专家述评

全身麻醉诱导后低血压的发生机制与危险因素

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【摘　要】全身麻醉诱导后低血压（post-induction hypotension, PIH）是全身麻醉诱导期间的常见并发症，动脉血压低于血管自动调节曲线的下限可能会导致心、脑、肾等重要器官缺血，PIH与病人不良结局之间存在联系，有研究表明当收缩压（systolic pressure，SBP）比术前基线下降40%～45%时，老年人的死亡率较高；术中低血压易致肌钙蛋白升高，并与30 d死亡率增加相关。对于全身麻醉诱导期间可接受的最低血压尚无共识，包括平均动脉血压（mean arterial blood pressure，MAP）下降＞40%，且MAP＜70 mmHg或MAP＜60 mmHg；动脉血压比术前基线值下降30%；SBP下降小于90 mmHg等。因此，识别与PIH发生相关的风险因素有助于避免和及时处理PIH。本文就全身麻醉PIH的发生机制与危险因素做一述评，以期为预防PIH的发生提供新的思路，进而提升临床麻醉的安全与品质。

【关键词】全麻诱导；诱导后低血压；危险因素
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Mechanism and risk factors of hypotension induced by general anesthesia

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【**Abstract**】Post-induction hypotension (PIH) of general anesthesia is a common complication during induction of general anesthesia. The decrease of arterial blood pressure less than the lower limit of the auto-regulation curve of blood vessels may lead to ischemia of heart, brain, kidney and other important organs. There is a link between PIH and the adverse outcome of patients. Some studies showed that when the systolic pressure (SBP) was 40% ~ 45% lower than the preoperative baseline, the mortality of the elderly was higher; Intraoperative hypotension contributed to the elevation of troponin and was associated with an increase in 30-day mortality. There is no consensus on the acceptable minimum blood pressure during the induction of general anesthesia, including the decrease of mean arterial blood pressure (MAP) > 40%, and MAP < 70 mmHg or MAP < 60 mmHg; Arterial blood pressure decreased by 30% for preoperative baseline; SBP drop is less than 90 mmHg, etc. Therefore, identifying the risk factors associated with PIH helps to avoid and deal with PIH in a timely manner. This article reviews the relevant contents of PIH in general anesthesia, with a view to providing ideas for the prevention of PIH and improving the quality of clinical anesthesia.

【**Key words**】Induction of general anesthesia; Post-induction hypotension; Risk factors



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全身麻醉诱导后低血压（post-indnction hypotension PIH）是指在全身麻醉下接受手术的病人，于麻醉诱导后至手术切皮前发生的血压过低[1]。PIH一般发生在全麻诱导后20 min内，主要是由于全麻药物抑制心功能，外周血管扩张，此期间手术刺激缺失所致[2]。麻醉医师常因调整呼吸机、记录麻醉事件、安置病人等原因，忽视PIH，延误处理从而导致许多不良后果[3-6]。目前诱导后低血压的判断标准尚未统一，既往研究显示PIH发生率存在较大波动（5%～99%）[7]，主要原因是PIH缺乏一个公认的定义，GREEN和BUTLER定义为收缩压低于80 mmHg或收缩压比基线值下降20%以

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上，或平均动脉压（mean atery pressure，MAP）低于60 mmHg[8]，SÜDFELD等定义为收缩压下降至低于90 mmHg或需要输注去甲肾上腺素高于6 ug/min[9]，BEECHAM等定义为收缩压降低至基线的80%以下[10]。

1 PIH危害

PIH是临床上常见的全麻诱导期间麻醉并发症，由于动脉血压低于血管自动调节曲线的下限可能导致重要器官（心脏、大脑和肾脏等）缺血[11-14]。许多临床研究已证明PIH与器官功能损害（如术后急性肾损伤、心肌损伤及术后30 d死亡率）存在关联[15-17]。一些研究表明：当术中MAP < 55 mmHg时，急性肾损伤和急性心肌损伤发生率显著增加[18]。在非心脏手术指南中，美国心脏病学会（ACC）和美国心脏协会（AHA）建议对手术病人的术前并发症进行个体化管理，这些研究和共识均强调了围术期血压管理的重要性[19-20]。

2 PIH发生机制

PIH的确切病理生理机制仍未完全明确，手术中发生低血压影响因素很多，可能是出血、手术操作、麻醉药物等诸多原因联合影响的结果[21]。而在各种类型手术中，术中低血压最常在全身麻醉诱导后出现，这与术中出血量、手术时间或手术侵入性操作均无关，PIH主要是由病人自身的身体状态以及麻醉管理所导致[22-23]。既往研究证明PIH发生机制是由多因素所致，包括麻醉前因禁饮禁食时间过长或体液丢失所致低循环血容量、麻醉药物抑制心肌作用和外周血管扩张作用，以及病人自身相关危险因素等可能共同导致了PIH[24-25]。

3 PIH危险因素

由于全麻诱导后低血压发病机制尚未完全明确，目前针对危险因素进行预防、尽早干预非常重要。危险因素如何分类，尚无统一标准，大体分为全麻诱导前、全麻诱导期间两种情况，但某些危险因素可贯穿整个围术期。

3.1 高龄

研究表明年龄是PIH的预测因素，REICH等发现麻醉诱导时发生低血压的危险因素是年龄 ≥ 50岁[26]；ZHANG等也在66岁人群的多元回归分析中发现年龄与PIH的发生呈正相关，即病人年龄越大，发生PIH的可能性越大[27]。老年病人麻醉诱导期间血压降低的机制可能与衰老导致动脉血管的结构变化，心脏收缩力降低，相对循环血容量不足以及血流动力学调节能力减弱有关[28-31]。因此，当围术期使用全麻药物时，老年特别是高龄病人更易发生PIH。

3.2 性别

有研究表明合并高血压的女性病人易发生PIH，原因可能与血容量相对较低，循环系统的代偿能力相对较弱有关[32]。据报道，在围术期应激期间，女性的交感神经与血管阻力反应相对减弱，尤其是当病人处于低血容量状态时[33]。因此，对性别差异的发现可能反映了自主神经功能的差异。此外，自主神经活动的变化可能与女性病人的体脂率（BMI）相关，有研究表明，BMI较低的病人PIH发生率更高，高BMI可能是PIH的保护因素之一[31,33-34]。

3.3 ASA分级

现有研究表明，美国麻醉医师协会（ASA）分级与PIH密切相关[5,35]。进一步研究也证明ASA Ⅲ或Ⅳ级病人PIH的发生率明显高于ASA Ⅰ或Ⅱ级病人，这是因为ASA等级较高的病人通常年龄较大，身体基础状态差且伴有重度的系统性疾病，有一定的器官脏器功能受损，对药物的耐受性更低，并且神经-体液调节系统能力也相对更弱[6]，REICH等报道了ASA Ⅲ和Ⅳ级是诱导后低血压的独立危险因素[26]。但ASA分级同时受到术前基础疾病，年龄等因素的影响，故其相关性还需要进一步探究。

3.4 术前合并疾病

麻醉前低血压是PIH的危险因素[27,36]，许多因素可导致麻醉前低血压，如身体状况差、伴随疾病、肠道准备和禁食等。麻醉前低血压的治疗或许有益，有研究提示预先提高麻醉前血压基线值，如提高容积负荷或使用儿茶酚胺预处理，可减轻PIH的发生及程度[13,37]。术前合并的其它基础疾病可以增加发生PIH的可能性，其中术前合并高血压疾病可能具有最大的预后价值。在一些研究中，高血压疾病病人在麻醉诱导过程中经常出现低血压，是PIH的独立危险因素[32,38-39]，高血压疾病控制不良更容易导致术中低血压，这可能是因为高血压引起了血管内皮细胞的损伤，出现脂质成分的堆积等，长期以往会使得动脉血管硬化，血管弹性减退，血压调节能力降低[40]。

3.5 术前药物治疗

术前合并高血压且长期服用ACEI/ARB类药物（如卡托普利、厄贝沙坦等），可增加PIH的发生风险，主要原因是使用ACEI/ARBs可使动脉血管扩张从而降低血压[32,41-42]。慢性动脉高血压病人术前停用肾素-血管紧张素-醛固酮系统拮抗剂可明显降低PIH发生的风险[37]，可能部分归因于停药后增加诱导前血压值，进而降低PIH的风险。同时，研究表明，钙通道阻滞剂和β受体阻滞剂的使用也会增加PIH发生的风险[43]。

3.6 急诊手术

目前的研究结果表明，急诊手术是PIH的重要危险因素，行急诊腹腔镜胆囊切除术的病人发生PIH的风险较择期手术病人更高[4,9]。接受紧急手术的病人术

前准备时间比择期手术病人少，可能因其潜在疾病而发生循环功能障碍[44]。因此，在进行麻醉管理时，应格外警惕急诊病人发生诱导后低血压的可能，并做好对循环系统的保护措施。

3.7 全麻诱导药物

3.7.1 异丙酚　异丙酚是一种常见的麻醉诱导药物，由于其具有较强的血管舒张和负性肌力作用，会导致收缩压和舒张压呈剂量依赖性下降，有研究发现异丙酚诱导是PIH的危险因素[8]，该药物的负性肌力作用不会随着血浆异丙酚水平的降低而恢复，并持续10 min[45-46]。TARAO等的研究表明异丙酚诱导与已存在的心脏功能障碍之间存在较好的负协同作用[43]。为降低PIH的发生率，可以使用较少的异丙酚或者换用依托咪酯进行诱导，特别是对于身体状态虚弱的ASA Ⅲ或Ⅳ级病人[47-48]。除了剂量外，异丙酚注射速度对于PIH的发展也很关键，但相关研究比较少[43]。

3.7.2 芬太尼类　临床常用芬太尼和舒芬太尼进行麻醉诱导，它们都是μ阿片类激动剂。其中，舒芬太尼属于芬太尼的衍生物，它对μ1受体的亲和力和亲脂性均是芬太尼的几倍，能较好地透过血脑屏障，是镇痛药物中镇痛效应最强的阿片类药物，镇痛强度是芬太尼的5～10倍[34-43]。在临床上麻醉医生进行气管插管操作时，强烈的气管刺激可使病人自主神经系统兴奋（交感神经），进而出现血压骤增、心率加快等循环系统波动。而芬太尼和舒芬太尼在临床上常被用来减轻这种循环波动，它们均能抑制压力感受器的敏感性，降低交感神经的兴奋性，增加迷走神经的兴奋性[18]。既往研究表明，舒芬太尼与芬太尼相比，能更明显地减少喉镜暴露会厌和气管插管所引起的应激反应，使病人循环系统波动更小[4,34,49]，但这些研究人群大多数为ASA Ⅰ或Ⅱ级病人，并且也只研究了舒芬太尼对气管插管所致血压升高的抑制作用。而另一研究结果表明，与芬太尼相比，舒芬太尼是ASA Ⅲ-Ⅳ级病人发生PIH的独立危险因素[7-8]。在麻醉医生进行气管插管之前，病人没有受到任何手术刺激的情况下，舒芬太尼扩张血管平滑肌的作用使外周血管阻力下降，并且剂量越高的阿片类药物越容易引起血管舒张[4,8]，再加上舒芬太尼抑制了插管所致应激反应，更容易发生PIH[50]。因此，对于身体状态虚弱的ASA Ⅲ级或Ⅳ病人而言，临床上可通过减少阿片类药物的用量或选择其他麻醉诱导药物进行诱导来降低PIH发生的可能性。

3.7.3 咪达唑仑　作为一种常用的苯二氮卓类药物，咪达唑仑有抗焦虑、镇静、催眠及肌肉松弛等作用，静脉注射后会产生短暂的顺行性记忆缺失，使病人不能回忆起在药物高峰期间所发生的事情。咪达唑仑具有起效快而持续时间短的作用特点，通常用于全身麻醉诱导[51]。有研究表明，咪达唑仑不仅可以减少气管插管引起的应激反应，还可以减少术中知晓等不良反应的发生[46]。但它也有不足之处，咪达唑仑剂量依赖性的呼吸抑制作用以及外周血管扩张作用，可使动脉血压降低，研究表明，它的不良反应在与阿片类药物联合使用时更为明显[51-52]。一项临床研究指出，咪达唑仑和瑞芬太尼联合异丙酚诱导比单独异丙酚诱导更有可能降低老年病人全身麻醉诱导时的血压，增加PIH发生率[41,53]。因此，对于基础状况差、ASA分级高的病人，临床麻醉医生应尽量不选用咪达唑仑这一药物进行麻醉诱导，或者减少咪达唑仑诱导的剂量以预防PIH的发生，而在围术期实施麻醉的过程中，临床医生也应该密切关注病人的心率、血压等基础生命体征的变化，为病人保驾护航。

4 小结与启示

随着医疗技术的进步，进行手术治疗的病人越来越多，提高对PIH的发生机制与危险因素的充分认识、尽早采取预防和干预措施，将会减少PIH的发生并减轻PIH引起的严重不良预后，起到重大而积极的作用。比如，术前进行仔细的危险分层、治疗相关合并疾病、尽量减少使用循环抑制作用强的麻醉诱导药物等，都是有利于减少PIH的方法。作为临床麻醉医生应该思虑周全，力求平稳，减少可能的危险因素等，并依据病人情况选择个体化麻醉诱导方案，确保手术病人麻醉诱导的安全、平稳和舒适。

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《西南医科大学学报》对医学论文论著“讨论”编写的基本要求

“讨论”是医学论文正文不可缺的重要组成部分。本刊要求作者来稿医学论文论著的讨论应围绕论文的主题、要旨及中心内容,阐明研究的原理、目的、意义;在讨论中,应将研究结果放到国内外最新最前沿的相关研究背景下进行解析、比较,说明所研究结果的创新性、亮点及重要性与价值。讨论中切勿夸大研究结果的作用与价值,报喜不报忧;应同时指出结果的可能误差,存在的问题与教训,以及研究中有无例外或尚难解释的问题;提出新的研究方向或建议与展望,给读者以启迪。讨论用语应精练、层次清楚、逻辑性强,切忌繁杂、冗长,主次不分,甚至脱离主题,或重点不突出。讨论篇幅以1500～2000个汉字符为宜。

(本刊编辑部)