· 综述 ·

高频振荡通气的机制及临床应用

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【关键词】 高频振荡通气； 呼吸机相关性肺损伤； 机械通气

自 1972 年 Lunkenheimer 等发现高频振荡通气(HFOV)可以保证气体有效交换以来，此后针对其机制、通气策略、设备改良、临床应用等方面的研究层出不穷。近 20 年，HFOV 在新生儿科的广泛应用[1]，大大增加了医师对 HFOV 临床应用的信心，使得越来越多成人呼吸衰竭(呼衰)患者在常频机械通气(CV)失败以后采用 HFOV。目前 HFOV 已成为治疗严重肺部病变如急性呼吸窘迫综合征(ARDS)的重要补救方法。

1 HFOV 的气体交换原理和呼吸机相关性肺损伤(VILI)

ARDS 患者在 CV 时主要面临的并发症是 VILI。在机械通气时，肺的各个区域通气不均匀可能引起 VILI，其主要机制有 3 个方面：①在顺应性较好的区域，吸气末容积(EILVs)过高可能导致肺损伤；②气道在吸气时骤然开放和呼气时突然陷闭可能损伤终末细支气管；③在肺塌陷区和充气区的边缘交界处，组织的联接点在呼吸时承受过高的相互作用力，可能导致严重肺实质损伤，其中后两条机制被命名为“剪切力”。剪切力是指当力的作用方向平行于物体表面，使得两个物体在相互作用力的作用下趋近或分开，对抗物体的滑动趋势。尤其在呼气末容积(EELVs)过低时，剪切力更是肺损伤的重要机制[2]。在正常肺，跨肺压是使肺泡均一扩张的力；而在损伤肺，肺泡局部的跨肺压不仅是对抗肺泡的不均一充气，更是倾向于使肺泡过度扩张。正如 Mead 等[3]很多年前提出的那样：当一个扩张的肺泡和一个塌陷的肺泡相邻时，两肺泡间的跨肺压仅 30 cm H_2O (1 cm H_2O = 0.098 kPa)；而当几个充分扩张的肺泡包绕一个塌陷肺泡时，局部所产生的跨肺压可能高达 140 cm H_2O，潜在的剪切力的损伤可想而知。

在肺损伤动物模型中可以看到过高 EILVs 的危害，过大的潮气量(V_T)主要分布在未受损肺区域中，其表现为炎症反应加重，肺血管通透性增加，肺泡内出血等[4-5]。这就是“容积伤”，即吸气时肺容积过高所引起的肺牵张损害[6-7]，小 V_T 通气策略的成功更加证实了该机制。过低 EELVs 造成的剪切力的损伤在很多动物实验中也得到了证实[8]。而高呼气末正压(PEEP)对于过低的 EELVs 具有肺保护作用，在 CV 模式下已经得以证实[9-12]。但是在 CV 模式下应用高 PEEP 的同时，也有可能增加 EILVs，进一步加重了肺损伤，除非 V_T 进一步减小。所以说，最佳 PEEP 在肺保护和肺损伤之间难以平衡，难以滴定，使得 CV 在治疗难治性呼衰时陷入窘境。故此 HFOV 较 CV 更有优势：①HFOV 的 V_T 极小，可以允许高 EELVs 去获得更佳的肺复张，而避免过高的 EILVs 损害；②HFOV 比 CV 的呼吸频率高，这就允许更小的 V_T 达到接近正常的 CO_2 清除率[2]。

HFOV 的实现是在呼吸回路中连接一个往复运动的活塞，活塞的往复运动将气体压入患者气道，也将患者气道内的气体吸入活塞内，这是一种吸气和呼气均为主动的工作方式[13]，由此在呼吸回路里产生了具有一定幅度和频率的振动压，且这个可调节幅度和频率的振动压可以叠加在人为可调的平均气道压(mPaw)上。人为可调的 mPaw 可以使肺泡充盈并支撑肺泡避免由于 ARDS 病理改变引起的肺泡塌陷[14]，并且由此增加了胸腔内气体与肺泡的接触面积，进而减少了剪切力引起的肺损伤；而通过极高的振动频率和调整振动压的幅度可以产生极低的肺泡通气量，远远低于生理死腔。尽管 HFOV 近端管路内压力极高，甚至吸气相远大于 100 cm H_2O，但气流在呼吸管路中经过气管插管后压力已衰减 80%～90%，仅有 10%～20% 振动压进入气道，再经过各级支气管的逐级衰减，进入肺泡的压力极低[15-16]，产生真正的极低肺泡 V_T，并且在肺泡内吸气相和呼气相压力变化极小，以达到减少肺损伤的目的。应用 HFOV 时每次振荡产生的 V_T 远远小于患者的解剖死腔，其通气方式和常频呼吸机有显著区别，具体机制尚未完全阐明，目前已知至少有对流、摆动式反复充气、不对称的流速剖面、Taylor 传播、心源性震动、分子弥散 6 种机制参与气体交换[17]。另外，值得一提的是，HFOV 振荡频率为 3～15 Hz(180～900 次/min)，而该频率正是人体肺脏共振频率，使得气体交换不只在肺泡进行，在各级肺段呼吸单元内都可进行，加速了肺内气体的混合；同时在共振情况下小气道阻力也最小，使得肺内气体分布更趋于均一性。

很多研究者推测，HFOV 的机械通气机制可以进一步降低 VILI 的发生率。一些小动物的实验研究也支持 HFOV 可以减少肺组织损伤和炎症反应[18]。在盐水灌洗兔急性肺损伤(ALI)的模型中，与 CV 相比，应用 HFOV 时肺透明膜形成、肺泡白细胞浸润、气道上皮损伤等显微镜下形态学表象有所改善；另外，一些炎症因子如肿瘤坏死因子(TNF)、白细胞介素(ILs)、转化生长因子、黏附分子等表达也有所减少[18-20]。早期动物实验比较 HFOV 和控制机械通气(CMV)时，CMV 参数设置多为大 V_T 和高吸气压，而现在的研究多数是应用肺保护性通气策略与 HFOV 进行比较[19,21-23]。Imai 等[19]对 ARDS 兔研究发现，HFOV 组中性粒细胞浸润、TNF 表达更低，肺泡间隙的病变更少。而另一项研究略有不同，在鼠 ARDS 模型中，HFOV 与压力控制通气(PCV)模式下应用高 PEEP 相比较，肺泡液的蛋白浓度无差异[21]。但上述小动物模型与成人的 HFOV 设置差别较大，也许大动物实验研究更接近成人，因为成人应用更大

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型号的气管插管，人工气道直径越大产生的振幅压力(ΔP)和 V_T 就越大。但目前比较 HFOV 和 CV 联合应用肺保护通气策略的大动物实验研究是不多的[24-26]。有研究对羊 ALI 模型进行了 PCV(V_T 8 ml/kg)和 HFOV 的比较，发现 PCV 组肺组织学损伤更重[27]。

2 HFOV 突破 ARDS 机械通气的困境

2.1 顽固性低氧

2.1.1 比 CV 能更好地改善低氧血症：一个前瞻性、多中心研究显示[28]：将 150 例 ARDS 患者随机分为 HFOV 组和 CV 组，HFOV 组起始频率 5 Hz，mPaw 较 CV 组高 5 cm H_2O；CV 组应用 PCV 模式，V_T 6～10 ml/kg，尽管 HFOV 组与 CV 组 30 d 病死率无明显差异(37%比 52%，$P=0.102$)，但 HFOV 组氧合指数(PaO_2/FiO_2)在最初 24 h 内较 CV 组明显改善($P=0.008$)。在传统的 CV 通气下，呼吸机参数在安全范围内进行最大调整也不能满足 ARDS 患者通气和氧合的要求时，HFOV 被当作一种补救性通气模式。有两个研究[29-30]证实了这一点。Mehta 等[30]在一组 24 例严重 ARDS 患者〔肺损伤评分为(3.4±0.6)分[31]，PaO_2/FiO_2 为(98.8±39.0) mm Hg，1 mm Hg=0.133 kPa〕的研究中采用 CV 高平台压通气仍难以纠正低氧，转为 HFOV 治疗后 PaO_2/FiO_2 在 8 h 内改善。Fort 等[29]在一组 17 例严重 ARDS 患者〔肺损伤评分(3.81±0.23)分[31]，PaO_2/FiO_2 (68.6±21.6) mm Hg，急性生理学与慢性健康状况评分系统Ⅱ(APACHEⅡ)评分 23.3 分〕的研究中，采用 CV 治疗失败后〔吸入氧浓度(FiO_2)≥0.70，动脉血氧分压(PaO_2)≤65 mm Hg，或吸气峰压(PIP)＞65 mm Hg，或 PEEP≥15 cm H_2O〕转为 HFOV 治疗，其中 13 例患者的氧合明显改善。越来越多的证据表明，尽管 HFOV 还不能降低病死率，但比传统的通气模式能更好地提高氧合，给 ARDS 患者赢取宝贵的时机治疗原发病。

2.1.2 HFOV 联合其他辅助治疗：和传统的通气模式比较，尽管 HFOV 能够提高氧合，但还有一部分顽固性低氧患者在单纯 HFOV 治疗中仍不能改善，一些辅助治疗如俯卧位通气、肺复张(RM)、吸入性一氧化氮(iNO)等也都尝试在 HFOV 治疗中联合应用，以期待比单纯应用 HFOV 能进一步提高氧合。

2.1.2.1 俯卧位通气：俯卧位通气在 ARDS 患者中广为应用，研究显示能改善 60%ARDS 患者的氧合水平[32-33]，但目前 HFOV 联合俯卧位通气多见于一些个案报道[34]，其中 Papazian 等[35]将 39 例 ARDS 患者分为仰卧位 HFOV、俯卧位 HFOV 和俯卧位 CMV，结果显示，俯卧位通气两组患者氧合均有改善，但仰卧位 HFOV 组无改善。这与其他研究发现 HFOV 能改善氧合的结果[29-30,36]不一样，可能与 mPaw 给予不够有关(只有 25 cm H_2O)。最近一项前瞻性、随机对照临床研究发现：HFOV 序贯俯卧位通气能持续维持氧合改善，43 例 ARDS 患者(PaO_2/FiO_2＜150 mm Hg，PEEP≥5 cm H_2O)被随机分成 3 组：A 组行俯卧位 CV 12 h 后序贯仰卧位 CV 12 h，B 组行仰卧位 CV 12 h 后序贯 HFOV 仰卧位 12 h，C 组行俯卧位 CV 12 h 后序贯仰卧位 HFOV 12 h，C 组 PaO_2/FiO_2 较 A 组有明显的改善($P<0.02$)[37]，提示 HFOV 联合俯卧位通气可以进一步提高氧合水平。

2.1.2.2 RM：最近研究显示，RM 联合 HFOV 治疗既安全，又能快速提高并且维持氧合水平[38]。有研究显示，患者在接受 HFOV 治疗时氧合改善较慢，约需 8 h[21]，而联合 RM 则可加速肺泡复张。一个前瞻性多中心研究显示[39]，对 25 例早期 ARDS 患者进行规律性的联合 RM 治疗(40 cm H_2O×40 min)，当氧合下降(需要 FiO_2＞0.4)或每日至少 2 次 RM 时，与标准 CMV 相比，PaO_2/FiO_2 迅速上升(200 mm Hg 比 92 mm Hg，时间＜1.5 h)，而氧合改善速度较其他 HFOV 的研究结果[28-30]相对较快，同时 RM 也相对安全，仅有 3.3%最终失败，其原因主要是低血压。HFOV 联合 RM 的代表性操作方法是：如果患者出现脉搏血氧饱和度(SpO_2)下降≥0.05，或吸痰、纤维支气管镜检查、呼吸机管路断开后需行 RM，要预先将 mPaw 的高限报警重新设置(如 50 cm H_2O)，将气管插管气囊充气，松气囊(cuff leak)去除，将振荡活塞关闭，再将 mPaw 短暂设置在较前提高 10 cm H_2O，持续 40～60 s[40]。

2.1.2.3 iNO：有研究表明，ARDS 患者在应用 HFOV 中仍有持续低氧血症，应用 iNO 治疗可进一步改善氧合[34,41]。Mehta 等[41]给 23 例成人 ARDS 患者 HFOV 联合 iNO 治疗，5～20 次/min，结果 30 min 后平均 PaO_2/FiO_2 提高 38%，有 83%的患者(19 例)对 iNO 治疗改善氧合有效，而这一比例较其他研究报道 CMV 联合 iNO 治疗要高。

2.2 难治性高碳酸血症

2.2 难治性高碳酸血症：随着 ARDS 病情的进展，肺泡的损毁不断加重，可通气的肺泡越来越少，随之带来的高碳酸血症为避免进一步肺损伤而迫不得已被“允许”，只能用纠正代谢性酸中毒的方法进行干预，在 CV 时的“迫不得已”能在 HFOV 中解决吗？

与 CV 相比，HFOV 重要的优势是通气和氧合水平可以分别调整。由于通气水平可以在相当低的振荡压下维持，所以在比 CV 高的 mPaw 和比 CV 低的肺泡峰压下仍可维持患者通气功能。在特定的 mPaw 和频率下，排出 CO_2 的惟一机制是 ΔP 产生的 V_T，即振荡压力波动所产生的高频振荡气量清除 CO_2，ΔP 越大，CO_2 清除率越高；如果最大 ΔP 仍不能改善通气，可通过降低频率来实现。这与我们在 CV 调整参数时的习惯性思维是不同的，其原因是频率越低，就允许更长的肺泡通气时间使肺泡 V_T 增加。其他调整还包括更换大号人工气道，直径越大，人工气道对振荡波的衰减作用越小，可增加远端肺泡 V_T。如果高碳酸血症仍存在，还可以把吸气时间延长，如把吸气时间百分比(Ti%)调整为 50%，可使振荡气量增加 10%。由此我们相信，HFOV 对难治性高碳酸血症不一定只有“允许”，更多是迎刃而解。

尽管 HFOV 较 CV 能在不增加肺损伤的情况下提高 CO_2 的清除率，但在 HFOV 中已经应用最高的 ΔP 和最低的频率，而高碳酸血症(pH＜7.20)仍然难以控制时，最近很多研究报道可以尝试 cuff leak[42-44]，在 HFOV 通气中可以进一步提高 CO_2 清除率。当已经应用 cuff leak 后仍无法获得较低的动脉血二氧化碳分压($PaCO_2$)，可能因为在气管插管周围已经出现上气道水肿，可以额外放置口咽通气道以促使气体排出[45]。一些研究中心甚至对所有应用 HFOV 的患者从一开始时就给予 cuff leak，是否到难以控制的高碳酸血症时再用 cuff leak 仍值得商榷。但临床具体操作时须注意，在应用 cuff leak 之前一定吸净口腔和后咽部的分泌物，高、低 mPaw 报警线必须重新设置，应用 cuff leak 前将偏流提高 5 L/min，然后缓慢地松气囊，同时

监测呼吸机 mPaw 下降 5～7 cm H_2O，一旦达到满意的 cuff leak 后再重新设置为原先的 mPaw 报警[40]。目前，正是因为 HFOV 仍存在一些不足之处，并没有作为所有 ARDS 患者的常规通气模式，而是在传统的常频呼吸机不能维持基本通气要求时，HFOV 越来越多地作为一种补救性的通气模式来拯救 ARDS 患者的生命，正在被广大医师认可接受。在肺保护性通气策略大行其道的今天，HFOV 治疗机制能最大程度趋近于该理念，使得我们有理由相信，HFOV 能通过减少 VILI 来进一步降低 ARDS 患者的病死率，但这还需要大规模的临床研究来证实。HFOV 技术的最理想应用、最佳开始时机、最适患者的筛选、行之有效的床旁监测手段以避免并发症的发生仍然有待进一步探索明确。

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高频振荡通气的机制及临床应用

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