·学术前沿：麻醉与肿瘤专题·

主编寄语：现代麻醉学科已突破术中镇痛保命的传统边界，成为调控肿瘤患者围术期安全、重塑机体免疫状态、影响远期肿瘤转归的核心关键学科，是肿瘤综合诊疗体系中不可或缺的重要环节。围术期应激反应、麻醉药物干预、脏器功能管控，均与肿瘤复发转移、并发症发生及预后改善密切相关，是当下肿瘤麻醉领域的前沿研究热点。本专题聚焦麻醉与肿瘤交叉领域的关键科学问题与临床痛点，系列研究兼具基础机制与临床应用价值。既有麻醉管理影响肿瘤转归的体系化探索，也有老年肿瘤患者围术期心脏安全的精细化管控研究，更有右美托咪定靶向通路调控肿瘤治疗效果的机制阐释，贯通基础实验与临床实践。希望本专题成果能为肿瘤围术期精准麻醉、个体化诊疗、预后优化提供新思路与新依据，推动麻醉学科从保障手术安全向赋能肿瘤全程康复进阶，助力肿瘤精准诊疗体系的创新发展。

麻醉药物与围术期管理影响肿瘤转归：从机制到临床的探索

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摘　要：围术期管理，特别是麻醉方案的选择与实施，正日益被视为影响肿瘤患者术后长期转归的潜在可调控因素。本文系统探讨了围术期影响肿瘤转归的关键病理生理机制，包括手术创伤引发的炎症与免疫抑制、生理应激反应以及由此导致的肿瘤微环境改变，为肿瘤细胞逃逸和进展创造了契机。核心聚焦于不同类别麻醉药物（镇静催眠药、吸入麻醉药、局麻药、镇痛药）对肿瘤生物学行为（如增殖、侵袭、转移、免疫应答）的潜在影响及其可能的分子机制。同时，本文综述了当前关于麻醉方式选择及具体麻醉管理策略对肿瘤患者预后影响的临床研究证据。尽管现有临床证据尚不足以确立能明确改善肿瘤预后的标准化围术期麻醉方案，但基础与临床研究均强烈提示，优化围术期管理（涵盖麻醉药物选择、麻醉技术应用及综合支持措施）具有通过多维度调控应激-炎症-免疫轴及肿瘤微环境，进而影响肿瘤转归的潜力。未来研究需致力于深入阐明相关机制，开展高质量的前瞻性临床试验，以推动基于循证医学的、精准化和个体化的肿瘤患者围术期管理策略的构建，最终改善患者长期生存。

关键词：围术期管理；麻醉药物；应激调控；免疫重塑；肿瘤转归；免疫抑制；临床证据；精准医疗
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Anesthetic Agents and Perioperative Management in Tumor Outcomes: from Mechanisms to Clinical Exploration

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Abstract: Perioperative management, especially the selection and implementation of anesthesia protocols, is increasingly regarded as a potentially modifiable factor affecting long-term postoperative outcomes in tumor patients. This paper systematically discusses the key pathophysiological mechanisms in the perioperative period that influence tumor outcomes, including inflammation and immunosuppression caused by surgical trauma, physiological stress responses, and subsequent changes in the tumor microenvironment, which create opportunities for tumor cell escape and progression. It focuses on the potential effects of different types of anesthetics (sedative-hypnotics, inhaled anesthetics, local anesthetics, analgesics) on the biological behaviors of tumors (such as proliferation, invasion, metastasis, and immune response) as well as their possible molecular mechanisms. Meanwhile, this paper reviews current clinical research evidence on the impact of anesthesia modality selection and specific anesthesia management strategies on the prognosis of tumor patients. Although existing clinical evidence is insufficient to establish standardized perioperative anesthesia protocols that can significantly improve tumor prognosis, both basic and clinical studies strongly suggest that optimizing perioperative management (including the selection of anesthetics, the application of anesthesia techniques, and comprehensive supportive measures) has the potential to influence tumor outcomes by multi-dimensionally regulating the stress-inflammation-immune axis and the tumor microenvironment. Future research should focus on further clarifying the relevant mechanisms and conducting high-quality prospective clinical trials, so as to promote the establishment of evidence-based, precise and individualized perioperative management strategies for tumor patients, and ultimately improve long-term patient survival.

Key words: perioperative management; anesthetic drugs; stress modulation; immune remodeling; oncologic outcomes; immunosuppression; clinical evidence; precision medicine

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肿瘤已成为全球范围内对生命的重要威胁,是仅次于心血管疾病的第二大致死病因[1]。尽管近年来对肿瘤的各种治疗方法在研究方面取得了显著进展,但对大多数实体瘤而言,手术切除依然是根治性治疗的首选策略[2]。传统围术期麻醉管理侧重于保障手术安全和缓解术中疼痛,但随着对肿瘤生物学行为理解的不断深入,人们逐渐认识到围术期麻醉管理可能通过多种机制影响肿瘤的转归[3]。本文旨在整合现有机制研究与临床证据,探讨围术期麻醉管理的各要素对肿瘤患者转归的影响,为优化临床实践提供依据。本文为叙述性综述,所引证据主要通过检索PubMed、Web of Science数据库获得,关键词包括“围术期管理”、“麻醉”、“肿瘤”为关键词。在证据选取上,侧重于近五年的文献,并优先纳入随机对照试验、大样本队列研究以及高质量的Meta分析或系统综述,并结合具有重要启发意义的基础机制研究。

1 围术期影响肿瘤患者转归的关键病理生理背景

1.1 手术的治疗作用与创伤引起的相关不良结果

外科手术是实体瘤治疗的基石,其核心目标是完整切除肿瘤原发灶及可能受累的淋巴结,以最大限度降低肿瘤负荷[2]。然而,手术不可避免地造成机体急性创伤,诱发系统性应激反应,短期内显著改变机体内环境,形成一个有利于残留肿瘤细胞存活、增殖和转移的“促癌窗口”[4]。

手术操作不仅可直接破坏肿瘤组织结构,促使肿瘤细胞脱落进入血液循环,形成循环肿瘤细胞,增加远处转移风险[5];同时,创伤激活交感神经系统(sympathetic nervous system, SNS)与下丘脑-垂体-肾上腺轴(hypothalamic-pituitary-adrenal axis, HPA axis),促使儿茶酚胺与糖皮质激素大量释放[6]。此外,手术中释放的损伤相关分子模式(damage-associated molecular pattern, DAMP)如高

迁移率族蛋白B1（high mobility group box 1，HMGB1）、腺苷三磷酸（adenosine triphosphate，ATP）、热休克蛋白（heat shock protein，HSP）等可激活免疫系统[7-8]，引起白细胞介素-6（interleukin-6，IL-6）、肿瘤坏死因子α（tumor necrosis factor alpha，NF-α）、白细胞介素-1β（interleukin-1 beta，IL-1β）等促炎因子升高，进而激活核因子κB（nuclear factor kappa-light-chain-enhancer of activated B cells，NF-κB）、信号转导与转录激活因子3（signal transducer and activator of transcription 3，STAT3）等信号通路[9]，增强肿瘤细胞的增殖、侵袭和免疫逃逸能力，最终加剧术后复发与转移风险[10-11]。

1.2　围术期免疫抑制：肿瘤逃逸的契机

肿瘤免疫微环境（tumor microenvironment，TME）由肿瘤细胞、免疫细胞、间质细胞及细胞外基质共同构成，在肿瘤免疫识别与逃逸中起着关键作用。

围术期多种因素，如手术创伤、麻醉药物、输血、疼痛和营养不良，均可扰乱TME稳态，例如患者氧分压下降、酸碱度异常及代谢产物堆积等，这些都会诱发免疫抑制，为肿瘤逃逸提供条件[12]。

围术发生的TME改变，包括自然杀伤细胞（natural killer cell，NK cell）杀伤能力下降、巨噬细胞向免疫抑制性M2型极化[5, 13]以及调节性T细胞（regulatory T cell，Treg cell）、髓源性抑制细胞（myeloid-derived suppressor cell，MDSC）等免疫抑制性细胞比例升高[14-15]，中性粒细胞胞外陷阱（neutrophil extracellular trap，NET）形成等[16]。这些TME的改变会进一步削弱机体抗肿瘤免疫应答，并显著提高肿瘤细胞逃避宿主免疫监视的风险，为其术后复发与转移创造条件。

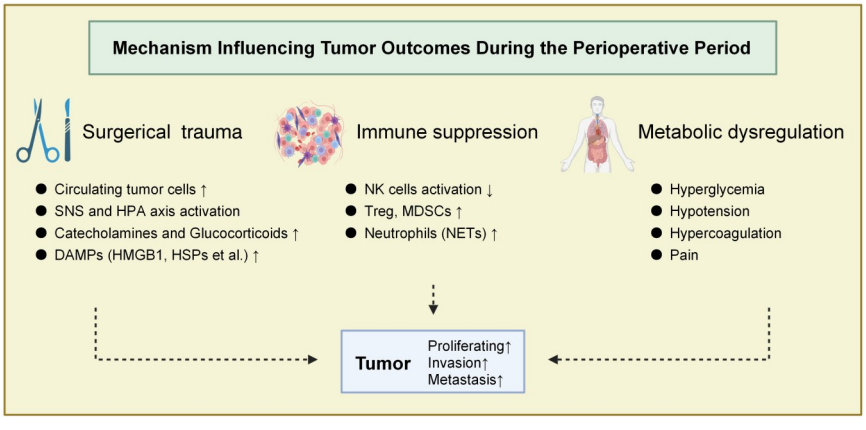
1.3　生理应激与稳态紊乱：肿瘤进展的助推器

围术期生理稳态的失衡通过代谢、内分泌及神经等多条路径影响肿瘤患者预后。其中，高血糖状态不仅直接促进肿瘤细胞的糖酵解代谢[17]，还会抑制免疫细胞功能[18]，形成利于肿瘤逃逸的微环境，从而最终影响肿瘤的预后[19-20]。而麻醉过程中由于应激反应引起的暂时性高血糖及其管理策略，也与患者术后伤口恢复密切相关[21]。

此外，术中低血压导致的组织低灌注与缺氧不仅损害心、脑、肾等重要脏器功能[22]，还可能通过改变肿瘤局部微环境促进癌细胞侵袭和转移[23]。围术期高凝状态既增加血栓事件风险，又可通过血小板聚集、纤维蛋白沉积等方式为肿瘤细胞定植提供支架，加速转移进程[24]。

在上述基础上，术后急性疼痛作为强烈应激源，可进一步放大炎症反应与免疫抑制效应[25-26]，并通过神经内分泌机制干扰肿瘤生物学行为[27]，同时增加心血管事件风险[28]。值得注意的是，虽然阿片类药物可有效镇痛，但其亦可抑制免疫功能，形成“镇痛—免疫”间的矛盾关系[29-30]。

因此，围术期在诸多层面影响肿瘤患者转归（图1）。麻醉管理的核心在于维持生理稳态、调控免疫反应、优化镇痛策略，以减轻肿瘤复发与转移风险。



SNS: sympathetic nervous system; HPA: hypothalamic-pituitary-adrenal axis; DAMP: damage-associated molecular patterns; HMGB1: high mobility group box 1; HSP: heat shock proteins; NK cell: natural killer cell; Treg cell: regulatory T cell; MDSC: myeloid-derived suppressor cell; NET: neutrophil extracellular trap.

图1　围术期影响肿瘤转归的关键机制整合图

Fig. 1　The key mechanisms influencing tumor outcomes during the perioperative period

2 麻醉药物选择对肿瘤转归的影响

2.1 镇静催眠药

丙泊酚是当前研究最广泛的镇静催眠药。研究显示其可抑制缺氧诱导因子-1α(hypoxia-inducible factor 1-alpha, HIF-1α)表达,下调血管内皮生长因子(vascular endothelial growth factor, VEGF)与葡萄糖转运蛋白1(glucose transporter type 1, GLUT1),从而削弱肿瘤血管生成与代谢适应能力,提示其可能具有抗肿瘤潜力[31-33]。但体外实验显示其效应存在争议:部分研究指出丙泊酚可促进肺癌细胞转移[34],亦有研究认为其抑制肝癌细胞生长[35]。该差异可能与肿瘤类型、实验条件等相关,提示其临床应用需谨慎权衡。

依托咪酯为另一种常用镇静催眠药,其可影响皮质醇合成,并具有免疫调节作用进而调控肿瘤转归[36-37]。部分体外研究提示其通过抑制STAT3通路或下调程序性死亡配体1(programmed death-ligand 1, PD-L1)表达,发挥抗肿瘤或增强免疫的作用[38-39],但其免疫抑制作用及对肾上腺皮质的长期影响仍需进一步评估[40]。

2.2 吸入麻醉药

多数基础研究提示吸入麻醉药(如异氟醚、七氟醚)可能具有促肿瘤潜力。例如,异氟醚可通过缺氧诱导因子-1α-β-联蛋白/Notch受体1(hypoxia-inducible factor 1-α-β-catenin / notch1, HIF-1-α-β-catenin/Notch1)信号轴促进膀胱癌上皮间充质化及转移[41],亦可通过雷帕霉素靶蛋白/组蛋白去乙酰化酶6(mechanistic target of rapamycin/histone deacetylase 6, mTOR/HDAC6)通路促进宫颈癌细胞增殖[42]。针对卵巢癌细胞的体外研究进一步发现,吸入麻醉药可显著抑制抑癌基因微RNA-210(microRNA-210, miR-120)和微RNA-138(microRNA-138, miR-138)的表达,从而促进肿瘤细胞的增殖与迁移[43]。此外,异氟醚和七氟醚还被发现可通过抑制淋巴细胞功能相关抗原-1(lymphocyte function-associated antigen 1, LFA-1)的表达,减弱NK细胞的细胞毒性,进而削弱机体的抗肿瘤免疫监视功能[44]。尽管上述研究揭示其潜在促瘤机制,但其临床相关性及普适性尚需深入验证。

2.3 局麻药

利多卡因等局麻药在体外研究中表现出一定抗肿瘤活性,如通过阻断钠通道诱导肿瘤细胞凋亡、减少NETs形成及术后炎症反应[45-47]。临床研究显示,乳腺癌手术中瘤周注射利多卡因可提高患者总生存期(overall survival, OS)和无病生存期(disease-free survival, DFS),并降低复发率[48],提示其潜在应用前景。

2.4 镇痛药

非甾体类抗炎药(non-steroidal anti-inflammatory drugs, NSAIDs)因其抗炎特性被认为具有肿瘤预防潜力,流行病学研究显示长期使用可降低结直肠癌风险[49],且长期服用对于携带体细胞磷脂酰肌醇-3-激酶催化亚基α(phosphoinositide-3-kinase catalytic subunit alpha, PIK3CA)突变的结直肠癌患者能够降低其肿瘤复发[50]。尽管其抗肿瘤机制明确,但临床起效缓慢,故近年来被开发用于联合免疫治疗等新方案中[51-52]。

阿片类药物的使用是围术期镇痛的重要组成部分。阿片类药物通过与肿瘤细胞表面的μ-阿片受体(mu-opioid receptor, MOR)结合,激活磷脂酰肌醇3-激酶/蛋白激酶B(phosphoinositide 3-kinase / protein kinase B, PI3K/Akt)与丝裂原活化蛋白激酶/胞外信号调节激酶(mitogen-activated protein kinase / extracellular signal-regulated kinase, MAPK/ERK)信号通路,进而刺激肿瘤细胞的增殖与存活[53-54]。

此外,MOR信号还可通过多种途径诱导肿瘤血管生成[55]。更重要的是,中枢及外周MOR的激活可抑制HPA轴功能,导致NK细胞毒性下降、T淋巴细胞增殖受限及巨噬细胞吞噬功能受损[56-57]。因此,在围术期镇痛管理中,应权衡阿片类药物的镇痛效益与其对免疫系统的潜在不利影响(表1)。

3 麻醉管理对肿瘤转归的临床证据

3.1 麻醉方式选择的影响

麻醉方式的选择是围术期管理的重要环节,其对肿瘤患者长期预后的潜在影响日益受到关注,但现有临床证据仍存争议,尚未形成统一推荐意见。

目前一些临床研究提示,相较于吸入麻醉,静脉麻醉可能对肿瘤患者更具优势。例如,一项研究指出,接受静脉麻醉的结肠癌患者术后复发率显著低于吸入麻醉组[58]。另有研究进一步证实,无论肿瘤分期如何,静脉麻醉(如丙泊酚)均较地氟烷吸入麻醉在改善结直肠癌的OS和DFS方面更优越[59]。然而,也有部分研究显示,两种麻醉方式在对乳腺癌、肝癌的5年生存率方面差异无统计学意义[59-60]。

表1　围术期常用麻醉药物对肿瘤转归影响的证据总结

Table 1　Summary of evidence on the influence of commonly used perioperative anesthetic drugs on tumor outcomes

Drug type	Representative drugs	Major potential mechanisms	Summary of preclinical evidence	Summary of clinical evidence	Evidence level	References
Sedative-hypnotics	Propofol	Inhibits HIF-1α/VEGF pathway; modulates immune response (controversial)	Most studies demonstrate antitumor effects; a minority suggest promotion of metastasis, which may be tumor-type dependent.	Retrospective studies suggest that, compared with inhalational anesthesia, propofol may be associated with improved survival in certain cancers (e.g., colorectal cancer). However, large RCTs have generally shown no significant difference between the two anesthetic approaches.	Moderate clinical evidence level. Although retrospective data indicate potential benefit for some tumor types, most prospective RCTs have not conclusively demonstrated a clear advantage.	[31-35] [58-60]
	Etomidate	Inhibits STAT3; downregulates PD-L1; affects cortisol synthesis	In vitro studies reveal antitumor potential and possible immunoenhancement.	Limited clinical data; mainly derived from preclinical models.	Low clinical evidence level; a mechanistic rationale exists, but human relevance remains to be established.	[36-40]
Inhalational anesthetics	Isoflurane/Sevoflurane	Activates HIF-1α/β-catenin, mTOR, and related pathways; suppresses NK cell function	Numerous in vitro studies demonstrate enhanced proliferation, migration, invasion, and immunosuppression.	Most retrospective studies have not shown a consistent survival disadvantage; some report outcomes similar to or slightly worse than those with intravenous anesthesia	Preclinical data raise concern, but definitive clinical impact is unclear; the evidence strength is limited.	[41-44] [58-60]
Local anesthetics	Lidocaine	Blocks voltage-gated sodium channels → induces apoptosis; anti-inflammatory; reduces NETs formation	Inhibits the growth and migration of various cancer cell types in vitro and in animal models	Peritumoral infiltration or regional anesthesia (e.g., in breast cancer) has been reported in some studies to improve DFS and OS.	Promising adjunctive strategy; however, the magnitude and dominance of direct antitumor effects in humans remain to be defined.	[45-48]
Analgesics	NSAIDs	Inhibit COX-2/PGE_2 pathway; anti-inflammatory	Demonstrated clear antitumor and antimetastatic effects in preclinical models.	Epidemiological and adjuvant therapy studies confirm reduced risk of specific cancers and lower recurrence rates.	Well-established anticancer and metastasis-suppressing properties; perioperative short-term use and long-term outcome data are limited.	[49-52]

续表

Drug type	Representative drugs	Major potential mechanisms	Summary of preclinical evidence	Summary of clinical evidence	Evidence level	References
	Opioid agonists	Activate MOR/PI3K/Akt pathway; suppress cellular immunity; promote angiogenesis	Promote cancer cell proliferation and migration; inhibit NK and T-cell function in experimental settings.	Complex "analgesia - immunity" trade-off; short-term perioperative use has unclear impact on long-term oncologic outcomes.	Mechanistic actions are multifaceted; clinical significance requires further investigation.	[53-57]

HIF-1α: hypoxia-inducible factor 1 alpha; VEGFR: vascular endothelial growth factor receptor; RCT: randomized controlled trial; STAT3: signal transducer and activator of transcription 3; PD-L1: programmed death-ligand 1; mTOR: mammalian target of rapamycin; NK: natural killer; NETs: neutrophil extracellular traps; DFS: disease-free survival; OS: overall survival; NSAIDs: non-steroidal anti-inflammatory drugs; COX 2: cyclooxygenase-2; PGE2: prostaglandin E2; MOR: mu-opioid; PI3K: phosphoinositide 3-kinase; Akt: protein kinase B (Akt).

区域麻醉技术在围术期中亦展现出潜在价值。以利多卡因为代表的局麻药广泛用于神经阻滞与切口浸润镇痛,其通过减少全身阿片类药物用量、缓解手术应激与炎症反应,可能间接改善肿瘤微环境,从而使肿瘤转归朝着好的方向发展[61]。回顾性研究提示,硬膜外麻醉或外周神经阻滞与骨肉瘤等实体瘤患者的良好长期生存结局相关[62-63]。然而,目前有关区域麻醉药物直接抗肿瘤效应的机制研究仍较为有限,其积极作用可能更多源于围术期整体管理的协同效应[64]。

需要指出的是,现有临床证据在麻醉方式对肿瘤患者预后影响的研究中普遍存在异质性,导致结论间存在一定不一致性。造成该现象的因素较为复杂,主要可归纳为以下几个方面:① 肿瘤类型与分期的差异性。由于临床病例具有高度个体化特征,而肿瘤本身在组织学类型、分子特征及病程分期等方面表现出显著异质性,这些差异可能直接影响患者对不同麻醉策略的生物学反应及术后转归。因此,不同研究之间因入组肿瘤类型或分期的差异,所获结局指标存在偏差,是导致大型临床研究结论不尽一致的重要原因之一;② 麻醉方案的多维复杂性。麻醉管理本质上为多要素协同作用的综合过程,除基础镇静与镇痛目标外,还涉及辅助用药选择、液体管理、体温维护等多方面内容,难以孤立评价某一麻醉方式的独立效应。例如,在静脉麻醉组的研究中,常联合应用区域神经阻滞以实现更佳镇痛,进而可能掩盖单纯静脉麻醉本身的效应;此外,阿片类药物因其强效镇痛特性被广泛应用,但不同研究中对阿片类药物的种类选择、给药剂量与患者耐受程度各异,进一步加大了组间比较的不确定性;③ 围术期其他关键变量的干扰。诸如围术期输血、术后并发症发生及其干预措施、辅助性放化疗方案等均可能对患者免疫状态及肿瘤复发风险产生影响。然而,在真实世界研究中,上述因素通常难以完全剥离或实现标准化控制,研究者往往需在综合考量的基础上进行评估,从而限制了因果推断的准确性。

综上所述,现有的证据初步表明,麻醉策略的选择可能影响肿瘤患者的术后转归。倾向性建议包括:优先选用丙泊酚等免疫抑制较轻的静脉麻醉药、减少吸入麻醉药及阿片类药物的使用,并积极探索区域麻醉技术的应用。然而,鉴于现有证据受多重混杂因素影响,对于某些看似明确的结论,如"静脉麻醉优于吸入麻醉"或"吸入麻醉促进肿瘤进展"等论断,目前尚缺乏充分的高质量证据支持,临床解读与推广仍需保持审慎态度。未来研究应通过严格设计的前瞻性随机对照试验,最大限度控制混杂偏倚,以厘清麻醉干预的真实效应。

3.2 麻醉管理的探索

除麻醉药物选择外,一系列围术期辅助管理策略亦被证实可能对肿瘤患者预后产生积极影响[65-66],特别是对于合并多种基础疾病的肿瘤患者。

近年来,麻醉深度的实时监测逐渐纳入常规管

理范畴。过深的麻醉状态[如脑电双频指数(bispectral index，BIS)< 40]可能加剧免疫抑制、激活HPA轴并促进炎症因子释放；而适当深度的麻醉(BIS：50~60)有助于保留皮质反馈环路，从而更好地调控应激与免疫稳态[67]。因此，实施精准麻醉深度管理对肿瘤患者的转归有着重要影响。此外，合适的麻醉深度也能够减少肿瘤病人术后的认知功能障碍、术后炎症因子释放[68-69]。

在呼吸管理方面，肺保护性通气策略(小潮气量联合适度呼气末正压通气)可有效减少肺泡炎症及全身氧化应激水平[70]，进而减少肿瘤病人术后的肺部并发症[71]。此外，术中低体温可能抑制免疫功能并促进凝血异常，而适当的体温维护则有助于改善术后免疫状态、促进康复并降低感染风险[72]。而对于肿瘤病人，麻醉期间维持适当的体温可以减少其深静脉血栓发生率，并可能减少肿瘤复发[73-74]。

目标导向液体管理策略能够减少组织水肿与炎性渗出，优化微循环与氧供，减少术后血乳酸浓度，可能间接降低肿瘤病人术后并发症风险，并促进肠道功能恢复，保持免疫功能[75-77]。

上述麻醉期间各项管理策略不仅对肿瘤患者有益，亦有助于减少围术期心脑血管事件及肺部并发症，对合并基础疾病的老年患者尤为重要。

此外，肿瘤患者常合并多种基础疾病，而特别是食管、胰腺、肺、肝和结直肠癌的手术在应激及麻醉药物引起的血流动力学波动作用下，更易诱发围术期心血管不良事件，如心肌梗死、缺血性卒中等[78]。另一些研究也提示，肿瘤病人经历的化疗及激素类新辅助治疗也会增加围术期心血管不良事件、血栓栓塞事件的发生[79-80]。因此，针对该类高风险人群，实施有效的围术期心血管监测策略，如术前及术后心肌肌钙蛋白的动态检测，有助于早期识别心肌损伤并及时干预，从而改善患者预后[81]。

4　从证据到实践：潜在临床转化方向

基于现有麻醉药物作用机制以及相关麻醉管理策略的临床证据，如何将其有效转化为可改善肿瘤患者术后结局的临床实践，已成为当前研究的重点与难点。本文围绕具有明确转化潜力的研究方向展开探讨，旨在为后续前瞻性研究的方案设计提供参考。

方向一：聚焦特定肿瘤类型的麻醉策略优化及其随机对照研究设计。已有大型观察性研究初步提示，不同麻醉策略的联合应用可能对特定肿瘤患者的预后产生积极影响。例如，回顾性研究结果显示，在以丙泊酚为基础的全凭静脉麻醉应用于结直肠癌及乳腺癌手术患者时，可能具有一定的肿瘤学获益趋势；同时，采用区域神经阻滞替代高剂量阿片类药物的镇痛方案亦显示出改善肿瘤相关预后的潜力。据此，建议在前瞻性随机对照试验中，纳入可手术的Ⅱ~Ⅲ期结直肠癌或乳腺癌患者，系统比较“丙泊酚联合椎旁神经阻滞/腹横筋膜平面阻滞”与“七氟醚吸入麻醉联合传统阿片类镇痛”两种麻醉模式对患者DFS及OS的影响。研究设计应严格控制手术方式、围术期护理路径及辅助治疗等混杂因素，以确保麻醉干预效应的独立性与科学性。

方向二：探索针对高风险肿瘤人群的多维度围术期综合麻醉管理策略。老年患者及接受新辅助放化疗的肿瘤患者常伴随免疫功能下降、合并症复杂及术后并发症风险升高等特点，属围术期管理的重点与难点人群。现有研究表明，合理调控麻醉深度、实施肺保护性通气策略、维持术中正常体温及推行目标导向液体治疗等措施，有望改善该类高危群体的围术期预后。因此，可构建一套“整合BIS监测联合血流动力学参数指导的精准麻醉”、“肺保护性通气策略”、“全程严格体温管理”以及“多模式镇痛(侧重限制阿片类药物使用)”为一体的“强化围术期麻醉管理方案”，并通过随机对照研究评估其在降低术后并发症发生率、提升短期生活质量及改善长期肿瘤学结局方面的临床效益。

上述研究方向兼顾基础机制向临床实践的转化潜力，强调围术期麻醉干预对肿瘤患者远期预后的可能影响，有望为构建以麻醉为核心的肿瘤多学科协作管理模式提供理论依据与实践路径。

5　总结与展望

实验室的基础研究为理解围术期干预如何影响肿瘤转归提供了一定的理论依据。但麻醉方法的选择和麻醉管理，在临床研究结果与实验室研究之间仍存在不一致之处，反映出患者围术期各种高度复杂的效应及真实世界多因素交互作用的干扰。

未来的个体化麻醉不应仅停留于理念层面，而应立足于生物标志物检测与数据驱动策略的系统融合。首先，应深入探索肿瘤分子分型与麻醉药物

敏感性及围术期管理策略反应性之间的内在联系，为构建“生物标志物导向的精准麻醉”模式奠定理论基础。例如，不同信号通路的活化状态可能决定特定肿瘤类型对麻醉干预的反应差异。基础研究已表明，微卫星高度不稳定的结直肠癌对免疫治疗表现出较高敏感性，提示其肿瘤免疫微环境具有较强的可调控性。在此背景下，或可推测部分具有免疫调节潜能的麻醉药物(如丙泊酚、右美托咪定)或特定麻醉策略(如区域阻滞联合低阿片方案)在该类患者中发挥更显著的肿瘤学保护作用。此类假设有待通过基础实验与临床队列研究加以验证，推动麻醉干预从经验性向机制导向转型。另一方面，随着人工智能技术的发展，借助其强大的数据处理与模式识别能力，整合肿瘤基因组信息、患者术前合并症特征及术中多模态生理监测数据，构建用于预测术后肿瘤复发风险或围术期免疫功能动态变化的预测模型，正成为大数据时代下麻醉学研究的新兴方向。进一步开展前瞻性临床试验，评估该类模型在术前麻醉方案个体化制定与术中实时决策支持中的可行性与临床应用价值，将有助于推动精准麻醉由理论构想迈向临床实践，真正实现以患者为中心的个体化围术期管理目标。

展望未来，很有必要推动麻醉学、肿瘤学、免疫学与生物信息学的深度交叉融合，设计并实施更加严谨的大样本、多中心、前瞻性随机对照临床试验，并结合生物标志物进行人群分层分析；同时，借助单细胞测序等新兴技术手段，有望进一步阐明麻醉干预对肿瘤干细胞、代谢重编程及免疫记忆的影响。

在临床实践中，应在保障手术安全与重要脏器功能稳态的前提下，优先考虑采用免疫抑制效应轻微、应激调控良好及多模式镇痛为核心的麻醉策略。此类策略包括但不限于：优先选用丙泊酚实施全静脉麻醉、合理联合区域阻滞技术、综合考虑阿片类药物的使用与疼痛二者之间对肿瘤的影响。至关重要的是，需依据患者的肿瘤生物学特征、临床分期及整体健康状态，构建个体化的麻醉方案，以期多维度改善患者近远期预后。

综上所述，围术期管理可通过多维度调控应激反应、免疫稳态及肿瘤微环境，进而影响肿瘤患者转归。尽管现有证据尚不足以确立可改变肿瘤预后的常规麻醉方案，但对相关机制的深入探索，无疑将为构建精准化、个体化的围术期管理体系奠定坚实的理论基础。围术期的管理，包括麻醉方案的选择和麻醉管理、手术前后的各种干预措施在肿瘤综合管理中的潜在价值，仍需要漫长和深入的探索。

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