. From a clinical perspective, strategies such as optimising perioperative management and considering adjunctive antioxidant approaches may help mitigate oxidative damage. Future studies should incorporate additional oxidative and inflammatory biomarkers, explore inter-individual variability in antioxidant capacity and evaluate multimodal anaesthesia approaches to minimise oxidative burden. A deeper understanding of these mechanisms will contribute to refining anaesthetic protocols and enhancing patient safety in anaesthesia practice.

Ethics

This study was approved by the Ethics Committee of the College of Sciences, Mustansiriyah University decision No: BCSMU/1224/00029C, dated 1 December 2024. All participants provided informed consent before enrolment.

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Conflicts of interest

The authors declare that there is no conflict of interest.

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Data access

The data that support the findings of this study are available from the corresponding author upon reasonable individual request.

Author ORCID numbers

Kareem Salim Abod (KSA):
0009-0001-2779-9460
Montadher Ali Mahdi (MAM):
0000-0002-6896-9278
Abdul Hussien Jaber Luaibi Al-Massoodi (AHJLA):
0009-0009-7895-2742
Karrar Jabbar Mansoor (KJM):
0009-0007-6207-3423
Jaafar Sadiq Jaafar (JSJ):
0009-0002-9474-7043

Author contributions

Conceptualisation: KSA, MAM, AHJLA, JSJ
Methodology: KSA, MAM, AHJLA, JSJ
Software: KSA, MAM, KJM
Validation: KSA, MAM, AHJLA, JSJ
Formal analysis: KSA, MAM, KJM
Investigation: KSA, MAM, AHJLA, JSJ
Writing - original draft: KSA, MAM, KJM
Writing - review and editing: KSA, MAM, KJM
Visualisation: KSA, MAM, KJM
Project administration: KSA, MAM, AHJLA, JSJ
Funding acquisition: KSA, MAM, AHJLA, JSJ

References

1. Liguori I, Russo G, Curcio F, Bulli G, Aran L, Della-Morte D, et al. Oxidative stress, aging, and diseases. Clin Interv Aging. 2018 Apr 26;13:757-72. doi: 10.2147/CIA.S158513.
2. Maldonado E, Morales-Pison S, Urbina F, Solari A. Aging hallmarks and the role of oxidative stress. Antioxidants (Basel). 2023 Mar 6;12(3):651. doi: 10.3390/antiox12030651.
3. Phaniendra A, Jestadi DB, Periyasamy L. Free radicals: properties, sources, targets, and their implication in various diseases. Indian J Clin Biochem. 2015 Jan;30(1):11-26. doi: 10.1007/s12291-014-0446-0.
4. Tomsič K, Nemec Svete A. A mini-review of the effects of inhalational and intravenous anesthetics on oxidative stress in dogs. Front Vet Sci. 2022 Sep 12;9:987536. doi: 10.3389/fvets.2022.987536.
5. Senoner T, Velik-Salchner C, Luckner G, Tauber H. Anesthesia-induced oxidative stress: are there differences between intravenous and inhaled anesthetics? Oxid Med Cell Longev. 2021 Nov 27;2021:8782387. doi: 10.1155/2021/8782387.
6. Burgos-Santamaría A, Rodríguez-Rodríguez P, Arnalich-Montiel A, Arribas SM, Fernández-Riveira C, Barrio-Pérez IM, et al. OXY-SCORE and volatile anesthetics: a new perspective of oxidative stress in endovascular aneurysm repair-a randomized clinical trial. Int J Mol Sci. 2024 Oct 7;25(19):10770. doi: 10.3390/ijms251910770.
7. Castillo R, Rodrigo R, Perez F, Cereceda M, Asenjo R, Zamorano Jet al. Antioxidant therapy reduces oxidative and inflammatory tissue damage in patients subjected to cardiac surgery with extracorporeal circulation. Basic Clin Pharmacol Toxicol. 2011 Apr;108(4):256-62. doi: 10.1111/j.1742-7843.2010.00651.x.
8. Yalcin S, Aydoğan H, Yuce HH, Kucuk A, Karahan MA, Vural M, Camuzcuoğlu A, Aksoy N. Effects of sevoflurane and desflurane on oxidative stress during general anesthesia for elective cesarean section. Wien Klin Wochenschr. 2013 Aug;125(15-16):467-73. doi: 10.1007/s00508-013-0397-0.
9. Kaşıkara H, Dumanlı Özcan AT, Biçer CK, Şenat A, Yalçın A, et al. The effect of low flow anesthesia with sevoflurane on oxidative status: A prospective, randomized study. Saudi Med J. 2022 Mar;43(3):227-35. doi: 10.15537/smj.2022.43.3.20210876.
10. Zhi-Bin Zhou ZB, Yang XY, Tang Y, Zhou X, Zhou LH, Feng X. Subclinical concentrations of sevoflurane reduce oxidative stress but do not prevent hippocampal apoptosis. Mol Med Reports. 2016;14:721-7. doi: 10.3892/mmr.2016.5336.
11. Zhang DX, Chen YF, Campbell WB, Zou AP, Gross GJ, Li PL. Characteristics and superoxide-induced activation of reconstituted myocardial mitochondrial ATP-sensitive potassium channels. Circ Res. 2001;89:1177–83. doi: 10.1161/hh2401.101752.
12. Sedlic F, Pravdic D, Ljubkovic M, Marinovic J, Stadnicka A, Bosnjak ZJ. Differences in production of reactive oxygen species and mitochondrial uncoupling as events in the preconditioning signaling cascade between desflurane and sevoflurane. Anesth Analg. 2009 Aug;109(2):405-11. doi: 10.1213/ane.0b013e3181a93ad9.
13. Danesh H, Ziamajidi N, Mesbah-Namin SA, Nafisi N, Abbasalipourkabir R. Association between oxidative stress parameters and hematological indices in breast cancer patients. Int J Breast Cancer. 2022 Oct 3;2022:1459410. doi: 10.1155/2022/1459410.
14. Freire Boullosa L, Van Loenhout J, Flieswasser T, De Waele J, Hermans C, Lambrechts H, et al. Auranofin reveals therapeutic anticancer potential by triggering distinct molecular cell death mechanisms and innate immunity in mutant p53 non-small cell lung cancer. Redox Biol. 2021 Jun;42:101949. doi: 10.1016/j.redox.2021.101949.
15. Mahdi MA, Dawood YJ, Sabah RS, Abd Al-Rahman SA. Evaluation of oxidative stress, anti-oxidant, vitamins and co-factor elements in the sera of gastric cancer in Iraqi patients. Asian Pac J Cancer Prev. 2024 Oct 1;25(10):3651-60. doi: 10.31557/APJCP.2024.25.10.3651.
16. Erel O. A novel automated method to measure total antioxidant response against potent free radical reactions. Clin Biochem. 2004 Feb;37(2):112-9. doi: 10.1016/j.clinbiochem.2003.10.014.
17. D'souza D, Subhas BG, Shetty SR, Balan P. Estimation of serum malondialdehyde in potentially malignant disorders and post-antioxidant treated patients: A biochemical study. Contemp Clin Dent. 2012 Oct;3(4):448-51. doi: 10.4103/0976-237X.107438.
18. Ge X, Zuo Y, Xie J, Li X, Li Y, Thirupathi A, et al. A new mechanism of POCD caused by sevoflurane in mice: cognitive impairment induced by cross-dysfunction of iron and glucose metabolism. Aging (Albany NY). 2021 Sep 21;13(18):22375-89. doi: 10.18632/aging.203544.
19. Halliwell B, Gutteridge JMC. Free radicals in biology and medicine (5th ed.). Oxford, UK: Oxford University Press; 2015.
20. Zivkovic A, Jotic A, Dozic I, Randjelovic S, Cirkovic I, Medic B, et al. Role of oxidative stress and inflammation in postoperative complications and quality of life after laryngeal cancer surgery. Cells. 2024 Nov 23;13(23):1951. doi: 10.3390/cells13231951.
21. Pang Y, Li Y, Zhang Y, Wang H, Lang J, Han L, et al. Effects of inflammation and oxidative stress on postoperative delirium in cardiac surgery. Front Cardiovasc Med. 2022 Nov 22;9:1049600. doi: 10.3389/fcvm.2022.1049600.
22. Watt SK, Hasselbalch HC, Skov V, Kjær L, Thomassen M, Kruse TA, et al. Increased oxidative stress with substantial dysregulation of genes related to oxidative stress and DNA repair after laparoscopic colon cancer surgery. Surg Oncol. 2020 Dec;35:71-8. doi: 10.1016/j.suronc.2020.06.009.
23. Qiu L, Li H, Li B, Ek J, Zhang X, Chen Y, et al. Sevoflurane exposure in early life: mitochondrial dysfunction and neurotoxicity in immature rat brains without long-term memory loss. Sci Rep. 2024 Nov 20;14(1):28747. doi: 10.1038/s41598-024-79150-3.
24. Cannesson M, Kain Z. Enhanced recovery after surgery versus perioperative surgical home: is it all in the name? Anesth Analg. 2014 May;118(5):901-2. doi: 10.1213/ANE.0000000000000177.
25. Yuan J, Zhang Y. Sevoflurane reduces inflammatory factor expression, increases viability and inhibits apoptosis of lung cells in acute lung injury by

microRNA-34a-3p upregulation and STAT1 downregulation. Chem Biol Interact. 2020 May 1;322:109027. doi: 10.1016/j.cbi.2020.109027.
26. Jiang Y, Zhu P, Gao Y, Wang A. miR-379-5p inhibits cell proliferation and promotes cell apoptosis in non-small cell lung cancer by targeting β-arrestin-1. Mol Med Rep. 2020 Dec;22(6):4499-508. doi: 10.3892/mmr.2020.11553.
27. Ozcan ATD, Doger C, Ozturk L, Yungul A, Kurtsahin M, Neselioglu S, e al. Comparison of the effects of sevoflurane and desflurane on thiol-disulfide homeostasis in patients undergoing laparoscopic cholecystectomy. Eurasian J Med. 2019 Feb;51(1):70-4. doi: 10.5152/eurasianjmed.2019.18299.
28. Riess ML, Kevin LG, McCormick J, Jiang MT, Rhodes SS, Stowe DF. Anesthetic preconditioning: the role of free radicals in sevoflurane-induced attenuation of mitochondrial electron transport in Guinea pig isolated hearts. Anesth Analg. 2005;100:46–53. doi: 10.1213/01.ANE.0000139346.76784.72.
29. Tanaka K, Weihrauch D, Kehl F, Ludwig LM, LaDisa JF, Jr, et al. Mechanism of preconditioning by isoflurane in rabbits: a direct role for reactive oxygen species. Anesthesiology. 2002;97:1485–90. doi: 10.1097/00000542-200212000-00021.
30. Hanouz JL, Zhu L, Lemoine S, Durand C, Lepage O, Massetti M, et al. Reactive oxygen species mediate sevoflurane- and desflurane-induced preconditioning in isolated human right atria in vitro. Anesth Analg. 2007;105:1534–9. doi: 10.1213/01.ane.0000286170.22307.1a.
31. Lv X, Wang ZM, Huang SD, Song SH, Wu FX, et al. Emulsified isoflurane preconditioning reduces lung injury induced by hepatic ischemia/reperfusion in rats. Int J Med Sci. 2011;8(5):353-61. doi: 10.7150/ijms.8.353.
32. Saraçoğlu KT, Şimşek T, Gürbüz H, Geyik FD, Kale E, Baydili KN, et al. Comparison of the effects of sevoflurane and desflurane on endothelial glycocalyx in patients undergoing laparoscopic hysterectomy: a randomized, double-blind trial. Turk J Anaesthesiol Reanim. 2024 May 3;52(2):76-82. doi: 10.4274/TJAR.2024.231323.
33. Lee YM, Song BC, Yeum KJ. Impact of volatile anesthetics on oxidative stress and inflammation. Biomed Res Int. 2015;2015:242709. doi: 10.1155/2015/242709.
34. van Harten AE, Scheeren TW, Absalom AR. A review of postoperative cognitive dysfunction and neuroinflammation associated with cardiac surgery and anaesthesia. Anaesthesia. 2012 Mar;67(3):280-93. doi: 10.1111/j.1365-2044.2011.07008.x.
35. Pitocco D, Tesauro M, Alessandro R, Ghirlanda G, Cardillo C. Oxidative stress in diabetes: implications for vascular and other complications. Int J Mol Sci. 2013 Oct 30;14(11):21525-50. doi: 10.3390/ijms141121525.
36. Jaruga K, Puścion-Jakubik A, Jakubów P. Regional anesthesia: a narrative review of impact on oxidative stress biomarkers. J Clin Med. 2025 Oct 23;14(21):7503. doi: 10.3390/jcm14217503.
37. Ritiu SA, Rogobete AF, Sandesc D, Bedreag OH, Papurica M, Popovici SE, et al. The impact of general anesthesia on redox stability and epigenetic inflammation pathways: crosstalk on perioperative antioxidant therapy. Cells. 2022 Jun 9;11(12):1880. doi: 10.3390/cells11121880.