

危重患者中无创颅内压监测技术及体位相关性进展

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摘 要

颅内压(ICP)监测对于反应颅脑病情变化极其重要。目前的金标准仍是侵入性的颅内压测量,但由于有创操作容易出现各种并发症,并且有创监测受到患者病情,环境及体位等相关因素而无法进行普遍的应用。故此非侵入性的ICP监测手段应运而生,受到大家的关注。目前在临床中应用的非侵入性ICP测量方法主要包括通过影像、眼科、超声、电生理及其他方法来侧面反映颅内压。本文旨在对当前临床上应用相对广泛的几种非侵入性检测ICP方法,介绍其原理、适应症及部分检测手段在体位改变时所产生的相应变化。虽然这些方法并不能完全替代侵入性监测方法,但在某些特定患者及环境中,可以发挥其特殊作用。

关键词

颅内压, 无创监测

Progress in Non-Invasive Intracranial Pressure Monitoring Technology and Positional Correlation in Critically Ill Patients

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Abstract

Intracranial pressure (ICP) monitoring is extremely important for reflecting changes in the condition of the brain. The current gold standard is still invasive intracranial pressure measurement, but due to the susceptibility of invasive procedures to various complications and the limitations of invasive monitoring due to factors such as patient condition, environment, and position, it cannot be widely applied. Therefore, non-invasive ICP monitoring methods have emerged and received widespread attention. The non-invasive ICP measurement methods currently used in clinical practice mainly include imaging, ophthalmology, ultrasound, electrophysiology, and other methods to indirectly reflect intracranial pressure. This article aims to introduce the principles, indications, and corresponding changes of some non-invasive detection methods for ICP that are currently widely used in clinical practice when there is a change in posture. Although these methods cannot completely replace invasive monitoring methods, they can play a special role in certain specific patients and environments.

Keywords

Intracranial Pressure, Non Invasive Monitoring

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1. 介绍

颅内压(ICP)是指颅腔内容物对颅腔壁所产生的压力,又称为脑压。由于脑脊液存在于蛛网膜下腔中,介于颅腔壁和脑组织之间,并与脑室和脊髓腔内蛛网膜下腔相通,所以脑脊液的静水压可代表颅内压,通常以侧卧位时行腰穿所测得的脊髓内蛛网膜下腔内压力作为脑脊液压力的代表。颅内压升高(ICP)是指在病理状态下颅内压超过 200 mm H₂O [1]。常见于颅腔内容物的体积增加并超出颅内压调节、代偿的范围,从而引起头痛、呕吐、视乳头水肿,甚至出现意识障碍及生命体征变化等临床表现。颅高压是疾病病情变化所导致的临床综合征[2],其变化成为评估患者病情严重程度的标准之一[3]。于 1960 年,伦德伯格首次实现了颅内压的有创持续监测,之后不断改进。目前其不仅在神经内外科具有重要意义,在眼科和航空航天医学等其他学科中也有广泛的应用[4]。

研究 ICP 测定常选择腰椎穿刺[5],但需要进行持续监测时,脑室外引流(EVD)则应用较多[6]。而有创操作存在许多并发症,如颅内感染、出血、导管脱落等[7]。不同的研究报告中,患者发生颅内感染风险的概率差别较大(1%~27%) [8]。而在危重症患者中,颅内感染风险更是大大提高。侵入性的颅内压监测方法受到了一定的限制。

随着医疗技术不断进步,非侵入性 ICP 测量方法不断发展,与侵入性 ICP 测量相比,非侵入性 ICP 测量更加安全与便捷,可以减少感染,出血等相关并发症[9]。具有相对安全、经济、低成本并且可以持续性监测的方法,不仅使得疾病得以早期发现、诊断和治疗[10]。而且有助于关注到危重症患者或手术患者因为治疗、观察、手术或是体位等因素,在体位改变时从而引发的 ICP 变化。本文主要旨在回顾不同临床场景中无创 ICP 测量与持续监测,明确其适应症以及主要优缺点,并总结体位改变是否对其产生相关影响。目前应用较多的非侵入性 ICP 测量方法主要有视神经鞘测量、眼压测定、经颅多普勒超声、闪

光诱发视觉点位和脑电图等。

2. 非侵入性测量方法

2.1. 视神经鞘

脑膜由内向外依次为软脑膜、蛛网膜、硬脑膜,视神经鞘(ONS)是硬脑膜的延续。硬脑膜在视神经管位置时分为内外两层,内层包绕视神经而形成ONS[11]。视神经与视神经鞘之间的间隙与视网膜下腔相通,脑脊液在其间流动。当急性或慢性疾病导致ICP的增加时(例如特发性颅内高压,颅脑创伤,恶性大脑中动脉搏动,颅内出血,失代偿性脑积水等),由于视神经鞘与视神经之间的间隙与颅内空间相通,随着ICP的增加,视神经鞘的直径会受到影响,出现ONSD的改变[12]。通过CT或MRI成像可以精确、多层次测量ONS,从而反映患者ICP变化,但患者需转运至放射科进行操作,危重症患者在复杂的转运以及路途中容易出现病情变化。而床旁超声测量ONSD与使用CT或MRI测量的结果具有较高一致性,因此床旁超声测量ONSD以价廉、便捷、无辐射、安全等独特优点日益成为监测ICP变化的良好辅助诊断工具[13]。在Tural Bayramov的研究中,视神经鞘的变化与体位也存在关系,当患者从坐姿变为头低脚高位时,视神经鞘宽度在20分钟内可明显增加。动态监测患者视神经鞘宽度时,应确保患者体位的统一性[14][15]。动态监测视神经鞘宽度也可以帮助提高某些被动体位患者(如俯卧位、特殊体位手术等)治疗的安全性[16]。同样视神经鞘的宽度也受药物影响,应用丙泊酚将明显影响视神经鞘的变化[17]。同时二氧化碳水平的升高亦会影响着视神经鞘宽度[18]。但结果并不完全统一,在Zoerle的研究中,10名蛛网膜下腔出血的患者在颅内压升高时并未观察到明显的视神经鞘变化[19]。同时并且由于患者眼部或全身性的相关疾病,如Graves、炎症、结节病或是眼部的出血,肿瘤压迫等情况时[20],也会出现视神经鞘的增宽,影响测定以及结果的解读。

根据观察性研究,健康人群的ONSD正常范围约为2.2~5毫米。在创伤性颅脑损伤、蛛网膜下腔出血、颅内出血等多种神经系统疾病中,ONSD增宽与ICP升高相关[21]。迄今最大规模的实验是由Rajajee等人进行的。他们在65名患者中,总共进行了536次ONSD测量。当视神经鞘>4.8毫米时颅内压>20毫米汞柱,灵敏度为96%,特异度为94%[22]。而动态的监测视神经鞘宽度的变化,可以从侧面及时反映患者颅内压变化情况,为我们及时发现患者颅内病情变化提供了帮助[23]。

2.2. 眼压

眼压又称眼内压,是眼球内容物对眼球壁所产生的压力。正常人的眼压稳定在10 mmHg~21 mmHg(1.33 kPa~2.80 kPa)间,眼球的正常形态需要稳定的压力维持,使各屈光介质界面保持良好的屈光状态。而在ICP升高时,压力会通过影响视网膜和视网膜中央静脉的循环从而导致静脉回流障碍,从而引起眼压(IOP)的变化。通过眼压测量来估测ICP具有一定程度的可靠性和准确性[24]。IOP受到许多因素的影响,在青光眼等具有眼部疾患的患者中可能会出现异常的IOP。目前临床上,很难确定异常的IOP是因颅压变化还是眼部疾病引起的。同时由于个体化差异和各种IOP测量技术之间的差别,目前暂无具体的IOP数值来衡量ICP[25]。一项130名患者的观察性研究中,我们发现ICP与IOP($r = 0.32, p < 0.001$)之间存在明显的相关性,大约有三分之二的患者(65.4%) IOP可以明确反映ICP水平变化[26]。另一项24名患者的研究中,手持眼压计所测得的眼内压与颅内压未见明显相关,因此建议不要使用手持眼压计来评估颅内压的变化[27]。

当患者由坐位改为仰卧位时,正常人和青光眼患者的眼压均会升高,且青光眼患者眼压升高的幅度更大。不论是正常人还是青光眼患者,侧卧位时位于下方的眼压均较上方的眼压高,在有视野损害的患者中,损伤越严重,其眼压升高幅度越大。坐位改为站位时眼压也会升高,但随时间推移,眼压逐渐降

低。同样在肥胖患者中表现更加明显,即便是仰卧位时,眼压增加都大于非肥胖人群[28]。在一项研究中,我们发现当 BMI 每增加 10 个单位,坐姿位 IOP 增加 0.55 ± 0.23 毫米汞柱($P = 0.0184$),仰卧位 IOP 增加 0.49 ± 0.24 毫米汞柱($P = 0.0409$) [29]。然而 Muchnok 的观察性研究显示,通过 IOP 升高来预测 ICP 升高的敏感度并不高(敏感度 24%, 特异度 63%) [25]。可能是因为 IOP 和 ICP 是两个相互关联、相对独立的压力系统,他们分别通过房水循环、脑脊液循环以及相应的神经调节机制来保持相对稳定的压力水平。所以轻度的颅内压变化并不能导致明显的眼压变化,只有在颅内压的急剧上升 $> 50 \text{ cmH}_2\text{O}$ 时才能导致眼内压显著升高[30]。因此,基于 IOP 测量简单、快捷,在颅内压急剧变化时,不失为危重患者颅脑疾病的筛查与动态评估的一种补充评价。

2.3. 经颅多普勒超声

经颅多普勒超声(TCD)是一种测量和评估颅底动脉血流动力学参数的无创监测方法,可以用于检查各种脑血管疾病。当 ICP 增加时,脑动脉平均血流速度、收缩期血流速度和舒张流速下降,脉动指数(PI)和阻力指数(RI)显著增加[31]。目前主要通过测量大脑中动脉的 PI 来反映颅内压。研究显示当 $PI \geq 1.26$ 时,预估 $ICP \geq 20 \text{ cmH}_2\text{O}$ 。灵敏度为 81.1%, 特异度为 96.3% [32]。但大脑中动脉流速易受包括全身动脉血压、血管顺应性、姿势、心律和心输出量等影响。单根血管的流速改变不足以反映 ICP 的变化[33]。

然而有研究显示,眼动脉 OA 的颅内段受脑室内压力,即 ICP 的影响,而颅外段受外部施加压力的影响。我们可以通过测量眼动脉的颅内和颅外段的流速,以相应步骤向眼球周围的组织施加外部压力。当测量到颅内段与颅外段眼动脉的血流流速近似时,则认为施加的压力等于颅内压[34]。Lucinskas 研究测定出当眼动脉颅内与颅外段流速相等时,施加的压力低于 ICP 约 6 mmHg,差值产生的原因可能与眼眶内压力等相关。通过数字建模及线性回归分析,两者存在明显的正相关($r = 0.94$) [35]。当然,目前 Lucinskas 研究中,仅在不校准的情况下监测了 1 小时。故其局限性可能在于无法进行连续、长程测量,且并不能显示出绝对的 ICP 值,只反映 ICP 动态的变化。对于患有某些神经疾病(例如严重颅脑创伤或中风)的患者,对眼睛施加压力的操作确实需要仔细斟酌。同时在体位变化时,眼动脉流速也会受到一定影响,在坐位改变为仰卧位时,眼动脉血流速度明显增加,RI 显著下降[36]。此项技术需要专门设备,测量血流速度时会受到探头与血管位置存在夹角影响,结果易出现偏差。同时老年患者(尤其女性)中,由于颅骨增厚、动脉迂曲、动脉移位等,多普勒超声测量的失败率会明显上升。故该项监测手段对操作人员及设备使用均需一定要求。

2.4. 脑氧监测

脑氧监测主要基于近红外光谱(NIRS)的原理。在 1977 年首次被提出,称为脑氧饱和度监测(rScO₂) [37]。NIRS 是一种非侵入性装置,是通过利用红外线,检测特定波长的近红外光,从而反映脑组织中氧合血红蛋白和脱氧血红蛋白比例,最后计算出局部脑氧饱和度[38]。脑组织中存在大量的微细血管,其中动脉血约占 20%,毛细血管血约占 5%,其余约 75%皆为静脉血。因此, rSO₂ 其实是局部脑组织中动、静脉血氧饱和度的加权平均值,更接近于静脉血氧饱和度,可以反映脑氧供给与消耗的动态平衡。在颅内压轻、中度增高时,脑氧代谢指标无显著变化,但对于重度颅内压升高者,脑氧可发生显著变化[39]。一项临床实验分析了 65 例重型颅脑外伤患者的颅内压与脑氧之间的关系,发现 ICP 与 rSO₂ 之间存在显著负相关[40]。使得 NIRS 成为无创颅内压监测方法具有一定可能。

脑氧同时也受体位变化的影响,在手术室中,患者由于手术体位要求,当患者由平卧位改为沙滩椅体位后,可以发现患者 rSO₂ 下降幅度约 20%。而在患者处于俯卧位时,由于非生理性的体位影响,胸廓活动受限、脊柱压迫、腹式呼吸受限等情况会影响呼吸和循环等系统[41]。俯卧位时局部受压,可使回心

血量进一步减少, 导致脑低灌注, 甚至脑缺氧, 老年患者表现得更明显。这些因素均可引起脑灌注改变, 有研究显示, 俯卧位手术脑缺氧的发生率是仰卧位手术的两倍。在患者存在俯卧位体位改变时, 双侧脑氧饱和度出现先下降后恢复到平卧位水平。当患者由仰卧位改为俯卧位后, 30 min 时, rSO₂ 可从 76.24% 下降至 73.18% [42]。另外, 在侧卧位时两侧大脑半球脑氧饱和度有所差距, 平均来看, 其上半球 rSO₂ 较下半球高 1.3%, 此时 rSO₂ 的值是两侧脑氧饱和度的平均值。所以不同体位对脑氧影响差距大, 在测量时同时还需关注体位情况[43]。

2.5. 闪光视觉诱发电位

闪光视觉诱发电位(FVEP)是指视网膜受到均匀闪光刺激后, 枕叶视皮层的变化。完整的视觉通路是从视网膜神经节到枕皮层[44]。ICP 升高引起的脑干和部分血管受压, 导致神经元和神经纤维缺血、缺氧, 随后的脑组织代谢受损, 神经元电信号传导受阻。在颅内压升高时, FVEP 波峰潜伏期延长, 波幅减小, 波宽增加。在严重颅脑创伤患者中, 如果出现脑疝, 这种现象会更明显[44]。因此, 可以通过 FVEP 波峰的变化和 ICP 之间的线性关系来间接测量 ICP。早在 1981 年, York 等人就证明了 VEP 的 N2 波延迟与 ICP 升高之间存在很强的正相关[44], 同时在 ICP > 30 cmH₂O 时, VEP 的预测与准确性明显提高[45]。

根据 FVEP 原理研发的 ICP 监测装置已在临床上使用。目前主要应用在颅脑外伤、脑积水、蛛网膜下腔出血和高血压性脑出血等颅压升高的患者[46]。FVEP 评估对患者有特定要求, 在癫痫、视网膜损伤或视神经压迫等疾患的患者中, 发挥的作用可能较为有限。甚至在部分有关研究中, FVEP 未显示其作用[47]。目前尚未明确 VEP 的变化与体位是否存在明显关系。

2.6. 神经性瞳孔指数

神经性瞳孔指数(NPi)是通过测量不同的瞳孔变量(即瞳孔大小、收缩百分比、收缩潜伏期、收缩速度、扩张速度)。基于这些变量的组合, 可以计算 NPi。NPi 算法量化瞳孔反应性, 删除了主观性的相关判断, NPi 的正常值范围为 0~5, >3 表示瞳孔对光反射正常, <3 则表示异常, 0 则代表无收缩, 无瞳孔对光反射[48]。中枢神经系统损伤加重恶化时, NPi 和瞳孔光反射可能会较早出现改变。在相关研究中, 瞳孔反应性正常的患者中, 平均颅内压为 19.6 mmHg, 而在瞳孔反应异常的患者中, 平均颅内压为 30.5 mmHg [49]。在严重的颅压升高之前的 15.9 小时便可检测到瞳孔异常, 提示 NPI 可提供颅内高压的早期预警。当排除药物及其他相关影响(如阿片类药物、部分降压药、有机磷农药、Horner 综合征或眼部炎症等), NPI 值 < 3 和颅内压升高之间具有明显的关系[50]。但在部分研究中, ICP 的升高和 NPi 值未见明显相关性[51]。目前尚无体位变化与瞳孔之间的相关性。

2.7. 视网膜血管

颅内压升高时, 压力通过视神经蛛网膜传递到视乳头, 从而导致视乳头水肿。而视乳头水肿加重视网膜中央静脉压增高, 影响静脉回流, 故 ICP 升高可引起视网膜静脉扩张[52]。在 80%至 90%的受试者中, 我们可以观察到视网膜自发性静脉搏动, 自发性视网膜静脉搏动指位于视盘边缘附近的视网膜静脉管径的振动, 是由眼压和 ICP 的波动引起的跨壁压力振动所产生[53]。当视网膜静脉压升高时, 跨壁压力振动的幅度降低, 脉动不明显。所以 ICP 增高时自发性视网膜静脉搏动消失[54]。动物模型实验中证实急性的颅内压升高与自发性视网膜静脉搏动消失同步发生, 故通过监测有无自发性视网膜静脉搏动存在来评估 ICP 增高也可作为一种临床工具, 但其只能进行定性分析, 尚不能定量反映颅内压, 同时由于视乳头水肿, 也限制了我们视网膜静脉的观察, 影响我们对视网膜静脉参数的准确评估。视网膜静脉搏动在姿势变化中可出现明显的振幅差异, 在 7 名受试者的研究中发现, 坐姿时患者的视网膜静脉搏动

振幅差达到最大值[53]。在 Georgevsky D 的研究中观察到 SVP 振幅从坐姿到仰卧位下降了 22%，而从坐姿到侧卧位时变化率为-18%，而在仰卧变为侧卧时，变化是微小的(4%) [55]。

2.8. 鼓膜位移

鼓膜位移是第一个通过听力学无创监测颅内压的方法[51]，在正常声音传导时，鼓膜振动通过中耳的听小骨传递到内耳，而镫肌和张鼓膜肌的收缩使鼓膜从静息位置进行小的位移。而在颅内压升高时，由于脑脊液和外淋巴液通过耳蜗导水管连通，颅内压(ICP)的增加直接传递到镫骨底，使鼓膜从其初始位置移位，影响鼓膜位移的方向与位移大小[56]。向内位移表示 ICP 增高，向外位移表示 ICP 正常或减低。Samuel 等人通过研究 8 名脑积水分流术后的患儿的颅内压与鼓膜位移的变化，鼓膜位移测试的灵敏度为 83%，特异性为 100% [57]。鼓膜位移无法获得精准的颅内压数值，同时在患者的测量过程中，鼓膜位移变异度很高，需要具有完整且正常的听觉系统，较难实现精确的测量。而鼓膜位移在坐姿改变为仰卧位时出现明显变化，这种变化在年轻患者中变化幅度较老年患者更加明显[58] [59]。

3. 结论

虽然目前无创颅内压监测有许多间接测量的手段，新技术也在不断进步与发展，但不可否认，各种测量方法各有其优缺点。部分监测方法只能定性，而部分能定量检测的方法只能侧面反映出颅内压变化趋势。并且这些测量方法受到设备、操作者、患者及环境等相关因素影响，同时其结果常常因为患者的体位变化而干扰结果的判读，从而产生误差。目前仍没有一种无创测量方法可以拥有足够的准确性和可靠性来完全取代有创颅内压监测。但在重症监护室、危重患者中，由于患者的特殊性及时场的局限性，有创监测常常受到很大程度的限制，无创监测的方法可以帮助我们更快捷，更方便进行颅内压升高患者的识别。这些无创方法在有创颅内压监测存在禁忌或无法进行时可作为有价值的替代方法。同时通过联合多种不同颅内压监测方法，或许对我们进行颅内压力的综合评价更具有指导意义。

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