

胸外科电视辅助胸腔镜手术的术后镇痛进展

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摘 要

开胸手术和电视辅助胸腔镜手术(video-assisted thoracic surgery, VATS)均会产生中度至重度以上的术后疼痛, 影响患者康复, 降低患者的医疗满意度。自1910年诞生以来, VATS以微创的方式逐步应用于肺、纵隔和胸膜等疾病。VATS虽然属于微创手术, 疼痛程度明显小于开胸手术, 但是术后疼痛程度依旧很高, 多数患者难以忍受。镇痛不足可影响患者术后功能恢复和气道分泌物排出, 从而导致患者发生局部肺不张、肺炎和肺栓塞等一系列并发症, 甚至死亡率增加。因此, VATS的良好术后镇痛至关重要。本文对当前VATS常用术后镇痛的研究进展进行文献总结。

关键词

电视辅助胸腔镜手术, 术后镇痛, 胸部

Advances in Postoperative Analgesia for Video-Assisted Thoracic Surgery in Thoracic Surgery

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Abstract

Both open thoracic surgery and video-assisted thoracic surgery (video-assisted thoracic surgery, VATS) produce moderate to more than severe postoperative pain, which affects patient recovery

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and reduces patient medical satisfaction. Since its inception in 1910, VATS has been progressively applied in a minimally invasive manner to diseases of the lung, mediastinum and pleura. Although VATS is a minimally invasive procedure with a pain level significantly lower than that of open-heart surgery, the level of postoperative pain is still high and intolerable for most patients. Inadequate analgesia may affect the postoperative functional recovery and airway secretion discharge, which may lead to a series of complications such as localized pulmonary atelectasis, pneumonia and pulmonary embolism, and even increased mortality. Therefore, good postoperative analgesia for VATS is crucial. In this paper, we summarize the literature on the current research progress of postoperative analgesia commonly used in VATS.

Keywords

Video-Assisted Thoracic Surgery, Postoperative Analgesia, Chest

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1. 胸腔镜术后疼痛原因、机制及危害

1.1. 胸腔镜术后疼痛原因与机制

胸外科患者术后疼痛严重且情况复杂, 包含了炎症性疼痛、躯体痛、内脏痛、神经病理性疼痛等急性和慢性疼痛。躯体痛是患者疼痛的主要来源, 由胸壁和胸膜损伤刺激肋间神经引起。皮肤切开、胸腔镜置入、肌肉离断、肋骨牵拉或引流管的放置都会引起疼痛。与开胸手术相比, VATS 由于恢复时间更快和术后疼痛减少而变得越来越受欢迎[1], 但是围手术期仍会出现明显疼痛, 常见原因包括: 手术创伤、留置引流管、肋间神经损伤或压迫、胸膜受损、焦虑或紧张等情绪以及术后体位不适等[2]。VATS 作为微创手术, 主操作口长度一般小于 4 cm, 其余孔径通常为 1~2 cm, 切口小且不用撑开肋骨或做过多的肌肉分离, 肋间切口疼痛和胸膜间引流管摩擦是造成本术后急性疼痛的主要原因[3]。这种疼痛信号从肋间神经传递到同侧脊髓背角, 通过对侧前外侧索传入边缘系统和大脑皮层而产生痛觉。前列腺素、缓激肽和组胺等炎症因子释放, 激活了伤害感受器并通过脊髓背角神经元的兴奋导致中枢敏化[4]。胸腔内各部位脏器和胸膜的伤害性刺激分别传入迷走神经和膈神经, 从而产生内脏痛。神经性疼痛可以由肋间神经的直接损伤引起, 并且可能导致超敏反应和神经痛, 包括感觉过敏、超敏和痛觉过敏[5]。

1.2. 胸腔镜术后疼痛产生的危害

咳嗽、咳痰有助于肺复张, 促进胸腔积液排出, 是胸腔镜手术患者术后肺功能恢复的关键因素。术后急性疼痛控制不良通常会导致患者对咳嗽咳痰相关疼痛产生恐惧, 从而加重术后肺部并发症[6]。术后疼痛剧烈的患者还会出现呼吸幅度减小和功能残气量下降[7], 进而导致患者发生气道闭合、肺不张、肺内分流和组织低氧血症[8]。免疫功能、肌肉功能、凝血功能和伤口愈合受到影响[9]。患者情绪和心理方面也会发生变化, 例如睡眠障碍、情绪低落、焦虑和抑郁等[10]。

急性疼痛控制不及时, 可引起外周伤害性感受器激活、中枢敏化和神经调节功能下降, 约 25% 的患者会最终演变为慢性疼痛[11], 急性疼痛越严重的患者发生慢性疼痛的概率越大, 且时间也越长[12]。VATS 具有类似开胸手术急、慢性疼痛的发生情况, Baymen EO 等人[13]的研究结果显示, VATS 慢性术后疼痛(chronic post-surgical pain, CPSP)的发生率和疼痛的严重程度与开胸手术并无差异。因此, 提供及

时有效的疼痛管理,显得尤为重要。

2. 目前国内外对于 VATS 术后主要镇痛方式

2.1. 全身镇痛

全身镇痛因其技术简单、有效、安全,临床实践中广泛应用于术后镇痛,常见的方式有:口服镇痛药、静脉注射镇痛、肌肉注射镇痛等。VATS 术后镇痛最常用的方法是患者静脉自控镇痛(patient controlled intravenous analgesia, PCIA) [14]。PCIA 让患者根据对自身疼痛的感知程度与需求在一定范围内调节镇痛药剂量[15],可提供 48~72 h 的镇痛效能。目前胸外科手术常用的全身镇痛药有阿片类药物、非甾体类抗炎药(NSAIDs)和其他辅助用药等。

2.1.1. 阿片类药物

阿片类药物最常通过静脉、口服、椎管内或贴皮途径给药。本类药物可与外周神经及脊髓背角神经元上的阿片受体结合,阻止疼痛信号向中枢传导;同时也作用于大脑和脑干的疼痛中枢,降低人体对疼痛的主观感受。阿片类药物静脉自控镇痛被广泛应用于胸痛的治疗[5],但由于其成瘾性以及不良副作用,如嗜睡、呼吸抑制、恶心、呕吐、皮肤瘙痒、尿潴留、便秘以及胆绞痛[16]等,使用阿片类药物时尤其应注意用量。Andrade 等人[17]关于胸外科手术无阿片类镇痛的研究发现无阿片类药物术后镇痛可能更有效,且术后恶心呕吐的发生率低。对于无阿片类镇痛是否能获益仍需进一步探讨。

2.1.2. 非甾体类抗炎药(Non-Steroidal Anti-Inflammatory Drugs, NSAIDs)

NSAIDs 主要是通过抑制环加氧酶(COX),阻断前列腺素(PG)和血栓素 A_2 (TXA₂)的产生从而发挥抗炎、镇痛、退热及抗血小板聚集等作用。NSAIDs 可以通过口服、肌注、静脉注射或直肠途径给药。常用的药物有塞来昔布、帕瑞昔布、氟比洛芬酯等。一般认为 NSAIDs 通过抑制 COX-2 发挥抗炎镇痛作用,而对 COX-1 的抑制导致包括胃肠道溃疡、急性肾功能衰竭、血小板功能异常等在内的副作用产生。NSAIDs 还被指出与心血管风险发生有关,心血管疾病高危患者需谨慎使用[18]。NSAIDs 的剂量具有封顶效应[19],临床使用应注意剂量。

2.1.3. 其他辅助用药

其他辅助用药包括:氯胺酮、曲马多、右美托咪定和 5-羟色胺-3 (5-HT₃)受体拮抗剂等。这些药物在临床上作为其他镇痛技术或药物的辅助用药,以增强镇痛效果,减少副作用的发生。氯胺酮是 N-甲基-D-天冬氨酸(N-methyl-D-aspartic acid, NMDA)受体的非竞争性拮抗药,其作用广泛,但镇痛机制尚不清楚[20]。近年来,氯胺酮因其抗抑郁作用发挥了很大的临床价值,亦作为阿片类药物的替代用药/辅助用药在急性疼痛管理方面广受欢迎[21][22]。曲马多是一种非典型阿片类药物,目前以联合用药为主,主要用于中到重度的各种急性疼痛及手术后疼痛的治疗,慢性癌性疼痛、神经病理性疼痛均有疗效。右美托咪定是 α_2 肾上腺素受体激动剂的代表,具有镇痛、抗焦虑、抗交感、稳定血流动力学和减少阿片类药物剂量的作用,患者在镇静状态下可唤醒且无呼吸抑制[23],其最常见的副作用为心率减慢和血流动力学不稳。

2.2. 区域阻滞

2.2.1. 胸段硬膜外镇痛(Thoracic Epidural Analgesia, TEA)

胸段硬膜外镇痛(thoracic epidural analgesia, TEA)过去一直是开胸手术镇痛的“金标准”[5],广泛用于胸腔镜手术后镇痛。药物通过硬膜外导管作用于脊髓、脊髓丘脑束的背侧和腹侧支、脊神经根和交感神经产生镇痛作用[5],可减轻躯体痛和内脏痛。临床研究表明 TEA 可有效缓解躯体痛和运动痛,并且

还能改善肺功能、减少阿片类药物的应用和缩短患者卧床时间[24]。

也有观点认为[9],硬膜外镇痛不是“金标准”,胸腔镜 PROSPECT 指南[1]不再推荐使用 TEA。原因在于,TEA 有许多不容忽视的不良反应,如低血压、皮肤瘙痒、尿潴留、硬膜外血肿、呼吸抑制等,这些并发症可能影响患者康复。一项回顾性研究[25]发现 VATS 肺叶切除术患者的住院时间延长可能与术后给予 TEA 有关,因此微创手术需要安全性更高的术后镇痛方式[26]。一项 Meta 分析[27]认为 VATS 术后镇痛更推荐采用单侧区域镇痛。其中,单次筋膜平面阻滞(如竖脊肌平面阻滞、前锯肌平面阻滞等)的不良反应发生率更小,且对于麻醉医生操作技术要求相对较低,在微创胸外科越来越受欢迎[28]。

2.2.2. 胸椎旁神经阻滞(Thoracic Paravertebral Block, TPVB)

在 1905 年,Hugo 施行了第一例椎旁神经阻滞[29]。胸椎旁神经阻滞(thoracic paravertebral block, TPVB)通过在相应的椎旁间隙给予局麻药,对交感神经和感觉神经产生阻滞效能,从而产生对同侧交感神经和躯体神经的阻滞,为胸科提供良好的镇痛。经典的 PVB 技术利用体表标志定位完成,其失败率为 10% [30],近年来各种可视化技术使 PVB 成功率大幅提高。

因为椎旁间隙贴近胸膜,肋间血管神经走行其内,故 TPVB 仍存在一定的并发症和不良反应。虽然椎旁穿刺引起血肿的机会较小[31],但是仍不建议对抗凝的患者实施 TPVB [32]。尽管 Meta 分析[33]表明,TEA 和 TPVB 仍是 VATS 的最佳区域镇痛干预措施,但是综合各种区域镇痛的不良反应发生率、操作时间和对麻醉医生的技术要求等因素来说,单次筋膜平面阻滞还有很大的发展前景和应用价值。

2.2.3. 肋间神经阻滞(Intercostal Nerve Block, ICNB)

肋间神经阻滞(intercostal nerve block, ICNB)是将局部麻醉药注入肋间神经沟以阻滞肋间神经的方法,潜在的肋间间隙使局部麻醉药可渗透至邻近的肋间神经,从而达到同时阻滞多条肋间神经的效果[34]。与 TEA 和 TPVB 相比,ICNB 可以在胸腔镜直视下操作,操作简便、安全,不需要特殊的设备和技术,不明显延长手术和麻醉时间。Li Shuo 等人[35]研究表明,ICNB 组在术后 48 小时内的 VAS 评分比静脉自控镇痛组低。

单根肋间神经阻滞范围小,而多孔胸腔镜手术切口距离较远,往往需要阻滞多根肋间神经才能达到满意的镇痛效果。多项研究表明[36][37],ICNB 的镇痛效果不如 TEA 和 TPVB 镇痛效果完善,但其并发症少,联合 PCIA 可提供有效的胸科术后镇痛,也可作为其他镇痛措施失败的补救措施。

2.2.4. 前锯肌平面阻滞(Serratus Anterior Plane Block, SAPB)

2013 年,Blanco 等[38]改良了 PecI 和 PecII 路径,首次提出前锯肌平面阻滞(serratus anterior plane block, SAPB)。超声引导下 SAPB 安全易实行且镇痛效果完善,通过阻断肋间神经外侧皮支、胸长神经和胸背神经提供有效的镇痛。SAPB 单点局麻药注射阻滞范围可达 T2~T9 [38],而大多数 VATS 切口都在 T4~T8 之间。前锯肌平面为筋膜层,其内无大血管走行,解剖位置表浅,较少出现穿刺部位血肿和神经损伤[39],对交感神经系统无阻滞作用,不影响血流动力学。与 TPVB 和 TEA 相比,超声引导下 SAPB 操作简便,具有广阔的临床应用前景。

张隆盛[40]等的研究认为使用 0.4%罗哌卡因的 SAPB 比 TPVB 持续的时间更长,操作时间更短、并发症少、安全性高。Zengin M 等人的研究对比了竖脊肌平面阻滞与浅层前锯肌平面阻滞对于 VATS 术后急性疼痛的影响,患者在术后 24 小时内的疼痛评分、阿片类药物消耗和副作用相似[41]。另有研究[42]认为浅层比深层 SAPB 镇痛效果更佳。Fajardo 等建议在前锯肌深层进行注射,因浅层平面注射易影响胸长神经,导致胸长神经的暂时性麻痹,而胸长神经受损可导致翼状肩胛骨综合征。当前对 SAPB 的研究仍不够透彻,在作用机制、药物代谢、临床应用等方面仍需进一步探讨。

2.2.5. 竖脊肌平面阻滞(Erector Spinalis Plane Block, ESPB)

2016 年, Forero 等人首次描述了竖脊肌平面阻滞(erector spinalis plane block, ESPB)用于治疗慢性胸神经性疼痛和胸外科手术后疼痛, 该研究发现在 T5 横突水平竖脊肌深层注射局麻药, 可阻断 T3~T9 脊神经支配区域[44]。竖脊肌处于脊柱两侧深面, 由最长肌、棘肌和髂肋肌构成。解剖学研究表明[44], ESPB 可能通过不同机制的组合起作用, 例如, 局部麻醉药可以扩散到胸椎旁间隙、硬膜外间隙和脊神经背侧支从而产生镇痛效果。ESP 阻滞的预期目标是实现局麻药扩散至胸椎旁间隙[44], 产生抑制内脏疼痛的效果。VATS 疼痛管理指南建议[1], 将 TPVB 或 ESPB 作为 VATS 术后首选区域镇痛。

ESPB 因注药点要高于椎旁阻滞的注药点, 穿透胸膜的风险低; 同时 ESPB 穿刺点远离脊髓, 脊髓损伤的风险小; 对循环和呼吸功能影响也小。虽然 ESPB 镇痛效果显著, 但对麻醉医生有一定的技术要求, 如果局麻药液硬膜外扩散还会发生低血压和单侧交感神经阻滞[45], 从而引起 Horner 综合症表现。

2.2.6. 肋间神经冷冻

直视下肋间神经冷冻预防手术后切口疼痛首次报道于 1974 年[46], 方法为: 把肋间神经直视下游离后, 于-90℃~-60℃时冷冻 30~45 s, 使神经髓鞘受损, 阻碍其疼痛信号的传导。由于轴突未遭到破坏, 其信息传导功能可在 1~3 个月内再生恢复[7]。该镇痛方式仍存在争议, 会使神经性疼痛、感觉迟钝和肋间肌麻痹的发生率增高[47] [48]。多位学者认为[49] [50], 肋间神经冷冻不仅不能有效降低胸外科术后疼痛的发生率, 还会增加慢性疼痛综合征的发生率。该技术还需要昂贵的冷冻设备, 因此推广应用方面也受到了限制。

2.2.7. 切口局部麻醉药浸润

由外科直视下进行切口局部麻醉药浸润, 其操作简便、安全、廉价、不良反应少、患者依从性高, 可反复多次使用。也可于切口皮下、筋膜下或肌层置入导管, 术后经导管持续泵入局部麻醉药, 延长镇痛时间。Paladni G [51]等人认为连续伤口浸润可以使疼痛强度和阿片类药物消耗普遍降低, 可以包含在术后疼痛的多模式治疗中, 或者在其他选择有禁忌症时作为有效的替代方案。

2.3. 多模式镇痛

国内多位专家[52]认为, 胸科手术属于重度疼痛手术, 建议术后使用多模式镇痛。胸外科多模式阿片类镇痛的基础是区域阻滞麻醉联合全身非阿片类镇痛, 重要原则是 NSAIDs 为术后镇痛基础用药, 以减少阿片类药物引起的不良反应如肠麻痹等[14]。

近年来已有多项研究[53] [54]采用联合区域阻滞镇痛的模式, 与单一镇痛模式相比为患者提供了更为全面的术后镇痛。在越来越多镇痛方式被发掘的今天, 胸外科术后最佳镇痛方式仍需大量临床研究。

3. 小结

在当今微创胸外科手术日渐发展的时代, 联合应用区域镇痛能提供更完善的术后镇痛效果, 进一步减少阿片类药物的使用、加速患者恢复、减少住院时间。ESPB、SAPB 等联合阻滞具有镇痛效果显著、操作安全、循环稳定的优势, 期待在临床上的发展。综上所述, 针对 VATS 镇痛的多项技术各有利弊, 例如镇痛效果最好的硬膜外镇痛因其高风险而渐渐退出历史舞台, 而目前微创区域镇痛因操作简便和低风险而越来越受欢迎。麻醉医师需要对比不同阻滞技术的效果和利弊, 针对具体的外科手术制定更合适的疼痛治疗方案。

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