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右美托咪定：临床新效应

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摘　要

右美托咪定(Dexmedetomidine)是一种强效且高选择性的α2-肾上腺素能受体激动剂，具有解交感神经、镇静、遗忘和阿片保留等特性。它在众多临床应用中被广泛认可为一种安全且有效的辅助药物，能够提供独特的“有意识的镇静”状态——容易从睡眠到清醒过渡。目前，右美托咪定的临床应用范围广泛，包括重症监护病房(ICU)的镇静(成人和儿童)、急诊科、区域和全身麻醉、神经外科、儿童手术中经鼻腔或直肠给药的镇静、新型光纤插管、心脏手术等。不同的给药途径，如静脉给药，鼻内给药，口服给药，神经阻滞等，其使用剂量及不良反应等可能也存在差异，对于术后恢复的影响也不尽相同。低血压、高血压和心动过缓是最常见的不良反应，但通常可通过适当的临床管理得到控制。总之，右美托咪定凭借其独特的药理特性，已成为临床镇静治疗中的重要选择之一。

关键词

右美托咪定，临床新应用，给药新途径

Dexmedetomidine: New Clinical Effects

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Abstract

Dexmedetomidine is a potent and highly selective α2-adrenergic receptor agonist with sympathetic, sedative, amnesia, and opioid retention properties. It is widely recognized as a safe and effective adjunct in numerous clinical applications, capable of providing a unique state of “conscious sedation”—

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an easy transition from sleep to wakefulness. Currently, dexmedetomidine has a wide range of clinical applications, including sedation (adults and children) in intensive care units (ICUs), emergency departments, regional and general anesthesia, neurosurgery, sedation administered through the nose or rectum in pediatric surgery, novel fiberoptic cannulation, cardiac surgery, etc. Different routes of administration, such as intravenous administration, intranasal administration, oral administration, nerve block, etc., may also have different dosages and adverse reactions, and their effects on postoperative recovery are also different. Hypotension, hypertension, and bradycardia are the most common adverse effects, but they are usually managed with appropriate clinical management. In conclusion, dexmedetomidine, with its unique pharmacological properties, has become one of the important choices in clinical sedation treatment.

Keywords

Dexmedetomidine, New Clinical Applications, Novel Administration Routes

1. 右美托咪定的作用

1) 灌注和呼吸安全性：右美托咪定的呼吸抑制是最小的，即使大剂量使用[1] [2]，不同于其他镇静剂和麻醉剂；与灌注阿片类药物，苯二氮卓类药物或异丙酚相反，右美托咪定可通过气管拔管或更远的地方安全地灌注[3]。这一有利特性可以在清醒时开颅和清醒时插管等特定情况下，对不良事件提供很大的保护。他的交感作用可在围手术期提供稳定的血流动力学，联合镇静和最小的呼吸抑制，在一些手术中为清醒患者和清醒纤维插管提供了安全和可接受的条件。它已被成功地用于帮助那些以前因过度激动而拔管失败的病人拔管[4] [5]。对于需要无创通气的激动患者也有类似的效果[6]。右美托咪定在纤维插管或其他困难的气道操作过程中可有效实现良好的镇静而不会抑制呼吸[7]-[9]。虽然考虑心动过缓和低血压的风险，但阿托品和血管活性药物可能很容易控制这些事件，口干也是其副作用之一[10]，这种抗唾液刺激有助于形成干燥的皮肤，营造了一个相对干燥的插管环境，插管条件进一步加强；同时减少了插管的次数，给病人减少刺激和伤害，提高了安全可行性[11]。但在临床标准化灌注给药方案层面，目前国内外临床实践与相关指南仍存在明显分歧，尚未形成统一、公认的围术期标准化输注剂量和负荷量的管理共识。且目前越来越多的麻醉中心已趋向无负荷量单纯维持灌注模式。其次对于不同年龄段，不同手术类型的维持灌注速率缺乏精准量化分层标准。期待未来右美托咪定临床灌注应用朝着标准化、个体化、精准化、智能化四个核心发展。

2) 心血管效应：全麻手术期间，在保证重要器官的氧供条件下，采用降压药物和技术，使得血压水平趋于我们的目标血压。而右美托咪定对血压(BP)有双相作用，右美托咪定的负荷剂量导致短暂性的血压升高和反射心率下降。右美托咪定辅助全麻可维持血流动力学的稳定性。右美托咪定在外周组织中可激动 α2A 受体，刺激血管收缩，在中枢神经系统中可激动突触前 α2B 和 α2C 受体，对去甲肾上腺素分泌予以抑制，进而弱化机体反应，在上述双重作用下，该药物可对机体血流动力学予以调整，维持其稳定性[12]。相反，低血压是常见的副作用，发生时血管舒张作用的中央 α-2a 受体占主导。剂量依赖性心动过缓主要是由交感神经张力的降低，压力感受器反射和迷走神经活动增强介导的[10] [13]。合理使用右美托咪定进行控制性降压，可以减少视野出血，为手术提供清晰的手术视野，同时还可以减少术中的输血

情况。使用右美托咪定降压还有明显的优势，可稳定血流动力学，防止脑血管扩张，减少脑水肿情况的发生[14]。

3) 镇静镇痛：右美托咪定镇静作用呈剂量依赖性，如果给药剂量足够大，右美托咪定会产生深度镇静甚至全身麻醉，这表明右美托咪定可以成为全静脉麻醉的一部分[15]。右美托咪定的镇痛特性是由多种机制介导的，包括脊髓，脊髓上和周围活动[16]。右美托咪定的阿片保留效果已经通过一些临床试验得到了很好的证明[17]，右美托咪定即使作为唯一的止痛剂，也能有效缓解腹腔镜下输卵管结扎术后疼痛，尽管在恢复期可能不适宜伴有嗜睡和心动过缓[18]。但也有研究的 Meta 分析表明，全麻手术中，右美托咪定优于瑞芬太尼，在术后 24 小时内疼痛评分低，低血压，颤抖和术后恶心呕吐更少。它通过神经轴向路径对躯体和内脏疼痛具有抗伤害性感受作用[19]。最近一项随机对照试验的荟萃分析显示，神经轴向右美托咪定明显降低术后疼痛强度和延长了镇痛时间[20]，但存在一定几率增加了心动过缓的风险。

2. 右美托咪定的临床应用

1) 麻醉佐剂：右美托咪定可显著降低吸入麻醉药和静脉麻醉药的麻醉需求。还可以降低各种外科手术患者围手术期和术后阿片类药物的用量。右美托咪定的阿片类保留作用减少了阿片类药物的使用，从而降低了阿片类药物引起的呼吸抑制的风险，如肥胖患者和呼吸系统严重的患者。此外，右美托咪定与其他麻醉镇静镇痛药物的协同配比争议突出。普遍临床上与丙泊酚、瑞芬太尼符合应用，但复合用药时相互作用叠加效应缺乏量化评估，不同配比下循环抑制、苏醒时间、术后谵妄发生率对比研究结论不一致，导致作为麻醉佐剂缺乏统一安全阈值标准。

2) 预防谵妄。镇静药物如苯二氮卓或丙泊酚可能增加 ICU 患者谵妄的风险[21]，几项随机对照试验表明，右美托咪定治疗的患者在 ICU 中的谵妄症状明显低于涝西泮[22] [23]，咪达唑仑或丙泊酚。然而在这些研究中，右美托咪定与众所周知的增加谵妄的 GABA 受体调节剂进行了比较[24]，因此右美托咪定是否不像其他镇静剂那样产生谵妄甚至预防谵妄，目前尚不清楚。在《柳叶刀》杂志社的一项研究表明，预防性低剂量右美托咪定有效预防了谵妄的发生[25]。非心脏手术后在 ICU 的头 7 天谵妄(右美托咪定组 9%，安慰剂组 23%；优势比 0.35，95% CI 0.22~0.54；$p < 0.0001$)在这项随机，双盲，安慰剂对照试验中，低剂量右美托咪定的使用时间小于 24 小时(手术当日入 ICU 至术后第 1 天上午 8 时)，心动过缓、低血压发生率无明显升高，但高血压、心动过速、低氧血症发生率明显降低。这些有希望的结果提高了右美托咪定可作为预防谵妄的可能性。但现有循证医学证据整体仍存在设计异质性大、结局定义不统一、高危人群分层不足、机制研究浅表等关键缺陷，药物究竟通过何种中枢通路、何种细胞靶点发挥抗谵妄作用至今尚不明确，右美托咪定降低谵妄发生是药物直接中枢保护效应还是改善睡眠、减轻应激、稳定血流动力学的间接综合效应？

3) 癌痛应用：癌症疼痛治疗面临着疼痛管理无效、大量阿片类药物使用和镇痛不足等挑战。右美托咪定是一种新型的 α-2 受体激动剂，具有稳定性好，半衰期相对较短等优点[26]。可作为一种潜在的辅助镇痛药物，广泛应用于参与围手术期疼痛和癌性疼痛治疗的多模式镇痛方案中，可扩大阿片类药物的使用，减少恶性副作用和耐药的发生[27]。其镇痛机制涉及中枢蓝斑细胞超级化，激活外周、脊髓和脊髓 α-2 受体，调节细胞信号通路和炎症因子。DEX 减少有害神经递质产生和疼痛信号传递，增强阿片类镇痛，同时减少阿片类药物的使用和耐受性[28]。然而由于强大的镇静作用，DEX 常产生低血压，心动过速等不良反应[29]，因此仅限于麻醉医师学会 ASA 一至二级及以下患者使用监测和管理，极大地限制了它的发展前景。

4) 多学科应用：1) 心血管外科：右美托咪定的交感活性可降低心肌耗氧量，围手术期通过降低代谢和预防心动过速，这些良好的效果可以减少术后心脏并发症的发生包括心脏缺血。2) 神经外科：右美托

咪定添加或不添加瑞芬太尼已成为在清醒患者的神经外科手术中提供安全和可接受条件的最有用的药物。特别是在需要复杂的神经学评估的清醒开颅手术中，许多研究表明右美托咪定有很多的优势[30]-[32]。协同镇静使得避免心动过速和高血压，此外，右美托咪定还具有潜在的神经保护作用，包括降低颅内压和剂量依赖性地降低脑血流量和脑代谢率[33]-[35]。一项随机对照试验表明，在清醒开颅时，右美托咪定的术中脑成像质量和镇静效果与丙泊酚瑞芬太尼相似，并且右美托咪定组的呼吸不良事件较少[36]。3) 儿科：在儿科 ICU 机械通气患者中，右美托咪定的镇静作用优于咪达唑仑，且血压无明显差异[37]。右美托咪定已被研究用于婴儿和儿童的放射诊断程序，与咪达唑仑或异丙酚相比，在 1~7 岁接受核磁共振的患者中，右美托咪定更有可能实现良好的镇静制止躁动，以及更快地苏醒和恢复[38]。据报道，右美托咪定成功用于侵入性手术，如中心静脉导管置入和支气管镜检查[39]。4) 胸外科：全麻辅以右美托咪定可提高呼吸顺应性，该药物可减少手术所致的肺组织炎症反应，改善机体氧化应激状态和氧合功能，进而对肺组织予以保护；也可减轻肺叶切除后肺损伤，维持肺容量和肺顺应性[40]-[42]。

3. 右美托咪定的给药新途径

1) 静脉给药：右美托咪定目前仅被批准用于成人静脉给药，也是其最传统，最广泛的应用途径。右美托咪定的消除半衰期是 2 小时左右，可乐定是 8 小时，右美托咪定的分布半衰期为 5~6 分钟[43]，血浆结合率高达 94%，起效非常迅速，并且可以长时间的维持较为稳定的血药浓度，是静脉滴注的理想药物。其根据药理动力学推荐最大输液速度为 1.4 μg/kg/h。通常在手术中快速诱导前后，先给予一个负荷剂量 0.5~1.0 μg/kg，20 分钟左右后，再给予静脉泵注 0.2~0.7 μg/kg/h 维持。有研究表明在腹腔镜手术中譬如妇产科或胆囊手术，麻醉快速诱导前或诱导后或手术结束前 40 分钟，给予负荷剂量 0.5 μg/kg，可以减少术后躁动[44] [45]。术中持续泵注 0.5 μg/kg/h (术毕前 1 小时停止)以及术后夜间小剂量 0.1~0.2 μg/kg/h 持续泵注，可有效减少术后谵妄[46]；若术中持续 0.5 μg/kg/h 泵注，个体化根据体重以及监测指标加用负荷剂量，可以大大减少术后恶心呕吐；或小剂量 0.1~0.2 μg/kg/h 则不良反应率更低，镇痛泵中加入 0.02 μg/kg/h 还可以改善术后睡眠质量[46] [47]。当然，在老年患者群体中，肝功能较差，BMI 较大，还合并基础疾病，则右美托咪定的清除速率可有不同程度的延长[48]，因此对于不同的手术群体应予以个体化的给药方案。

2) 口腔或鼻内给药：右美托咪定是一种无色，无味，无刺激性的液体，而在鼻腔内或口腔内给药途径，是一种无创、便捷的。在健康志愿者中，颊部右美托咪定(即药物溶液在口腔中滞留 5 分钟)具有 82% 的高全身生物利用度[43]。鼻内右美托咪定可用滴剂或喷雾器给药至鼻腔粘膜。盐酸右美托咪定单次给药的生物利用度为 41%~65%，起效快(30~45 分钟) [49] [50]，达到了临床有效的血药浓度。鼻内给药的优势在于可适用在无法静脉给药的儿科患者中[51]；另外一方面可以避免静脉给药后的高血药浓度水平，减少因血药浓度升高导致的呼吸、心血管等方面的不良反应[48]。另外，鼻黏膜和口腔黏膜非常薄且面积巨大，药物极易通过黏膜被吸收作用于机体，有研究表明可能绕过血脑屏障的途径，如嗅神经、三叉神经直接产生效应。并且无论滴定还是雾化，药代动力学没有显著差异，可以安全地应用于患者，可以避免肝脏的首过消除作用，有效提高右美托咪定的生物利用度[48]。目前使用的鼻内右美托咪定制剂(100 μg/ml)明显超过了鼻内给药时推荐的最大剂量(0.1~0.2 ml)，且作用时间相对较短(3~4 小时) [50] [51]。右美托咪定相对迅速地吸收并引起显著的镇静作用，展现出无可替代的临床应用，尤其是对于无法配合的儿童患者。与鼻内给药相比，口服右美托咪定起效较慢(50~60 分钟)作用时间较长(10 小时) [52] [53]，因为存在较强的首过效应，但口服是最简单，最便捷的方式；即使是儿童也具有较高的依从性。因此口服的右美托咪定能否达到最低起效的血药浓度，仍在研究当中，口服右美托咪定具有一定的应用前景，但剂量、制剂类型、药物效应、不良反应、影响因素、特殊人群的应用规范仍需进一步探索。开发作用时间更长、浓度更高的右美托咪定制剂是有必要的。其他潜在但很少研究的给药途径包括皮下、肌内、直肠、经皮、鞘

内和硬膜外给药途径。

3) 神经阻滞：麻醉医生们一直在寻求延长单次注射周围神经阻滞的好处的策略，超过常用的局部麻醉(LA)的持续时间[54]；神经周围阻滞辅助治疗就是一种技术上简单的方法，可以用于这一目的[55]。右美托咪定可以延长局部阻滞的持续时间[56]，而且在神经阻滞给药时的全身镇静作用尚不清楚[57]。有研究[57]表明，右美托咪定在腋窝神经阻滞术(ABPB)术后的镇静作用，研究人员发现除了延长感觉和运动阻滞持续时间[58] [59]，加快感觉和运动阻滞的发生。使用右美托咪定的镇痛效果还包括延长镇痛时间，减少术后 24 小时的累积镇痛量，改善疼痛控制，提高疼痛缓解满意度，右美托咪定的神经阻滞给药可能提供了相当安全舒适的镇静，对血流动力学或呼吸稳定性没有显著影响，并产生高水平的患者满意度。但在下肢阻滞中使用右美托咪定可能会延迟门诊手术后的恢复，并增加跌倒的风险[60]。此外心动过缓和低血压很容易通过监测手段被发现，但这些副作用可能会妨碍高危患者与心率和血压变化固有的相关手术，如坐位手术[61]。但这仍是一个很有前途的新研究领域，尽管血流动力学副作用的潜在机制和相对风险尚未被探索。在围术期神经阻滞管理中，右美托咪定既可通过静脉持续输注发挥全身性镇静与抗交感效应，亦可作为佐剂鞘内或神经周围局部注射以延长阻滞时效、强化术后镇痛效果。两种给药途径在临床获益、作用机制及安全谱上存在显著差异，迄今尚未形成统一的规范化选择共识，是当前区域麻醉领域的核心争议议题。从镇痛和阻滞质量来看，鞘内与神经周围局部给药在多项临床研究中展现出明确的增效优势。然而静脉给药仍存在不可替代的价值。其效应的稳定性，同质化程度高，不受穿刺操作和药物扩散差异的影响，较为可控但镇痛增益有限。在其静脉给药的核心风险是全身性循环抑制，而在鞘内/神经周围给药则存在神经局部毒性和远期安全性不明的风险。

4. 总结

右美托咪定凭借良好的镇静镇痛效果及轻微呼吸抑制优势，已在围术期麻醉与神经阻滞辅助镇痛中得到广泛应用，短期临床获益明确。但目前临床仍以静脉短期给药为主，缺乏长效口服制剂，限制了其在慢性癌痛长期镇痛管理中的推广应用。未来应重点研发长效口服缓释剂型，优化药代动力学特征，拓展其在慢性癌痛非阿片类镇痛治疗中的应用价值。此外，右美托咪定神经周围及鞘内给药虽镇痛增效显著，但神经毒性安全性仍存在争议，现有临床证据质量不足。后续亟需开展大规模多中心长期随访 RCT，明确局部给药安全剂量范围与远期神经损伤风险，明确其临床安全应用边界。通过剂型改良与高质量循证验证，可进一步推动右美托咪定临床应用向长效化、精准化与安全化方向发展。

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