

## • 短篇论著 •

## 体外腹主动脉按压对老年患者麻醉诱导时循环的影响

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**【摘要】** 目的 探讨体外腹主动脉按压对老年患者麻醉诱导时循环的影响。方法 采用前瞻性随机对照研究方法, 将2017年1月至4月武警总医院收治的美国麻醉医师协会(ASA)分级Ⅱ~Ⅲ级、拟行全麻非腹部手术的60~75岁老年患者按随机数字表法分为腹主动脉按压组和对照组, 每组20例。两组均予以咪唑安定、丙泊酚、芬太尼和顺式阿曲库铵诱导麻醉, 丙泊酚、瑞芬太尼、顺式阿曲库铵维持麻醉, 插管成功后接麻醉机行机械通气; 腹主动脉按压组则在咪唑安定注入1 min后给予腹主动脉按压至插管后5 min。分别于麻醉诱导前、诱导后、插管后即刻、插管后5 min和10 min监测平均动脉压(MAP)、心率(HR)和脉搏血氧饱和度(SpO<sub>2</sub>)等, 观察低血压、心动过缓等不良反应发生情况和麻黄碱、阿托品的使用情况。结果 腹主动脉按压组和对照组患者麻醉诱导前MAP [mmHg (1 mmHg=0.133 kPa): 83.6±4.7、82.9±4.7]、HR (次/min: 67.3±5.9、65.9±5.7)、SpO<sub>2</sub> (0.962±0.007、0.960±0.009) 差异均无统计学意义(均P>0.05)。麻醉诱导后, 对照组MAP、HR较诱导前明显下降[MAP(mmHg): 70.0±8.7比82.9±4.7, HR(次/min): 60.7±6.7比65.9±5.7, 均P<0.05], 且明显低于腹主动脉按压组[分别为(83.1±3.9)mmHg、(66.8±4.9)次/min, 均P<0.05]。气管插管后即刻, 对照组MAP、HR较麻醉诱导后明显升高[MAP(mmHg): 78.9±7.9比70.0±8.7, HR(次/min): 67.3±2.7比60.7±6.7, 均P<0.05], 而腹主动脉按压组MAP、HR变化不明显。两组麻醉诱导期间SpO<sub>2</sub>变化差异无统计学意义。对照组麻醉诱导期间4例出现低血压, 其中2例使用了麻黄碱; 2例出现心动过缓并使用了阿托品。而腹主动脉按压组麻醉诱导期间未见不良反应发生。结论 老年患者麻醉诱导时行腹主动脉按压有助于维持血流动力学稳定。

**【关键词】** 腹主动脉按压; 麻醉; 血流动力学; 老年

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### Effect of external abdominal aorta compression on circulation during anesthesia induction in elderly patients

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**【Abstract】 Objective** To investigate the effect of external abdominal aorta compression on circulation during anesthetic induction in elderly patients. **Methods** A prospective randomized controlled trial was conducted. Patients with age of 60–75 years old, requiring a general anesthesia for non-abdominal surgery, and with II–III class of American Society of Anesthesiologists (ASA) physical status classification, and admitted to General Hospital of Chinese People's Armed Police Forces from January to April in 2017 were enrolled. They were divided into abdominal aorta pressure group and control group according to random number method, with 20 patients in each group. In both groups, anesthesia was induced with midazolam, propofol, fentanyl and cisatracurium, and was maintained with propofol, remifentanyl and cisatracurium. After successful intubation, the anesthesia machine was changed into mechanical ventilation. The patients in abdominal aorta pressure group were given abdominal aorta pressure 1 minute after induction of general anesthesia with midazolam till 5 minutes after intubation. The mean arterial pressure (MAP), heart rate (HR) and blood oxygen saturation (SpO<sub>2</sub>) were observed before anesthesia induction, immediately after anesthesia induction, immediately after intubation, 5 minutes and 10 minutes after intubation, respectively. The incidence of hypotension or bradycardia, and usage of ephedrine or atropine were recorded. **Results** There were no significant differences in MAP [mmHg (1 mmHg = 0.133 kPa): 83.6±4.7 vs. 82.9±4.7], HR (bpm: 67.3±5.9 vs. 65.9±5.7) and SpO<sub>2</sub> (0.962±0.007 vs. 0.960±0.009) before anesthesia induction between abdominal aorta pressure group and control group (all P > 0.05). Immediately after anesthesia induction, the MAP and HR in control group were significantly decreased as compared with those before anesthesia induction [MAP (mmHg): 70.0±8.7 vs. 82.9±4.7, HR (bpm): 60.7±6.7 vs. 65.9±5.7, both P < 0.05], and they were also significantly lower than those of abdominal aorta pressure group [MAP (mmHg): 83.1±3.9, HR (bpm): 66.8±4.9, both P < 0.05]. Immediately after intubation, the MAP and HR in control group were significantly

increased as compared with those immediately after anesthesia induction [MAP (mmHg): 78.9 ± 7.9 vs. 70.0 ± 8.7, HR (bpm): 67.3 ± 2.7 vs. 60.7 ± 6.7, both *P* < 0.05], but the changes in MAP and HR in abdominal aorta pressure group were not obvious. During the anesthesia induction period, there was no statistical difference in SpO<sub>2</sub> change between the two groups. During induction of anesthesia, no adverse reaction was found in the abdominal aorta pressure group, but 4 patients with hypotension and 2 patients with bradycardia were found in the control group. Two patients with hypotension were treated with ephedrine, and 2 patients with bradycardia were treated with atropine. **Conclusion** Anesthesia induction of elderly patients with abdominal aorta pressure can help maintain hemodynamic stability.

**【Key words】** Abdominal aorta compression; Anesthesia; Hemodynamics; Elderly patient

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全麻诱导气管插管可引起血压增高和心率加快,而麻醉诱导药物在减轻插管应激的同时,也会导致心血管系统抑制。老年人心血管系统功能储备明显减退,循环代偿能力下降,在麻醉诱导期间循环系统反应更加明显<sup>[1]</sup>。麻醉诱导期间大幅度的循环波动可能导致心脑血管意外的发生<sup>[2]</sup>。如何保证有效抑制全麻插管的心血管反应,同时保证血流动力学稳定,是老年患者麻醉诱导期间防治缺血性心脑血管病变的关键<sup>[3]</sup>。腹主动脉是人体的大动脉,直接延续于发自左心室的主动脉、胸主动脉,沿脊柱左侧下行,主要负责腹腔器官和腹壁的血供。腹主动脉阻断术及腹主动脉起搏术已经广泛用于临床<sup>[4-6]</sup>。有动物实验显示,腹主动脉按压能提高心室纤颤(室颤)猪的冠状动脉灌注压(CPP)和自主循环恢复(ROSC)率<sup>[7]</sup>,但鲜见临床报道。本研究旨在观察体外腹主动脉按压对老年患者麻醉诱导时循环的影响,为体外腹主动脉按压的临床应用提供依据。

1 资料与方法

1.1 一般资料:采用前瞻性随机对照研究方法,选择2017年1月至4月武警总医院非腹部手术患者40例。

1.1.1 纳入标准:美国麻醉医师协会(ASA)分级II~III级;全麻非腹部手术患者;年龄60~75岁;性别不限。

1.1.2 排除标准:有腹部手术史或腹部疾病;肥胖;伴有高血压、糖尿病等疾病者。

1.1.3 伦理学:本研究符合医学伦理学标准,并经医院伦理委员会批准(审批号:2017-07-04),所有治疗及处理均获得患者和家属的知情同意。

1.2 分组及麻醉方法:将40例择期非腹部手术患者按随机数字表法分为腹主动脉按压组和对照组,每组20例。术前禁食12h、禁水8h,拟选择全身麻醉。患者于手术室后连接心电监护,建立外周静脉通路,行左侧桡动脉有创测压。腹主动脉按压组予咪唑安定0.1mg/kg、丙泊酚2mg/kg、芬太尼4μg/kg、顺阿曲库铵0.15mg/kg诱导麻醉,在咪唑安定注入1min后给予腹主动脉按压<sup>[8]</sup>至插管后5min,插管成功后接麻醉机行机械通气,通气参数设置:潮气量8mL/kg,呼吸频率12次/min。麻醉维持:丙泊酚4mg·kg<sup>-1</sup>·h<sup>-1</sup>,瑞芬太尼0.2μg·kg<sup>-1</sup>·min<sup>-1</sup>,顺式阿曲库铵1.5μg·kg<sup>-1</sup>·min<sup>-1</sup>,维持血压波动不超过基础值的20%。平均动脉压(MAP) < 60mmHg(1mmHg = 0.133kPa),则给予麻黄碱10mg;

心率(HR) < 50次/min,则给予阿托品0.5mg。对照组单纯用咪唑安定、丙泊酚、芬太尼和维库溴铵诱导麻醉,给药剂量和顺序、麻醉维持及处理均同腹主动脉按压组。

1.3 观察指标:监测并记录麻醉诱导前、诱导后、插管后即刻、插管后5min和10min的MAP、HR及脉搏血氧饱和度(SpO<sub>2</sub>)等,记录低血压和心动过缓等不良反应发生情况以及麻黄碱和阿托品的应用情况。各指标均由一位不知分组情况的观察员记录。

1.4 统计学分析:采用SPSS 17.0软件进行数据分析。先对计量资料进行正态性检验,正态分布的计量资料以均数 ± 标准差( $\bar{x} \pm s$ )表示,组间比较采用*t*检验;非正态分布的计量资料以中位数(四分位数)[*M*(*Q*<sub>L</sub>, *Q*<sub>U</sub>)]表示,组间比较采用非参数检验;计数资料比较采用χ<sup>2</sup>检验。*P* < 0.05为差异具有统计学意义。

2 结果

2.1 两组患者一般资料比较(表1):40例患者均纳入最终分析,其中男性30例,女性10例;年龄(67.4 ± 4.0)岁;体重(63.4 ± 6.3)kg;身高(167.8 ± 6.4)cm;诊断:甲状腺肿瘤8例,眼眶肿瘤16例,下肢手术16例。两组患者性别、年龄、身高、体重及ASA分级差异均无统计学意义(均*P* > 0.05),说明两组基线资料均衡,具有可比性。

2.2 两组患者不同时间点MAP、HR和SpO<sub>2</sub>比较(表2):在麻醉诱导前,两组MAP、HR和SpO<sub>2</sub>差异均无统计学意义(均*P* > 0.05)。对照组麻醉诱导后MAP、HR较诱导前明显下降,插管后即刻则均较诱导后明显升高(均*P* < 0.05),并维持该水平至插管后10min;腹主动脉按压组诱导后MAP、HR略有降低,插管后即刻有所增高,但变化幅度不大;对照组诱导后MAP、HR明显低于腹主动脉按压组(均*P* < 0.05)。两组各时间点SpO<sub>2</sub>均无明显变化。

2.3 两组患者诱导期间不良反应比较:麻醉诱导期间对照组4例出现低血压,其中2例使用了麻黄碱;2例出现心动过缓并使用了阿托品。腹主动脉按压组患者均未出现低血压和心动过缓现象。

表1 两组老年非腹部手术患者一般资料比较

组别	例数(例)	性别(例)		年龄(岁, $\bar{x} \pm s$ )	体重(kg, $\bar{x} \pm s$ )	身高(cm, $\bar{x} \pm s$ )	ASA分级(例)	
		男性	女性				II级	III级
腹主动脉按压组	20	14	6	66.8 ± 4.1	64.2 ± 6.0	168.9 ± 5.2	15	5
对照组	20	16	4	67.0 ± 3.9	62.7 ± 6.5	166.7 ± 7.3	14	6

注:ASA为美国麻醉医师协会

表2 腹主动脉按压对老年非腹部手术患者麻醉诱导期间MAP、HR及SpO<sub>2</sub>的影响( $\bar{x} \pm s$ )

组别	例数(例)	MAP(mmHg)				
		诱导前	诱导后	插管后即刻	插管后5 min	插管后10 min
腹主动脉按压组	20	83.6±4.7	83.1±3.9	82.7±4.0	81.4±3.7	83.7±2.7
对照组	20	82.9±4.7	70.0±8.7 <sup>ab</sup>	78.9±7.9 <sup>a</sup>	79.1±4.1	82.3±3.1
组别	例数(例)	HR(次/min)				
		诱导前	诱导后	插管后即刻	插管后5 min	插管后10 min
腹主动脉按压组	20	67.3±5.9	66.8±4.9	67.4±4.1	67.3±2.9	66.4±2.1
对照组	20	65.9±5.7	60.7±6.7 <sup>ab</sup>	67.3±2.7 <sup>a</sup>	65.6±2.5	64.9±2.7
组别	例数(例)	SpO <sub>2</sub>				
		诱导前	诱导后	插管后即刻	插管后5 min	插管后10 min
腹主动脉按压组	20	0.962±0.007	0.984±0.007	0.986±0.005	0.985±0.006	0.983±0.008
对照组	20	0.960±0.009	0.983±0.009	0.985±0.006	0.985±0.005	0.983±0.007

注:MAP为平均动脉压,HR为心率,SpO<sub>2</sub>为脉搏血氧饱和度;与本组前一时间点比较,<sup>a</sup> $P<0.05$ ;与腹主动脉按压组比较,<sup>b</sup> $P<0.05$ ;1 mmHg=0.133 kPa

### 3 讨论

及时精确监测麻醉时的血压,对老年患者围手术期安全至关重要<sup>[9]</sup>。本研究中对老年非腹部手术患者均行左侧桡动脉有创测压,结果显示,对照组麻醉诱导后血压明显下降、心率明显减慢,气管插管后血压明显升高、心率明显加快;而腹主动脉按压组麻醉诱导后和气管插管时血压、心率变化均不大。说明麻醉诱导时按压腹主动脉能有效阻断下向血流,从而提升上肢血压,有助于维持血流动力学稳定。

丙泊酚是目前国内外应用较广的静脉麻醉药,其诱导剂量一般为1.0~2.5 mg/kg,对血流动力学影响较大,特别是对老年人,其心血管抑制作用更为显著<sup>[10]</sup>。本研究中观察到,对照组患者在麻醉诱导后血压出现大幅下降,其中4例出现低血压,2例在插管后尚未恢复,使用了麻黄碱升压;2例出现心动过缓,使用阿托品后得以纠正。腹主动脉按压组患者在麻醉诱导期间无明显血压下降和心率减慢,说明腹主动脉按压阻断了下肢血流,一方面增加了上半身的循环血量,另一方面也减弱了丙泊酚的血管扩张作用,从而保证了血压的稳定。

早在1967年,Harris等<sup>[11]</sup>就提出腹带复苏术。Lottes等<sup>[12]</sup>认为,腹带复苏术可以提高CPP,且方法简单、无创、可控性强。但Park等<sup>[13]</sup>发现持续性腹部按压有发生肝脏损伤的可能。我们发现在腹主动脉的中点进行阻断,损伤血管的可能性最小。

Baele等<sup>[14]</sup>在1991年采用体外腹主动脉按压法逆转了法洛四联征患儿的临床危象。但临床上多采用药物纠正血压下降,而升压药可引起全身性血管收缩,导致微循环缺氧。腹主动脉阻断术也被应用于盆腔及下肢大出血等紧急情况,但需要一定的技术和时间才能实施。体外腹主动脉按压具有起效快、简便易行的优点,只需熟悉解剖学、掌握方法就能快速有效地阻断下向血流,从而保证上向血流,达到止血或提升血压的目的。动物实验表明,腹主动脉按压在心肺复苏(CPR)领域也有广泛的应用前景<sup>[7]</sup>。

心搏骤停时CPP是评价CPR效果最重要的血流动力学

指标<sup>[15]</sup>。Zhou等<sup>[7]</sup>报道持续性腹主动脉按压能够升高室颤猪的CPP和ROSC率,其效果与肾上腺素相当。Wang等<sup>[16]</sup>研究表明,持续性腹主动脉按压可以增加窒息性心搏骤停兔的CPP,改善动物预后。本课题组就腹主动脉按压进行了一系列的动物实验研究,结果显示,腹主动脉按压可以提高心搏骤停兔的MAP和CPP,以及复苏成功率<sup>[17-18]</sup>。但腹主动脉按压的临床应用尚为首次探讨。

综上,老年患者麻醉诱导时行腹主动脉按压有助于维持血流动力学稳定,在急救领域具有较

高的应用价值。由于本试验样本量小,可能存在抽样误差,而且对于按压时间窗无明确界定,存在一定的局限性,尚需大样本临床研究证实。

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• 科研新闻速递 •

右美托咪定对需机械通气的脓毒症患者病死率及无机械通气天数的影响：一项随机对照试验

右美托咪定对机械通气患者有镇静作用,但其对脓毒症患者病死率及无机械通气天数的影响尚不明确,为此,日本学者进行了一项多中心随机对照试验。研究对象为 2013 年 2 月至 2016 年 1 月收治于日本 8 个重症加强治疗病房 (ICU) 中需要机械通气 24 h 以上的成年脓毒症患者。受试对象被随机分为右美托咪定镇静组和对照组。对照组的用药有芬太尼、丙泊酚和咪达唑仑;右美托咪定镇静组在对照组的基础上应用右美托咪定镇静。主要评价指标为患者 28 d 病死率和无机械通气天数;其他评价指标包括 1、2、4、6、8 d 序贯器官衰竭评分 (SOFA),以及镇静控制情况、谵妄和昏迷发生情况、ICU 住院时间、肾功能、炎症及营养状态。结果显示:受试对象平均年龄为 69 岁,男性占 63%。右美托咪定镇静组和对照组患者 28 d 病死率差异无统计学意义 [ 22.8% 比 30.8%, 风险比 (HR) = 0.69, 95% 可信区间 (95%CI) = 0.38 ~ 1.22, P = 0.20 ], 两组患者无机械通气天数差异也无统计学意义 (d: 20 比 18, P = 0.20)。右美托咪定镇静组达到良好镇静效果的患者比例明显高于对照组 (P = 0.01)。两组其他评价指标差异均无统计学意义。右美托咪定镇静组和对照组不良反应事件发生率分别为 8% 和 3%。研究人员据此得出结论:对于需要机械通气的脓毒症患者,使用右美托咪定镇静并不能降低患者病死率,也不能增加无机械通气天数。

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替代性复苏策略、损伤性生物标志物与脓毒性休克

早期脓毒性休克规范化治疗试验 (ProCESS 试验) 表明,替代性复苏策略在不同原因导致死亡患者间没有差异。近期,美国学者对该研究进行了二次分析,旨在了解复苏策略的差异是否影响与脓毒症临床预后相关的关键生物标志物,以及对不同基线水平生物标志物而言,各治疗组间的存活率是否存在差异。ProCESS 试验的研究对象来自美国 31 家医院的 628 例脓毒性休克患者。该试验的干预方法为两种替代性复苏方案与常规方案。研究者于治疗 0、6、24、72 h 观察了 4 类生物标志物:包括炎症指标肿瘤坏死因子-α (TNF-α)、白细胞介素 (IL-6、IL-10);凝血指标 D-二聚体、凝血酶-抗凝血酶复合物;氧化应激指标尿异前列腺素及反映组织缺氧指标血乳酸;并分析了 72 h 后替代性复苏策略是否影响生物标志物水平及 90 d 住院病死率。结果显示,在生物标志物基线水平时 (0 h),高浓度生物标志物与患者 90 d 死亡风险增加相关。然而,不同治疗组对随后的生物标志物变化没有显著影响。研究者还发现,IL-6 和 IL-10 基线水平下四分位数的患者接受替代治疗的疗效要优于常规治疗。研究人员据此得出结论:在脓毒性休克患者中,炎症、凝血、氧化应激和组织缺氧的生物标志物变化与预后不良相关,但常规治疗与替代治疗之间并无明显差异。然而,与预期相反,基于规范方案的复苏似乎在具有较低浓度的炎症生物标志物的患者中效果更优,但这一效应机制尚不清楚。

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