

压力控制法与呼气末正压递增法肺复张对百草枯致急性肺损伤家猪血流动力学的影响

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【摘要】 目的 探讨压力控制(PC)法与呼气末正压(PEEP)递增法肺复张(RM)对百草枯(PQ)中毒致急性肺损伤(ALI)家猪血流动力学的影响。方法 健康雌性家猪10只,经腹腔注射20% PQ溶液20 mL建立PQ中毒致ALI/急性呼吸窘迫综合征(ARDS)模型,制模成功后按随机数字表法将动物分为PC法RM组(RM1组)和PEEP递增法RM组(RM2组),每组5只。于制模前(基础值)、制模成功时及RM后5、15、30 min用脉搏指示连续心排血量(PiCCO)监测仪监测心率(HR)、平均动脉压(MAP)、心排血指数(CI),记录动脉血氧分压(PaO₂)及动脉血二氧化碳分压(PaCO₂),并计算氧合指数。于制模前、制模成功时及RM后30 min取肺组织,苏木素-伊红(HE)染色后光镜下观察肺组织病理学改变。结果 两组制模成功时HR、MAP、PaCO₂均较基础值明显上升,CI、PaO₂和氧合指数明显下降,符合ALI/ARDS表现。随RM时间延长,两组HR呈逐渐下降趋势,MAP、CI呈上升趋势;RM1组RM后5 min时HR、MAP明显低于RM2组[HR(次/min):126.8±5.2比134.0±3.8,MAP(mmHg, 1 mmHg=0.133 kPa):98.4±3.3比102.8±2.6,均P<0.05],RM后5 min和15 min时CI明显高于RM2组(mL·s⁻¹·m⁻²):56.7±5.0比46.7±6.7,65.0±5.0比56.7±5.0,均P<0.05)。RM后5 min,两组PaO₂和氧合指数均较制模成功时显著升高;随RM时间延长,两组PaO₂和氧合指数逐渐下降,PaCO₂逐渐升高,但两组各时间点间比较差异无统计学意义(均P>0.05)。RM后30 min两组均出现肺泡上皮细胞脱落、肺泡间隔进一步增宽及肺泡过度膨胀,部分可见肺泡间隔断裂;RM2组较RM1组肺泡间隔明显增宽,肺泡间隔断裂也更为多见。结论 PC法与PEEP递增法RM均可明显改善PQ中毒猪ALI/ARDS时的氧合状况,且PC法RM对血流动力学的影响更小。

【关键词】 压力控制法; 呼气末正压递增法; 肺复张; 百草枯; 中毒; 血流动力学; 猪

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Effects of pressure control and positive end-expiratory pressure incremental method lung recruitment maneuvers on haemodynamics in piglets with acute lung injury induced by paraquat Wang Jinzhu, Sun Renhua, Li Li, Lan Chao, Hu Bangchuan, Han Fang, Zheng Yang

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【Abstract】 Objective To discuss the effects of pressure control (PC) and positive end-expiratory pressure (PEEP) incremental method lung recruitment maneuver (RM) on haemodynamics in piglets with acute lung injury (ALI) induced by paraquat (PQ) poisoning. **Methods** The ALI/acute respiratory distress syndrome (ARDS) model was reproduced by intraperitoneal injection of 20% PQ (20 mL) in 10 healthy female piglets, and they were randomly divided into PC lung RM group (RM1 group) and PEEP incremental method lung RM group (RM2 group), with 5 piglets in each group. Heart rate (HR), mean arterial pressure (MAP), and cardiac index (CI) were monitored by pulse-indicated continuous cardiac output (PiCCO) monitoring before model reproduction (baseline), on the time of successfully set up of model and at 5, 15 and 30 minutes after RM. At the same time the arterial partial pressure of oxygen (PaO₂) and arterial partial pressure of carbon dioxide (PaCO₂) were recorded, and oxygenation index was calculated. Lung tissues were collected before model reproduction, on the time of successfully set up of model, and at 30 minutes after RM respectively, and pulmonary pathology changes were observed after hematoxylin and eosin (HE) staining under light microscopy. **Results** The HR, MAP, and PaCO₂ on the time of successfully set up of model in both groups were

increased obviously while CI, PaO₂, and oxygenation index were decreased obviously as compared with those at baseline, all of which conformed to the expression of ALI/ARDS. With RM time extended, the HR in both groups was declined while MAP and CI were increased gradually. The HR and MAP at 5 minutes after RM of RM1 group were significantly lower than those of the RM2 group [HR (bpm): 126.8±5.2 vs. 134.0±3.8, MAP (mmHg, 1 mmHg = 0.133 kPa): 98.4±3.3 vs. 102.8±2.6, both *P* < 0.05]. The CI at 5 minutes and 15 minutes after RM of RM1 group was significantly higher than that of the RM2 group (mL·s⁻¹·m⁻²: 56.7±5.0 vs. 46.7±6.7, 65.0±5.0 vs. 56.7±5.0, both *P* < 0.05). PaO₂ and oxygenation index at 5 minutes after RM in both groups were significantly higher than those on the time of successfully set up of model. The PaO₂ and oxygenation index were gradually decreased, and PaCO₂ was increased with RM time extended, but no statistically significant differences at all time points were found between the two groups (all *P* > 0.05). The lung tissue in both groups showed a variety of pathological changes at 30 minutes after RM. The main performances were the loss of alveolar epithelial cells, the further wideness of alveolar interval and the distension of alveolar, and the part breakage of alveolar interval. The wideness of alveolar interval was more significant in RM2 group than that of RM1 group, and alveolar cleft was more common too. **Conclusion** Both PC and PEEP incremental method lung RM can improve the oxygenation of the piglets with ALI/ARDS induced by PQ, and the PC lung RM has less impact on haemodynamics.

【Key words】 Pressure control method; Positive end-expiratory pressure incremental method; Lung recruitment maneuver; Paraquat; Poisoning; Haemodynamics; Piglet

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百草枯(PQ)中毒致肺损伤是导致患者死亡的主要原因, PQ中毒早期主要表现为急性肺损伤/急性呼吸窘迫综合征(ALI/ARDS), 而晚期则表现为进行性肺纤维化^[1]。已有研究证实, 采取肺保护性通气策略(LPVS)治疗PQ中毒所致ALI/ARDS取得了良好的效果^[2-3]。在LPVS基础上实施肺复张(RM)策略可以促进塌陷的肺泡复张, 并可改善氧合及呼吸力学和血流动力学指标^[4]; 杨万杰等^[5]研究证实, 患侧单肺RM可改善血流动力学和死腔比例, 且效果优于传统RM。但目前国内外对于不同策略RM对PQ中毒致ALI/ARDS的临床效果鲜有报道。本研究旨在比较压力控制(PC)法与呼气末正压(PEEP)递增法RM对PQ中毒致ALI/ARDS家猪血流动力学的影响。

1 材料与方 法

1.1 实验动物及模型制备: 65~70日龄健康雌性家猪10只, 体重(25.0±2.1)kg, 由河南省动物实验中心提供, 动物合格证号: 0008201。动物术前禁食12h, 自由饮水。参照本课题组前期研究方法, 经腹腔注射20mL 20% PQ溶液建立ALI/ARDS动物模型^[6]。

1.2 动物分组及处理: 制模成功后将动物按随机数字表法分为PC法RM组(RM1组)和PEEP递增法RM组(RM2组), 每组5只。两组动物均通气2min进行过渡, 呼吸机模式: 压力控制通气(PCV), PC 15 cmH₂O (1 cmH₂O=0.098 kPa), PEEP 10 cmH₂O,

呼吸频率(RR)30次/min, 吸入氧浓度(FiO₂)1.00。

1.2.1 RM1的实施: 采用PCV模式, RR 30次/min, 吸呼比(I:E)1:2, FiO₂ 0.50, PC 30 cmH₂O, PEEP 25 cmH₂O, 通气60s; RM结束后将呼吸机设置为容量控制通气(VCV)模式, 潮气量(VT)6 mL/kg, RR 30次/min, I:E为1:2, FiO₂ 0.50, PEEP 10 cmH₂O。

1.2.2 RM2的实施: 采用PCV模式, RR 30次/min, I:E为1:2, FiO₂ 0.50, 每30s上调5 cmH₂O PC和PEEP至PC为30 cmH₂O、PEEP为25 cmH₂O, 通气60s, RM结束后逐渐下调PC和PEEP, 每30s下调5 cmH₂O至PC 15 cmH₂O、PEEP 10 cmH₂O, 再将呼吸机调为与RM1组复张后相同的模式和参数。

1.3 监测指标及方法

1.3.1 血流动力学监测: 用脉搏指示连续心排量(PiCCO)监测仪监测制模前(基础值)、制模成功时及RM后5、15、30min的心率(HR)、平均动脉压(MAP)和心排血指数(CI)。

1.3.2 动脉血气分析: 于制模前、制模成功时及RM后5、15、30min记录动脉血氧分压(PaO₂)和动脉血二氧化碳分压(PaCO₂), 并计算氧合指数。

1.3.3 肺组织病理学观察: 于制模前、制模成功时及RM后30min取右肺下叶组织, 置于4%甲醛溶液中固定, 石蜡包埋、切片, 苏木素-伊红(HE)染色后在光镜下观察并拍照。

1.4 统计学方法: 使用SPSS 17.0软件进行统计学分析。符合正态分布的计量资料以均数±标准差

($\bar{x} \pm s$)表示,组间和组内数据比较采用独立样本 t 检验。 $P < 0.05$ 为差异有统计学意义。

2 结果

2.1 血流动力学变化(表 1): 两组间 HR、MAP 和 CI 基础值差异无统计学意义(均 $P > 0.05$)。两组制模成功时 HR、MAP 均较基础值明显升高,而 CI 则明显降低(均 $P < 0.05$)。随着 RM 时间延长,两组 HR 呈逐渐下降趋势,而 MAP 和 CI 则呈逐渐升高趋势。RM1 组 RM 后 5 min 时 HR 和 MAP 均明显低于 RM2 组,而 RM 后 5 min 和 15 min 时 CI 明显高于 RM2 组(均 $P < 0.05$)。

2.2 血气分析变化(表 1): 两组间 PaO₂、PaCO₂ 和氧合指数基础值差异无统计学意义(均 $P > 0.05$)。与基础值相比,两组制模成功时 PaO₂ 和氧合指数均明显下降,PaCO₂ 明显上升(均 $P < 0.05$)。RM 后 5 min 两组 PaO₂ 和氧合指数均显著高于制模成功时(均 $P < 0.05$);随 RM 时间延长,PaO₂ 和氧合指数逐

渐下降,PaCO₂ 逐渐升高,但两组各时间点间比较差异无统计学意义(均 $P > 0.05$)。

2.3 肺组织病理学改变(图 1): 模型动物肺泡组织明显充血、水肿,毛细血管肿胀,肺间质可见大量红细胞渗出,肺泡间隔增宽,部分透明膜形成。RM 后 30 min 表现为肺泡上皮细胞脱落,肺泡间隔进一步增宽及肺泡过度膨胀,部分肺泡间隔断裂;RM2 组肺泡间隔较 RM1 组明显增宽,肺泡间隔断裂也更为多见。

3 讨论

PQ 进入血液后蓄积在肺脏,由于肺内存在胺类摄取系统,其在肺脏的浓度可高于其他组织 10 倍以上^[7]。PQ 中毒后活性氧大量产生,细胞膜受到脂质过氧化损伤,引起肺泡毛细血管膜损伤,导致肺脏发生明显的病理学改变,如严重肺淤血、肺出血、细胞坏死、细胞透明膜变性等^[2],进而出现肺水肿、肺容积和肺通气量减少、肺顺应性降低以及通气/血

表 1 不同肺复张方法对 20% 百草枯中毒致急性肺损伤 / 急性呼吸窘迫综合征 (ALI/ARDS) 家猪血流动力学和血气分析指标的影响 ($\bar{x} \pm s$)

组别	时间	动物数 (只)	HR (次/min)	MAP (mmHg)	CI (mL·s ⁻¹ ·m ⁻²)	PaO ₂ (mmHg)	PaCO ₂ (mmHg)	氧合指数 (mmHg)
RM1 组	制模前	5	110.8 ± 7.4	99.4 ± 3.6	81.7 ± 11.7	166.6 ± 5.2	33.8 ± 2.5	555.3 ± 17.2
	制模成功时	5	128.2 ± 7.7 ^a	110.4 ± 3.9 ^a	63.3 ± 10.0 ^a	83.4 ± 3.7 ^a	42.6 ± 2.4 ^a	277.9 ± 12.4 ^a
	RM 后 5 min	5	126.8 ± 5.2	98.4 ± 3.3 ^b	56.7 ± 5.0 ^{ab}	253.2 ± 6.4 ^b	45.0 ± 2.9	506.4 ± 12.8 ^b
	RM 后 15 min	5	123.0 ± 7.0	104.2 ± 5.5 ^b	65.0 ± 5.0	198.6 ± 5.1 ^b	47.6 ± 2.5	397.2 ± 10.2 ^b
	RM 后 30 min	5	116.8 ± 4.3 ^b	108.0 ± 4.9	71.7 ± 11.7	163.4 ± 5.5 ^b	51.2 ± 3.6 ^b	326.8 ± 10.9 ^b
RM2 组	制模前	5	115.0 ± 5.2	103.2 ± 4.6	75.0 ± 13.3	170.2 ± 6.1	35.2 ± 2.9	567.3 ± 20.3
	制模成功时	5	131.6 ± 5.9 ^a	111.2 ± 4.1 ^a	61.7 ± 11.7 ^a	82.0 ± 3.2 ^a	43.6 ± 3.0 ^a	273.3 ± 10.5 ^a
	RM 后 5 min	5	134.0 ± 3.8 ^c	102.8 ± 2.6 ^{bc}	46.7 ± 6.7 ^{abc}	251.0 ± 7.8 ^b	46.8 ± 3.9	502.2 ± 15.6 ^b
	RM 后 15 min	5	126.8 ± 4.4	105.6 ± 4.7 ^b	56.7 ± 5.0 ^c	201.0 ± 7.1 ^b	48.8 ± 4.8	402.0 ± 14.1 ^b
	RM 后 30 min	5	114.4 ± 8.7 ^b	110.6 ± 3.7	70.0 ± 10.0	165.0 ± 4.9 ^b	50.6 ± 3.3 ^b	330.0 ± 9.8 ^b

注: RM1 组为压力控制法肺复张组, RM2 组为呼气末正压递增法肺复张组; HR 为心率, MAP 为平均动脉压, CI 为心排血指数, PaO₂ 为动脉血氧分压, PaCO₂ 为动脉血二氧化碳分压; 1 mmHg = 0.133 kPa; 与本组制模前(基础值)比较, ^a $P < 0.05$; 与本组制模成功时比较, ^b $P < 0.05$; 与 RM1 组同期比较, ^c $P < 0.05$

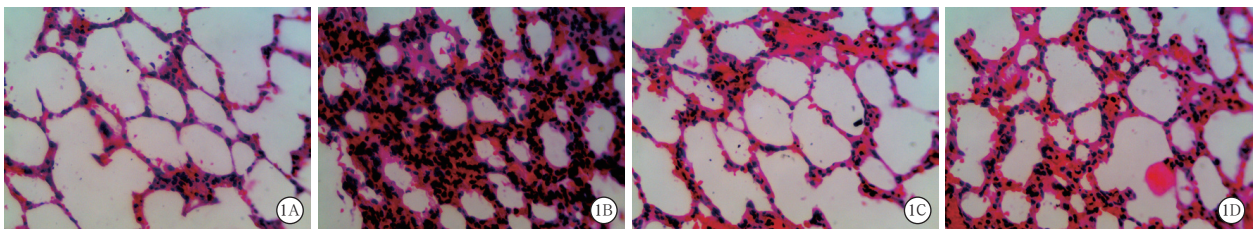


图 1 光镜下观察各组 20% 百草枯中毒致急性肺损伤 / 急性呼吸窘迫综合征 (ALI/ARDS) 家猪肺组织病理学改变 制模前(A)肺组织结构正常;制模成功时(B)肺泡组织明显充血、水肿,毛细血管肿胀,肺间质可见大量红细胞渗出,肺泡间隔增宽,部分透明膜形成;压力控制法肺复张组(RM1 组,C)和呼气末正压递增法肺复张组(RM2 组,D)复张后 30 min 肺泡上皮细胞脱落,肺泡间隔进一步增宽,肺泡过度膨胀,部分肺泡间隔断裂,且 RM2 组肺泡间隔破坏更为明显 HE 染色 高倍放大

流比例严重失调等 ARDS 病理学改变^[8]。

LPVS 是 ALI/ARDS 的有效治疗策略,主张实施小 VT (6~8 mL/kg) 通气和允许性高碳酸血症,同时使用一定的 PEEP 使肺泡保持开放状态^[9-10]。近年来研究证实,单纯实施 LPVS 并不能使已经塌陷的肺泡复张,而且小 VT 通气可以加重肺泡萎陷^[11]。但本课题组前期研究证实 LPVS 能明显改善 PQ 中毒引起 ALI 猪的氧合状况^[12]。李家琼等^[13]发现,小 VT + 最佳 PEEP + 控制性肺膨胀(SI)组(LVBP + SI 组)ARDS 模型家兔制模后氧合指数显著高于常规 VT + 0 PEEP 组(MVZP 组)和小 VT + 0 PEEP 组(LVZP 组),且 HR、血压均无明显变化。PC 法和 PEEP 递增法是研究较多的两种 RM 方式^[14-15]。本研究发现,ALI/ARDS 模型猪氧合指数 ≤ 300 mmHg (1 mmHg=0.133 kPa) 时肺组织表现出明显的损伤性改变,与国内杨舟和沈锋^[16]观察到的 ALI/ARDS 时炎性细胞浸润、肺泡塌陷、透明膜形成等病理学改变一致;RM 后两组 PaO₂ 和氧合指数显著升高;随 RM 时间延长,PaO₂ 和氧合指数逐渐下降,PaCO₂ 逐渐上升,但两组差异无统计学意义,表明在 LPVS 基础上实施 RM 可以改善氧合状况,且不同 RM 方式改善作用相当。

研究表明,RM 时由于胸腔内压升高会对血流动力学造成不同程度的影响,且不同 RM 实施方式的影响程度也有所不同^[17]。熊旭明等^[18]对肺内、外源性 ARDS 模型犬分别实施 RM 后发现,两组平均肺动脉压(MPAP)、中心静脉压(CVP)和肺动脉楔压(PAWP)均显著增加,而 MAP 和 CI 明显下降,且均在 RM 后 30 min 左右恢复至基础水平。徐磊等^[19]对 ARDS 绵羊模型实施 SI 策略并设置不同水平的 PEEP 后发现,PEEP ≥ 15 cmH₂O 时 CVP 明显升高且 CI 明显降低,因而主张 RM 后将 PEEP 设置在 10~20 cmH₂O 即可有效改善氧合状况,且对血流动力学影响较小。本研究中两组 PEEP 的设置相同(均为 10 cmH₂O),但 PC 法 RM 对血流动力学的影响较小,尤其是在 RM 后 5 min 两组间 HR、MAP、CI 显示出明显的统计学差异,但在 RM 后 30 min 均恢复至基础水平。分析原因为:PC 法 RM 是一次性将 PC 和 PEEP 调至高水平状态,而 PEEP 递增法 RM 则是逐步上调 PC 和 PEEP,每次调整均会对胸腔内压产生一定影响,因而 PEEP 递增法 RM 更容易引起 HR、MAP、CI 改变。

综上,PC 法和 PEEP 递增法 RM 均可有效改善

PQ 中毒致 ALI/ARDS 家猪的氧合状况,且 PC 法对血流动力学的影响较小。本研究仅观察了单次复张的效果,而临床上往往需要多次复张才能取得较好的疗效^[20],因而还需要更多研究进一步证实。

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• 科研新闻速递 •

复杂性腹腔感染合并脓毒症患者不需要进行长疗程的抗菌药物治疗

最近一项多中心前瞻性随机对照试验(RCT)发现,与长疗程抗菌药物治疗方案相比,短疗程抗菌药物治疗方案(控制腹腔感染后连续使用4 d 抗菌药物)对复杂性腹腔感染合并脓毒症患者同样有效。为验证这一结果,研究人员对SPOP-IT临床试验(Study to Optimize Peritoneal Infection Therapy)中符合脓毒症诊断标准(体温 $<36\text{ }^{\circ}\text{C}$ 或 $>38\text{ }^{\circ}\text{C}$,白细胞计数 $<4.0\times 10^9/\text{L}$ 或 $>12.0\times 10^9/\text{L}$)患者的数据进行了分析。在SPOP-IT试验中患者被随机分为两组,一组持续接受抗菌药物治疗至患者体温、白细胞恢复正常,肠梗阻解除(最长时间达10 d, $n=45$);另一组接受短疗程抗菌药物治疗方案[平均 $(4\pm 1)\text{ d}$, $n=67$]。评价指标包括:切口感染率及时间、腹腔感染复发率、艰难梭菌感染率、腹腔外感染率、住院时间及病死率。结果显示:SPOP-IT试验588例患者中共有122例符合此次纳入标准,短疗程组和长疗程组患者切口感染率分别为11.9%和8.9% ($P=0.759$),腹腔感染复发率分别为11.9%和13.3% ($P=1.000$),腹腔外感染率分别为11.9%和8.9% ($P=0.759$),住院时间分别为 $(7.4\pm 5.5)\text{ d}$ 和 $(9.0\pm 7.5)\text{ d}$ ($P=0.188$),腹腔感染复发时间分别为 $(12.5\pm 6.6)\text{ d}$ 和 $(18.0\pm 8.1)\text{ d}$ ($P=0.185$),腹腔外感染复发时间分别为 $(12.6\pm 5.8)\text{ d}$ 和 $(17.3\pm 3.9)\text{ d}$ ($P=0.194$),病死率分别为1.5%和0 ($P=1.000$)。两组患者均未发现有艰难梭菌感染的情况。短疗程组患者切口感染的发生时间要早于长疗程组($d: 6.9\pm 3.5$ 比 21.3 ± 6.1 , $P<0.001$)。研究人员据此得出结论:长疗程和短疗程两种抗菌药物治疗方案对复杂性腹腔感染合并脓毒症患者的疗效相近,因此,只要腹腔内感染源得以控制,并不需要进行长疗程的抗菌药物治疗。

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