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恶性高热和肌肉萎缩症

Malignant Hyperthermia and Muscular Dystrophies

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Anesth Analg 2009 109: 1043-1048.

背景：据报道患有肌肉萎缩症（肌营养不良）的病人在全身麻醉时和麻醉后可能发生很多致命的并发症。作者对患有肌肉萎缩症的病人做了一项系统分析，旨在定义此类病人麻醉相关并发症的范畴，重点强调了恶性高热的易感性。
方法：作者使用了多个搜索引擎进行文献检索并对合适的文献进行评价从而确定患肌肉萎缩症病人麻醉相关并发症。在所有肌肉萎缩症的类型中，Duchenne 型肌营养不良（DMD）和 Becker 型肌营养不良（BD）占据了几乎所有麻醉相关的报道。
结果：DMD 和 BD 病人麻醉相关并发症包括术中心力衰竭、吸入麻醉相关的横纹肌溶解症（不用琥珀酰胆碱）和琥珀酰胆碱引起的横纹肌溶解症及高钾血症。
结论：与普通人群相比，并没有发现 DMD 和 BD 病人增加了恶性高热的易感性。但是，暴露于吸入麻醉药的营养不良病人可能引起疾病相关的心脏并发症，或罕见的以横纹肌溶解为特征的恶性高热相似症状。后者也可能发生在术后。琥珀酰胆碱可以引起致命性高钾血症，应避免用于 DMD 和 BD 患者。
（李潺 译 陈杰 校）

BACKGROUND: Patients with muscular dystrophy have been reported to experience a variety of life-threatening complications during and after general anesthesia. We performed a systematic analysis to define the spectrum of anesthetic-related complications in patients with muscular dystrophy, with an emphasis on malignant hyperthermia susceptibility.
METHODS: A literature search was undertaken using multiple search engines and the appropriate articles were reviewed by the authors to determine anesthetic-associated complications in patients with muscular dystrophy. Of all the types of muscular dystrophy,

Duchenne muscular dystrophy (DMD) and Becker dystrophy (BD) represent nearly all the anesthesia-related reports.
RESULTS: Anesthetic complications in patients with DMD and BD include intraoperative heart failure, inhaled anesthetic-related rhabdomyolysis (absence of succinylcholine), and succinylcholine-induced rhabdomyolysis and hyperkalemia.
CONCLUSION: We did not find an increased risk of malignant hyperthermia susceptibility in patients with DMD or BD compared with the general population. However, dystrophic patients who are exposed to inhaled anesthetics may develop disease-related cardiac complications, or rarely, a malignant hyperthermia-like syndrome characterized by rhabdomyolysis. This latter complication may also occur postoperatively. Succinylcholine administration is associated with life-threatening hyperkalemia and should be avoided in patients with DMD and BD.

劳累性热病、运动性横纹肌溶解症及恶性高热之间的关系

The Relationship Between Exertional Heat Illness, Exertional Rhabdomyolysis, and Malignant Hyperthermia

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Anesth Analg 2009 109: 1065-1069.

劳累性热病、运动性横纹肌溶解症和恶性高热（MH）具有相似病理生理学的综合征。三者均有高代谢特征，包括：三磷酸腺苷的高需求、氧化和代谢的加速、肌肉的机械应力，以及不受控制的细胞内钙的增加。尽管没有临床的对照研究来支持其中的关系，但有证据表明预期外的热/运动的不耐受和 MH 易感性相关。有多个病理报告和小样本的临床研究已经用于体外肌肉挛缩实验和/或基因监测来证实这种关系。然而，这种方法存在问题，因为这些研究在与麻醉有关系的临床 MH 中证实有效，而不是与劳累性热病或恶性高热有关。然而，这些相互关系对某些 MH 易感的患者以及他们运动的能力可能有意义，同时对临床医生的治疗和伴随有原因不明劳累性热病和运动疾的患者的麻醉具有重要意义。
（怀晓蓉 译 陈杰 校）

Exertional heat illness, exertional rhabdomyolysis, and malignant hyperthermia (MH) are complex syndromes with similar pathophysiology. All three are hypermetabolic states that include high demand for adenosine triphosphate, accelerated oxidative, chemical, and mechanical stress of muscle, and uncontrolled increase in intracellular calcium. Although there are no controlled clinical studies to support a relationship, there is evidence to suggest an association between unexpected heat/exercise intolerance and MH susceptibility. There are multiple case reports and a small number of clinical studies that have used *in vitro* muscle contracture testing and/or genetic testing to make the association. However, such methodology is problematic in that these tests are validated for clinical MH in association with anesthesia, and not for exertional heat illness or exertional rhabdomyolysis. Nevertheless, these relationships may have implications for some MH-susceptible patients and their capacity to exercise, as well as for clinicians treating and anesthetizing patients with histories of unexplained exertional heat and exercise illnesses.

N_2O 的镇痛作用不影响其制动需求

Nitrous Oxide-Induced Analgesia Does Not Influence Nitrous Oxide's Immobilizing Requirements

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Anesth Analg 2009 109: 1111-1116.

背景：氧化亚氮（N_2O）作用于脊髓上去甲肾上腺素神经元产生镇痛，但尚不清楚这一作用是否与氧化亚氮的制动作用有关。本研究作者拟检验如下假设：在脊髓上去甲肾上腺素神经元选择性清除或初次接受 N_2O 且镇痛缺乏的动物（naïve animals ）N_2O 最低肺泡麻醉浓度（MAC）不变。
方法：研究者测定了 70% N_2O 、一个 MACN_2O 或一个 MAC 异氟醚下在脑室内注射抗多巴胺-β 羟化酶共轭皂草素(SAP-DBH; n = 7)或对照抗体共轭皂草素(n = 5)前或后甩尾潜伏期(TFL)和后爪退缩潜伏期(HPL) 。naïve animals 组(n = 8) N_2O MAC 在吸入 N_2O25–45 min 后测定（镇痛作用高峰期），并在 120–140 min （TFL 和 HPL 恢复到基础值后）后再测定
结果：吸入 N_2O30min 后，TEL 和 HPL 值显著升高，但在 120min 内回到基础值。接受 SAP-DBH 大鼠 N_2O 镇痛未达到效果。然而，N_2O 和异氟醚的 MAC 在 SAP-DBH 组和对照组间无显着差异（N_2O 平均值±标准差：1.7 ± 0.1 vs 1.7 ± 0.2; 异氟醚：1.6 ± 0.2%vs 1.7 ± 0.2%）。naïve 动物的 N_2O 吸入 30min 与 120min 时 MAC 没有差别（1.8 ± 0.1 vs 1.8 ± 0.2）。
结论：去除脑干去甲肾上腺素能神经元或长期接触 N_2O 将减弱其镇痛效应，但不改变 MAC。N_2O 的制动作用的机制不依赖其镇痛作用。
（陈毓雯 译 陈杰 校）

BACKGROUND: Nitrous oxide (N_2O) acts on supraspinal noradrenergic neurons to produce analgesia, but it is unclear if analgesia contributes to N_2O's immobilizing effects. We tested the hypothesis that N_2O minimum alveolar anesthetic concentration (MAC) is unchanged after selective ablation of supraspinal noradrenergic neurons, or in naïve animals at N_2O exposure timepoints when analgesia is absent.
METHODS: We determined tailflick latency (TFL) and hindpaw withdrawal latency (HPL) under 70% N_2O, N_2O MAC, and isoflurane MAC before and after intracerebroventricular injections of anti-dopamine-β hydroxylase conjugated to saporin (SAP-DBH; n = 7), or a control antibody conjugated to saporin (n = 5). In a separate group of naive rats (n = 8), N_2O MAC was determined at 25–45 min after initiation of N_2O exposure (during peak analgesia) and again at 120–140 min (after TFL and HPL returned to baseline).
RESULTS: After 30 min of N_2O exposure,（TFL and HPL increased significantly but declined back to baseline within 120 min. N_2O did not produce analgesia in rats that received SAP-DBH. However, N_2O and isoflurane MAC were not significantly different between SAP-DBH and control-injected animals (Mean ± sd for N_2O: 1.7 ± 0.1 atm vs 1.7 ± 0.2 atm; isofurane: 1.6 ± 0.2% vs 1.7 ± 0.2%). In naïve animals, N_2O MAC was not

different at the 30 min period compared with the 120 min period (1.8 ± 0.1 atm vs 1.8 ± 0.2 atm).
CONCLUSIONS: Destroying brainstem noradrenergic neurons or prolonged exposure to N_2O removes its analgesic effects, but does not change MAC. The immobilizing mechanism of N_2O is independent from its analgesic effects.

头戴式显示器监测：全方位模拟器和部分任务训练器的操作和安全性

Monitoring with Head-Mounted Displays: Performance and Safety in a Full-Scale Simulator and Part-Task Trainer

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背景：头戴式显示器可以帮助麻醉医师在任何时候都可以看到术中病人所监测的生命体征，即使麻醉医师忙于操作或者无法看到监视器的时候。相关的麻醉文献显示使用 HMD（头戴式显示器）有其优点，但也有研究表明 HMD 会加重注意迟钝（使用者很有可能因专注于显示器而忽略眼前的其他事物），同时可能产生于焦距深度有关的知觉问题。本研究调查了两种模拟器的差异。
方法：实验一，研究戴上 HMD 是否会影响麻醉医师发现病情的速度，以及 HMD 设置的不同焦距（近或远）其结果是否会有差别。12 名麻醉医师在三个自然场景中进行麻醉，这三个场景中均用模拟器模拟手术环境。在病人的监视器上或者手术室内出现 24 种不同的病情变化。实验二，研究麻醉医师由于进行操作而受到身体上的限制，使用 HMD 的麻醉医师是否会比没有使用 HMD 的麻醉医师更快发现病情变化。12 名麻醉医师在监测模拟病人生命体征的同时，要在部分任务训练器上进行复杂的模拟临床任务。所有的参与者在两种场景中共会碰到 4 种不同的情况。
结果：实验一表明带上 HMD 或者调整设置的焦距都不会降低参与者发现病情变化的能力（包括发现病情情况的数量和时间）。总的来说，使用 HMD 后的参与者较只用标准监视器的时候会花更多的时间去看病人，更少的时间去看麻醉机、显示器。参与者报告他们更喜欢近焦距的设置。实验二显示参与者使用 HMD 后发现其中 2 种病情变化的速度会更快，而另一种病情变化发现较慢。参与者使用 HMD 后去看麻醉机显示器的频率大大下降。使用 HMD 后，参与者表示他们没这么忙了，监测也更容易，而且他们相信发现异常情况变化的速度会更快。
结论：HMD 可以帮助麻醉医师在身体受限的时候也能发现病情变化，而不是在身体不受限的时候。尽管没有足够的证据说明会加重不注意，但在航空方面发现，与 HMD 有关的知觉问题会影响到测试者是否能发现到情况的变化。麻醉医师使用

HMD 后应该自我调整焦距，从而使眼睛的限制降到最低，同时也应该注意到一点，就是某些变化可能无法引起他们的注意。对于建立设计 HMD 的原则，评估其他型号的 HMD，及临床应用方面还需进一步研究。
（张婷 译 陈杰 校）

BACKGROUND: Head-mounted displays (HMDs) can help anesthesiologists with intraoperative monitoring by keeping patients' vital signs within view at all times, even while the anesthesiologist is busy performing procedures or unable to see the monitor. The anesthesia literature suggests that there are advantages of HMD use, but research into head-up displays in the cockpit suggests that HMDs may exacerbate inattentional blindness (a tendency for users to miss unexpected but salient events in the field of view) and may introduce perceptual issues relating to focal depth. We investigated these issues in two simulator-based experiments.
METHODS: Experiment 1 investigated whether wearing a HMD would affect how quickly anesthesiologists detect events, and whether the focus setting of the HMD (near or far) makes any difference. Twelve anesthesiologists provided anesthesia in three naturalistic scenarios within a simulated operating theater environment. There were 24 different events that occurred either on the patient monitor or in the operating room. Experiment 2 investigated whether anesthesiologists physically constrained by performing a procedure would detect patient-related events faster with a HMD than without. Twelve anesthesiologists performed a complex simulated clinical task on a part-task endoscopic dexterity trainer while monitoring the simulated patient's vital signs. All participants experienced four different events within each of two scenarios.
RESULTS: Experiment 1 showed that neither wearing the HMD nor adjusting the focus setting reduced participants' ability to detect events (the number of events detected and time to detect events). In general, participants spent more time looking toward the patient and less time toward the anesthesia machine when they wore the HMD than when they used standard monitoring alone. Participants reported that they preferred the near focus setting. Experiment 2 showed that participants detected two of four events faster with the HMD, but one event more slowly with the HMD. Participants turned to look toward the anesthesia machine significantly less often when using the HMD. When using the HMD, participants reported that they were less busy, monitoring was easier, and they believed they were faster at detecting abnormal changes.
CONCLUSIONS: The HMD helped anesthesiologists detect events when physically constrained, but not when physically unconstrained. Although there was no conclusive evidence of worsened inattentional blindness, found in aviation, the perceptual properties of the HMD display appear to influence whether events are detected. Anesthesiologists wearing HMDs should self-adjust the focus to minimize eyestrain and should be aware that some changes may not attract their attention. Future areas of research include developing principles for the design of HMDs, evaluating other types of HMDs, and evaluating the HMD in clinical contexts.

2001–2005 纽约州麻醉引起的恶性高热的流行率

Prevalence of Malignant Hyperthermia Due to Anesthesia in New York State, 2001–2005

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背景：恶性高热是一种极易被诱因触发的药物遗传学综合症。在美国恶性高热流行病学尚未很好的建立。本研究作者对纽约医院的恶性高热流行性进行评估。
方法：通过纽约 2001 年至 2005 年间的出院病人数据，使用第九版疾病的国际分类法，修改代码 995.86，来识别由麻醉导致恶性高热的病人。通过人口统计学和临床症状来评估恶性高热的流行性。
结果：在这段研究期间纽约医院的 12749125 位出院病人中，有 73 例发生麻醉引起的恶性高热。近 3/4 患者为男性、71%的病人为急诊入院患者。手术出院病人的恶性高热患病率为 0.96/100000（可信区间 95%，误差 0.67-1.24），接受麻醉的出院病人中恶性高热患病率为 1.08/100000（可信区间 95%，误差 0.75-1.41）。这项恶性高热流行性的评估中男性是女性的 2.5~4.5 倍。
结论：在纽约州医院手术后的病人中，麻醉引起的恶性高热流行率约 1/100000。男性患恶性高热的风险显著大于女性。
（杨秋娟 译 陈杰 校）
BACKGROUND: Malignant hyperthermia (MH) is a pharmacogenetic syndrome that variably expresses itself on exposure to triggering agents. MH prevalence in the United States is not well documented. In this study, we assessed the prevalence of MH in New York State hospitals.
METHODS: Using New York hospital discharge data for the years 2001 through 2005, we identified all patients with a diagnosis of MH due to anesthesia using International Classification of Diseases, Ninth Revision, Clinical Modification code 995.86. MH prevalence was evaluated by demographic and clinical characteristics.
RESULTS: Of the 12,749,125 discharges from New York hospitals during the study period, 73 patients had a recorded diagnosis of MH due to anesthesia. Nearly three quarters of the MH patients were male and 71% were patients from emergency/urgent admissions. The estimated prevalence rate of MH was 0.96 (95% confidence interval [CI] 0.67–1.24) per 100,000 surgical discharges and 1.08 (95% CI 0.75–1.41) per 100,000 discharges in which there was any indication of exposure to anesthesia. The estimated prevalence of MH for males was 2.5 to 4.5 times the rate for females.
CONCLUSION: The prevalence of MH due to anesthesia in surgical patients treated in New York State hospitals is approximately 1 per 100,000. MH risk in males is significantly higher than in females.

在病态肥胖的病人中阻塞性睡眠呼吸暂停不是困难插管的危险因素

Obstructive Sleep Apnea Is Not a Risk Factor for Difficult Intubation in Morbidly Obese Patients

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背景：我们普遍认为病态肥胖症(MO)，阻塞性呼吸暂停(OSA)和颈围是气管插管的独立危险因素。本研究中，作者试图确定这些因素对于在经历减肥手术的病人是否会增加困难插管风险。通过测定呼吸暂停低通气指数(AHI)、性别、颈围和体重指数(BMI)的因素来确定阻塞性呼吸暂停(OSA)和它的严重程度。
方法：所有已登记的病态肥胖症病人术前描记多导睡眠图。阻塞性呼吸暂停的严重程度是通过呼吸暂停低通气指数和美国麻醉医师协会的阻塞性呼吸暂停严重等级来确定。全部病人用统一标准的麻醉药及方法，包括使用倾斜位直接喉镜法。
结果：有 180 位连续病例入选，140 位女性，40 位男性。阻塞性呼吸暂停的发生率为 68%，平均体重指数是 49.4 kg/m^2，平均呼吸暂停低通气指数是 31.3 (范围, 0-135)。所有病人的气管插管均未用急救气道并由麻醉住院医生完成。6 例病人尝试了 3 次及 3 次以上插管才成功。困难插管的发生率为 3.3%，困难喉镜暴露（Cormack and Lehane 3 级或 4 级）的发生率为 8.3%。颈围和困难气管插管无关(优势比 1.02, 95% 可信区间 0.93-1.1)，阻塞性呼吸暂停和困难插管之间无显著相关(*P* = 0.09)，体重指数和困难插管之间也无显著相关(优势比 0.99, 95%可信区间 0.92-1.06, *P* = 0.8)。插管的尝试次数和体重指数 BMI (*P* = 0.8), 呼吸暂停低通气指数 AHI (*P* = 0.82) 及颈围 NC (*P* = 0.3)之间没有关系。Mallampati 评级 III 或者更高预示着困难插管(*P* = 0.02)，男性也是如此(*P* = 0.02)。最后，Cormack and Lehane 分级与体重指数 BMI (*P* = 0.88)；呼吸暂停低通气指数 AHI (*P* = 0.93)；阻塞性呼吸暂停 OSA (*P* = 0.6)无关。颈围的增大与喉镜暴露困难有关，但是和困难插管无关(*P* = 0.02)。
结论：病态肥胖症病人斜坡位减肥手术时，困难插管或者喉镜暴露困难和阻塞性呼吸暂停, 体重指数, 或者颈围这些因素的有无及严重程度无关。只有 Mallampati 评分为 3 或者 4 和男性病人才预示着困难插管。
（陈灵科 译 陈杰 校）

BACKGROUND: Morbid obesity (MO), obstructive sleep apnea (OSA), and neck circumference (NC) are widely believed to be independent risk factors for difficult tracheal intubation. In this study, we sought to determine whether these factors were associated with increased risk of difficult intubation in patients undergoing bariatric surgery. The predictive factors tested were OSA and its severity, as determined by apnea-hypopnea index (AHI), gender, NC, and body mass index (BMI).
METHODS: All sequentially enrolled MO patients underwent preoperative polysomnography. Severity of OSA was quantified using AHI and the American Society of Anesthesiologists' OSA severity scale. All patients had a standardized anesthetic that included positioning in the "ramped position" for direct laryngoscopy.
RESULTS: One hundred eighty consecutive patients were recruited, 140 women and 40 men. The incidence of OSA was 68%. The mean BMI was 49.4 kg/m^2. The mean AHI was 31.3 (range, 0-135). All the patients' tracheas were intubated successfully without the aid of rescue airways by anesthesiology residents. Six patients required three or more

intubation attempts, a difficult intubation rate of 3.3%. There was an 8.3% incidence of difficult laryngoscopy, defined as a Cormack and Lehane Grade 3 or 4 view. There was no relationship between NC and difficult intubation (odds ratio 1.02, 95% confidence interval 0.93-1.1), between the diagnosis of OSA and difficult intubation ($P = 0.09$), or between BMI and difficult intubation (odds ratio 0.99, 95% confidence interval 0.92-1.06, $P = 0.8$). There was no relationship between number of intubation attempts and BMI ($P = 0.8$), AHI ($P = 0.82$), or NC ($P = 0.3$). Mallampati Grade III or more predicted difficult intubation ($P = 0.02$), as did male gender ($P = 0.02$). Finally, there was no relationship between Cormack and Lehane grade and BMI ($P = 0.88$), AHI ($P = 0.93$), or OSA ($P = 0.6$). Increasing NC was associated with difficult laryngoscopy but not difficult intubation ($P = 0.02$).
CONCLUSIONS: In MO patients undergoing bariatric surgery in the "ramped position," there was no relationship between the presence and severity of OSA, BMI, or NC and difficulty of intubation or laryngoscopy grade. Only a Mallampati score of 3 or 4 or male gender predicted difficult intubation.

PEEP 提高使用大剂量肾上腺素 CPR 大鼠模型的存活率

Positive End-Expiratory Pressure Improves Survival in a Rodent Model of Cardiopulmonary Resuscitation Using High-Dose Epinephrine

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背景：多种干预措施用于心肺复苏（CPR）的研究，以优化药物的使用、胸外按压和通气功能。没有研究显示呼气末正压（PEEP）对于 CPR 结果的作用。作者推测，由于呼气末正压可扭转肺不张，降低肺血管阻力，并有可能改善心输出量，因此，CPR 中使用 PEEP 将提高存活率。
方法：麻醉的 Sprague - Dawley 大鼠暴露于 1 分钟窒息心脏骤停。复苏程序为标准化的胸部按压、吸氧（Fio_2 1.0）和静脉注射肾上腺素 30μg/kg（组 1）和 10μg/kg（组 2）。使用超声心动图评估左心功能（组 1），大鼠在 CPR 开始阶段或者是整个复苏过程中随机接受 5cm H_2O PEEP 或 0 PEEP。存活定义为初步复苏后，自主循环恢复并持续 60min 或者 120min（组 2）。
结果：组间基础情况无差异。在组 1，与 0 PEEP 相比较，接受 5 cm H_2O PEEP (Fio_2 1.0 and 0.21)可提升其存活率（7/9 and 6/6 vs 0/9, $P < 0.01$ and <0.001）。应用 5 cm H_2O PEEP (Fio_2 1.0)能够增加左室舒张末期容积，全身氧供和功能残气量。呼气末正压的使用并没有影响左室收缩功能或动脉血压。其结果差异不是因为氧合的增加，因为其存活率依次为 5 cm H_2O PEEP (Fio_2 1.0) ≥5 cm H_2O PEEP (Fio_2 0.21) >

zero PEEP (Fio_2 1.0)，然而其动脉血氧分压的排序依次为 5 cm H_2O PEEP (Fio_2 1.0) > 5 cm H_2O PEEP (Fio_2 0.21) ≈zero PEEP (Fio_2 1.0)。在另外一个组中，使用 10 μg/kg 肾上腺素，即使其存活率为 100% ，PEEP 的益处仍有限。

结论：啮齿动物窒息心跳骤停模型，在 CPR 期间和之后持续呼气末正压通气（5 cm H_2O），对于存活率有多方面的有益作用，但与吸入氧浓度无关，且无不良心血管效应。

（张蕾 译 陈杰 校）

BACKGROUND: Multiple interventions have been tested in models of cardiopulmonary resuscitation (CPR) to optimize drug use, chest compressions, and ventilation. None has studied the effects of positive end-expiratory pressure (PEEP) on outcome. We hypothesized that because PEEP can reverse pulmonary atelectasis, lower pulmonary vascular resistance, and potentially improve cardiac output, its use during CPR would increase survival.

METHODS: Anesthetized Sprague-Dawley rats were exposed to 1 min of asphyxial cardiac arrest. Resuscitation was standardized and consisted of chest compressions, oxygen (Fio_2 1.0), and IV epinephrine 30 μg/kg (Series 1) and 10 μg/kg (Series 2). Left ventricular function was assessed by echocardiography (Series 1), and animals were randomized to receive either 5 cm H_2O PEEP or zero PEEP at commencement of CPR and throughout resuscitation. Survival was defined as the presence of a spontaneous circulation 60 or 120 min (Series 2) after initial resuscitation.

RESULTS: There were no baseline differences between the groups. In Series 1, administration of 5 cm H_2O PEEP (Fio_2 1.0 and 0.21) was associated with improved survival compared with zero PEEP (7/9 and 6/6 vs 0/9, $P < 0.01$ and <0.001, respectively). Application of 5 cm H_2O PEEP (Fio_2 1.0) increased left ventricular end-diastolic area, systemic oxygenation, and functional residual capacity. Use of PEEP during CPR did not adversely affect left ventricular systolic function or arterial blood pressure. The outcome differences were not due to increased oxygenation because the rank order of survival was 5 cm H_2O PEEP (Fio_2 1.0) ≈5 cm H_2O PEEP (Fio_2 0.21) > zero PEEP (Fio_2 1.0), whereas the rank order of Pao_2 was 5 cm H_2O PEEP (Fio_2 1.0) > 5 cm H_2O PEEP (Fio_2 0.21) ≈zero PEEP (Fio_2 1.0). In an additional series in which epinephrine 10 μg/kg was used (Series 2), the survival was 100% with no beneficial effects of PEEP.

CONCLUSION: In asphyxial cardiac arrest in a small rodent model, continuous application of PEEP (5 cm H_2O) during and after CPR had beneficial effects on survival that were independent of oxygenation and without adverse cardiovascular effects.

肥胖对于妊娠患者神经阻滞技术难度的影响：一项前瞻性、观察研究

The Effect of Obesity on Neuraxial Technique Difficulty in Pregnant Patients: A Prospective, Observational Study

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背景：从业者常常假定肥胖会增加妊娠患者神经阻滞技术的难度，但是很少有人系统地调查过与硬膜外或蛛网膜下腔阻滞相关的危险因素。作者设计这项前瞻性研究来预测妊娠患者神经阻滞技术困难的因素。
方法：使用一前瞻性、观察表，观察妊娠患者神经阻滞困难的多项潜在的危险因素，包括体重指数，能够触摸到棘突的程度，最大的背弯曲度，脊柱侧凸和从业人员的经验。用两种方法来评估神经阻滞麻醉的难点：1）到达所需间隙的进针深度；2)从皮肤进针到蛛网膜下腔注射或硬膜外导管放置妥善所需的时间。将这些数据代入一个广义的负误差二项式线性模型从而决定总的进针过程的预测。神经阻滞麻醉时间的预测取决于其麻醉操作时间的线性模型。生存模型用来说明主治医生干预住院医生过程中的偏差。
结果：研究了 427 例妊娠患者的神经阻滞麻醉程序，对于进针深度和操作时间，重要的预测难点是医生触摸病人骨标志的能力和病人的背弯曲程度。肥胖，即体重指数不是一个独立的预测因子。尽管如此，肥胖的确能预测触摸骨标志的能力和背的弯曲度。
结论：尽管肥胖可能会导致神经阻滞困难，但一些肥胖病人令人惊讶的很容易进行神经阻滞。当进行任何一例妊娠患者尤其是肥胖产妇的神经阻滞麻醉时，背弯曲度和骨标志的触摸能预测神经阻滞技术的难度。
（唐颖 译 陈杰 校）

BACKGROUND: Practitioners often presuppose that obesity will increase neuraxial technique difficulty in pregnant patients, but few investigators have systematically examined this population for risk factors associated with difficult epidural or spinal needle placement. We designed this study to prospectively identify factors that predict neuraxial technique difficulty in pregnant patients.
METHODS: Using a prospective, observational format, pregnant patients were examined for multiple potential risk factors for neuraxial technique difficulty, including current body mass index, ability to palpate spinous processes, maximum back flexion, scoliosis, and experience of the practitioner. Neuraxial technique difficulty was then assessed using two measures: 1) the number of needle passes needed to reach the desired space, and 2) the placement time from skin infiltration to either spinal injection or epidural catheter threading. Predictors of total needle passes were determined by fitting the data to a generalized linear model with negative binomial error. Predictors of neuraxial anesthetic time were determined by fitting a linear model to the log of neuraxial anesthetic placement time. A survival model was used to account for bias introduced when attending physicians intervened in resident physician procedures.
RESULTS: Neuraxial procedures in 427 pregnant patients were studied. For both the number of needle passes and the neuraxial anesthetic placement time, the significant predictors of difficulty were the practitioner's ability to palpate the patient's bony landmarks and the patient's ability to flex her back. Obesity, as measured by body mass index, was not an independent predictor of either end point. Obesity did, however, strongly predict both the ability to palpate landmarks and flex the back.

CONCLUSIONS: Despite concerns that obesity may cause difficulty with neuraxial technique, some obese patients have surprisingly easy neuraxial block placements. When approaching any neuraxial anesthetic in a pregnant patient, and especially in the obese parturient, back flexion and landmark palpation predict neuraxial technique difficulty.

羟考酮与吗啡在腹腔镜子宫切除术后的病人静脉自控镇痛之比较

A Comparison of Intravenous Oxycodone and Intravenous Morphine in Patient-Controlled Postoperative Analgesia After Laparoscopic Hysterectomy

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背景：在这项研究中，作者研究了术后有内脏疼痛患者所需羟考酮和吗啡的剂量，缓解疼痛的程度以及副作用。
方法：91 位行腹腔镜子宫切除术的女患者在手术结束前接受羟考酮或吗啡静脉注射，并在术后 24 小时给予病人自控镇痛术。
结果：羟考酮的累积消耗量比吗啡少（13.3 ± 10.4 mg 比 22.0 ± 13.1 mg, P = 0.001）。使用羟考酮，术后第一小时的视觉模拟量表评分显著低，但术后 24 小时的镇静效果差，P=0.006。
结论：与吗啡相比，羟考酮更适于缓解内脏疼痛，但不适用于镇静。
（邹巧群 译 陈杰 校）

INTRODUCTION: In this study, we investigated the dose requirements, pain relief, and side effects of oxycodone versus morphine after surgery with visceral pain.
METHODS: Ninety-one women received IV oxycodone or morphine before the end of laparoscopic hysterectomy and then continued with patient-controlled analgesia for 24 h postoperatively.
RESULTS: The accumulated oxycodone consumption was less (13.3 ± 10.4 mg vs 22.0 ± 13.1 mg, P = 0.001) than morphine. With oxycodone, the visual analog scale scores were significantly lower in the first hour postoperatively and sedation was less during the 24-h postoperative period, P = 0.006.
CONCLUSIONS: Oxycodone was more potent than morphine for visceral pain relief but not for sedation.

内啡肽-1 和犬尿喹啉酸在大鼠炎症关节模型中的外周抗伤害效应

The Peripheral Antinociceptive Effects of Endomorphin-1 and Kynurenic Acid in the Rat Inflamed Joint Model

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背景：大量数据显示，吗啡和 n-甲基-d-天冬氨酸（NMDA）受体都位于外周水平，而作用于这些受体的药物经局部给药后，均可产生抗伤害作用，但是这些受体的内源性配体的抗伤害效应目前仍不明确。本次研究的目的在于确定内源性阿片肽，即内啡肽-1（EM1）和内源性 NMDA 受体拮抗剂犬尿喹啉酸（KYNA）的抗伤害效能，并在大鼠验证关节模型中研究两者在外周水平的相互作用。
方法：在大鼠右后足胫骨跗骨关节内注射角叉菜聚糖（300 μg/20μL）以产生机械性超敏反应。用 von Frey 细丝（0.064-110 g）评估其机械痛阈。将 EM1（30、100 和 200μg）、KYNA（30、100、200 和 400μg）以及以 1:1 的比例相混的两者的混合液分别注入受感染的关节，并在给药后 75min 时再次测定痛阈。
结果：向炎症关节注射 EM1 或 KYNA 对非炎症侧的痛阈没有影响。两者的配体都能产生剂量依赖性的抗痛觉过敏效应，且最大剂量可导致延迟效应。相较于 KYNA（30%有效剂量[ED30]和 50%有效剂量[ED50]分别为 204μg[可信区间{CI}：160-251]和 330μg[CI：280-407]），EM1（ED30 和 ED50 分别为 112μg[CI：80-146]和 167μg[CI：135-220]）的抗伤害效能更强大。预先皮下应用纳曲酮可抑制 EM1 的抗伤害效应。同时应用 EM1 和 KYNA 可产生增强的和/或延迟的抗伤害效应。混合液的 ED30 和 ED50 分别为 141μg[CI：83-182]和 231μg[CI：190-293]，这与两者理论上的叠加值（ED30 和 ED50 分别为 145μg[CI：68-237]和 220μg[CI：144-230]）没有明显的差异，也就是说两个配体的相互作用产生的是叠加效应。所有实施的治疗均未产生任何并发症。
结论：外周给予内源性阿片类激动剂和 NMDA 受体拮抗剂的配体可能可以对抗炎性疼痛。由于这两种药物很少越过血脑屏障，因此局部使用不会产生中枢性的副作用。
（周姝婧 译 陈杰 校）
BACKGROUND: Several data suggest that both opioid and *N*-methyl-d-aspartate (NMDA) receptors are localized at the peripheral level, and drugs acting on these receptors may produce antinociception after topical administration; however, the antinociceptive effect of endogenous ligands at these receptors is poorly clarified. Our goal in this study was to determine the antinociceptive potency of the endogenous opioid peptide, endomorphin-1 (EM1), and the endogenous NMDA receptor antagonist, kynurenic acid (KYNA), and their interaction at the peripheral level in the rat inflamed joint model.
METHODS: Mechanical hypersensitivity was produced by injection of carrageenan (300 μg/20 μL) into the tibiotarsal joint of the right hind leg. The mechanical pain threshold was assessed by von Frey filaments (0.064-110 g). EM1 (30, 100, and 200 μg), KYNA (30, 100, 200, and 400 μg), and their combinations in a fixed-dose ratio (1:1) were injected into the inflamed joint, and the pain threshold was determined repeatedly for 75 min after the drug administrations.
RESULTS: Neither EM1 nor KYNA administered to the inflamed joint influenced the pain threshold at the noninflamed side. Both ligands produced dose-dependent antihyperalgesia, and the highest doses caused a prolonged effect. EM1 had higher potency (30% effective dose [ED_{30}] and 50% effective dose [ED_{50}] values were 112 μg [confidence interval {CI}: 80-146] and 167 μg [CI: 135-220], respectively) compared

with KYNA (ED_{30} and ED_{50} values were 204 μg [CI: 160-251] and 330 μg [CI: 280-407], respectively). The antinociceptive effect of EM1 was prevented by subcutaneous naltrexone pretreatment. The coadministration of EM1 with KYNA caused an enhanced and/or prolonged antinociceptive effect. The ED_{30} and ED_{50} values of the combination were 141 μg [CI: 83-182] and 231 μg [CI: 190-293], respectively, which did not differ significantly from the theoretically additive values (ED_{30} and ED_{50} values were 145 μg [CI: 68-237] and 220 μg [CI: 144-230], respectively), thus the interaction between these ligands is additive. None of the treatments caused any sign of side effects.
CONCLUSION: Peripherally administered endogenous opioid agonist and NMDA receptor antagonist ligands might be beneficial in inflammatory pain. Because both drugs barely cross the blood-brain barrier, their local administration causes no central side effects.

大鼠通过舔（Licking）后爪可减少福尔马林致痛实验后脊髓背角胞外信号调节激酶的磷酸化

Licking Decreases Phosphorylation of Extracellular Signal-Regulated Kinase in the Dorsal Horn of the Spinal Cord After a Formalin Test

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背景：伤害性行为可能减弱痛觉。最近研究表明胞外信号调节磷酸激酶(pERK)是由脊髓后角神经伤害性刺激引起的。作者研究福尔马林实验中脊髓后角的pERK是否受通过舔（licking）的影响。
方法：将 24 只成年大鼠分为四组：对照组，福尔马林致痛实验组，活动受限对照组及活动受限福尔马林致痛实验组。福尔马林致痛实验组及活动受限福尔马林致痛实验组大鼠在其后爪皮下注射 10%福尔马林。对照组与福尔马林致痛实验组小鼠放置在一干净塑料小室中，而活动受限对照组及活动受限福尔马林致痛实验组大鼠置于一改良制动管状小室中。所有大鼠在 25min 后处死。使用免疫组化技术卵白素生物素过氧化物酶方法检测腰椎十二节段的 pERK 值。
结果：在活动受限福尔马林致痛实验中，患侧浅层脊髓后角 pERK 阳性细胞数量显著高于其它三组($P < 0.05$)。福尔马林致痛实验组的 pERK 表达与其他两组并无显著性差异。
结论：大鼠通过舔后爪减少福尔马林致痛实验中脊髓后角pERK的表达。这一发现表明通过舔后爪减弱了福尔马林致痛实验的疼痛。
（赵嫣红 译 陈杰 校）

BACKGROUND: Nociceptive behaviors might attenuate pain sensation. Phosphorylation of extracellular signal-regulated kinase (pERK) was recently reported to be induced by noxious stimuli in dorsal horn neurons. We investigated, in a formalin test, whether pERK of the dorsal horn is affected by licking.
METHODS: Twenty-four adult male rats were divided into four groups: control, formalin test, restricted control, and restricted formalin test. Ten percent formalin was

injected subcutaneously into the left rear paw of the formalin test and restricted formalin test groups. The control and formalin test group rats were kept in a clear plastic chamber, whereas the restricted control and restricted formalin test group rats were kept in a modified-restraint, pipe-shaped chamber. All rats were killed after 25 min. Twelve sections of the lumbar spinal cord were processed for p-ERK immunohistochemistry using the avidin-biotin peroxidase method.
RESULTS: The number of p-ERK positive cells in the restricted formalin test group was significantly higher than in the other three groups in the ipsilateral-side superficial dorsal horn ($P < 0.05$). However, there was no significant difference between the formalin test group and the two control groups in pERK expression.
CONCLUSION: Licking decreased pERK of the spinal cord of the formalin test group. The findings suggested that licking attenuated the pain of the formalin test.

下肢手术鞘内注射时布比卡因、左旋布比卡因以及罗哌卡因的半数有效剂量

The Median Effective Dose of Bupivacaine, Levobupivacaine, and Ropivacaine After Intrathecal Injection in Lower Limb Surgery

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背景：鞘内麻醉普遍应用于下肢手术，布比卡因、左旋布比卡因以及罗哌卡因都已被用作鞘内药物，但它们的相对效价目前还没完全确定。在这项研究中，作者拟确定这三种局部麻醉药用于下肢手术鞘内麻醉的半数有效剂量(ED_{50})，从而评估三者的相对效价。
方法：75 名腰硬联合麻醉下行下肢手术的病人随机分组，分别鞘内注射布比卡因或左旋布比卡因或罗哌卡因。局部麻醉药的剂量使用升降序贯分配法。每组第一个病人的剂量为 8mg，剂量增加值设定为 1mg。每组的序贯剂量由先前一个病人接受腰麻成功或失败的结果来决定。鞘内注射后双侧 T12 冷感觉阻滞保留 20 分钟，并且手术至少能顺利进行至鞘内注射后 50min，其间不补充硬膜外麻醉药物，则为成功事件。用 Dixon and Massey 方法计算 ED_{50}。
结果：鞘内麻醉时布比卡因的 ED_{50} 为 5.5mg，（95%的置信区间[CI]：4.90-6.10mg），左旋布比卡因的 ED_{50} 为 5.68mg，（95%的 CI：4.92-6.44mg），罗哌卡因的 ED_{50} 为 8.41mg，（95%的 CI：7.15-9.67mg）。左旋布比卡因与布比卡因麻醉相对效价比值是 0.97 (95%的 CI:：0.81-1.17)，罗哌卡因与布比卡因麻醉相对效价比值是 0.65 (95%的 CI： 0.54-0.80)，而罗哌卡因与左旋布比卡因麻醉相对效价比值是 0.68 (95%的 CI：0.55-0.84)。

结论：这项研究显示，下肢手术鞘内麻醉时罗哌卡因的效价低于左旋罗哌卡因和布比卡因，而左旋罗哌卡因和布比卡因的效价相当。
（黄丹 译 陈杰 校）

BACKGROUND: Intrathecal anesthesia is commonly used for lower limb surgery. Bupivacaine, levobupivacaine, and ropivacaine have all been used as intrathecal drugs, but their relative potency in this context has not been fully determined. In this study, we determined the median effective dose (ED_{50}) of these three local anesthetics for intrathecal anesthesia in lower limb surgery and hence their relative potencies.
METHODS: Seventy-five patients scheduled for lower limb surgery under combined spinal-epidural anesthesia were randomly allocated to one of three groups receiving intrathecal bupivacaine, levobupivacaine, or ropivacaine. The dose of local anesthetic was varied using up-down sequential allocation technique. The dose for the first patient in each group was 8 mg, and the dosing increment was set at 1 mg. Subsequent doses in each group were determined by the outcome in the previous patient using success or failure of the spinal anesthesia as the primary end point. A success was recorded if a bilateral T12 sensory block to cold was attained within 20 min after intrathecal injection, and the surgery proceeded successfully until at least 50 min after the intrathecal injection without supplementary epidural injection. The ED_{50} was calculated using the method of Dixon and Massey.
RESULTS: The ED_{50}s were 5.50 mg for bupivacaine (95% confidence interval [CI]: 4.90–6.10 mg), 5.68 mg for levobupivacaine (95% CI: 4.92–6.44 mg), and 8.41 mg for ropivacaine (95% CI: 7.15–9.67 mg) in intrathecal anesthesia. The relative anesthetic potency ratios are 0.97 (95% CI: 0.81–1.17) for levobupivacaine/bupivacaine, 0.65 (95% CI: 0.54–0.80) for ropivacaine/bupivacaine, and 0.68 (95% CI: 0.55–0.84) for ropivacaine/levobupivacaine.
CONCLUSION: This study suggests that in intrathecal anesthesia for lower limb surgery, ropivacaine is less potent than levobupivacaine and bupivacaine, whereas the potency is similar between levobupivacaine and bupivacaine.

体外全血实验发现抑制 XIII 因子会阻碍血凝形成，降低血凝块稳定性并增加纤维蛋白溶解效应

In Vitro Inhibition of Factor XIII Retards Clot Formation, Reduces Clot Firmness, and Increases Fibrinolytic Effects in Whole Blood.

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背景：在围手术期进行血栓弹力图的检查又重新引起了人们的兴趣。血栓弹力图检查结果的主要决定因素包括凝血因子浓度（各种酶原和纤维原）和血小板计数，因此血小板抑制剂会使得主要受凝血因子影响的检测指标变得无意义。从而使得合并应用血小板抑制剂与否的检测结果逐渐被用于评估和检测血液制品替代治疗的效果。在本次研究中，我们评估了 XIII 因子抑制剂配伍应用糖蛋白（GP）IIb/IIIa（血小板抑制）在全血血栓弹力图中的效应，以及应用 XIII 因子抗体对常规检测做一改进是否可用于检测 XIII 因子缺乏症。
方法：正常全血会随着非特异性抗体、抗 GPIIb/IIIa 抗体或者中性抗 XIII 因子抗体数量的增加而被孵化改变。样本在经过组织因子激活和血小板抑制的处理后进行分

析，得到全血血栓弹力图。凝血时间、血凝块形成时间、最大血凝块稳定性以及60分钟内血凝块溶解程度被一式三份进行检测。另外，25个全血常规样本分别用以下两种方法来检测XIII因子缺乏症：合并应用XIII因子抗体的新血栓弹力图分析法和标准XIII因子检测法，并将两种方法进行比较。
结果：虽然GPIIb/IIIa抑制剂并没有改变经血小板抑制的全血血栓弹力图的检测结果，但是XIII因子抑制剂却明显地降低了最大血凝块稳定性（(P = 0.020），延长了血凝块形成时间（P = 0.025）以及血凝块溶解程度（P = 0.007），而凝血时间并未改变；并且在全血（而非血浆）中随着抗体浓度的增加这一改变存在着封顶效应。应用这一血栓弹力图检测方法来发现XIII因子缺乏症，当检测效能<70%时，敏感度为90%，且存在阴性预测值（受试者特征性曲线下面积为0.803，P = 0.0015）；当检测效能<60%时，敏感度和阴性预测值均为100%（受试者特征性曲线下面积为0.84，P = 0.0037）。
结论：XIII因子会明显地影响血小板抑制剂激活的全血血栓弹力图结果。在分析出血患者，特别是接受促凝血治疗的患者的血栓弹力图结果的时候，应考虑到这一现象。由抗体介导的XIII因子抑制剂可被用于建立以血栓弹力图为基础的检验方法来发现XIII因子缺乏症。
（单嘉琪译 薛张纲校）
BACKGROUND: Thrombelastography has received renewed interest in the perioperative setting. The main determinants of thrombelastographic results are coagulation factor concentrations (various zymogens and fibrinogen) and platelet count; thus, platelet inhibition renders these assays mainly coagulation factor dependent. Assays with and without platelet inhibition are thus increasingly used to trigger and monitor replacement therapy with blood products. In this study, we evaluated the effect of factor XIII inhibition and additional glycoprotein (GP) IIb/IIIa blockade on (platelet-inhibited) whole blood thrombelastography and whether a modified routine assay (using factor XIII antibody) can be used to detect factor XIII deficiency.
METHODS: Normal whole blood was incubated with increasing amounts of a nonspecific antibody, an anti-GPIIb/IIIa antibody, or a neutralizing anti-factor XIII antibody; samples were analyzed with a tissue factor-activated and platelet-inhibited whole blood thrombelastographic assay. Clotting time, clot formation time, maximum clot firmness, and clot lysis at 60 min were evaluated in triplicate. Also, 25 whole blood routine samples were evaluated for factor XIII deficiency using a new thrombelastographic assay incorporating a factor XIII antibody and using a standard factor XIII assay for comparison.
RESULTS: Although GPIIb/IIIa inhibition did not alter the results of the platelet-inhibited whole blood thrombelastography, factor XIII inhibition significantly reduced maximum clot firmness (P = 0.020) and increased clot formation time (P = 0.025) and clot lysis (P = 0.007), leaving clotting time unchanged; a ceiling effect seemed to be present with increasing antibody concentrations in whole blood (but not plasma). The thrombelastographic assay for factor XIII deficiency (<70% activity) had a 90% sensitivity and negative predictive value (area under receiver operating characteristic curve 0.803, P = 0.0015); for a deficiency <60%, sensitivity and negative predictive value were 100% (area under receiver operating characteristic curve 0.84, P = 0.0037).

CONCLUSION: Factor XIII has significant impact on platelet-inhibited activated whole blood thrombelastography. This phenomenon should be considered when interpreting thrombelastographic results in the bleeding patient, especially when the results trigger procoagulant therapy. Antibody-mediated factor XIII inhibition can be used to establish thrombelastography-based assays to detect factor XIII deficiency.

肌强直和恶性高热的易感性

The Myotonias and Susceptibility to Malignant Hyperthermia

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恶性高热是一种骨骼肌遗传性疾病，吸入麻醉药可以触发肌浆网内 Ca^{2+}的释放从而导致机体高代谢、肌肉强直、横纹肌溶解，严重可以致死。肌强直是一种疾病，它是由于与骨骼肌兴奋收缩耦联相关的基因突变所造成的，特殊的 DNA 序列重复造成诸如骨骼肌通道在内的许多蛋白质不稳定，从而影响的骨骼肌的兴奋性。一般认为有肌强直的病人增加了发展为恶性高热的风险。在这篇文章中，我们概述了肌肉兴奋性及兴奋收缩耦联的生理过程，恶性高热及肌强直的病理过程，并参考了和恶性高热易感性相关的文献。我们得出了如下结论，除了低钾周期性麻痹外，其他肌病患者出现恶性高热的几率与普通人群相似。由于没有低钾周期性麻痹的病人发展为恶性高热的报道，尽管我们认为这类病人的发病率很低，但是我们尚不能评估其发展恶性高热的风险。

（陈珺珺译 薛张纲校）

Malignant hyperthermia (MH) is a pharmacogenetic disorder of skeletal muscle in which volatile anesthetics trigger a sustained increase in intramyoplasmic Ca^{2+} via release from sarcoplasmic reticulum and, possibly, entry from the extracellular milieu that leads to hypermetabolism, muscle rigidity, rhabdomyolysis, and death. Myotonias are a class of myopathies that result from gene mutations in various channels involved in skeletal muscle excitation-contraction coupling and sarcolemmal excitability, and unusual DNA sequence repeats that result in the inability of many proteins, including skeletal muscle channels that affect excitability, to undergo proper splicing. The suggestion has often been made that myotonic patients have an increased risk of developing MH. In this article, we review the physiology of muscle excitability and excitation-contraction coupling, the pathophysiology of MH and the myotonias, and review the clinical literature upon which the claims of MH susceptibility are based. We conclude that patients with these myopathies have a risk of developing MH that is equivalent to that of the general population with one potential exception, hypokalemic periodic paralysis. Despite the fact that there are no clinical reports of MH developing in patients with hypokalemic periodic paralysis, for theoretical reasons we cannot be as certain in estimating their risk of developing MH, even though we believe it is low.

长时间丙泊酚麻醉与血乳酸增加不相关

Prolonged propofol anesthesia is not associated with an increase in blood lactate.

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背景：乳酸性酸中毒常被认为是异丙酚输注综合征的先期标志。此研究旨在探讨长时间使用丙泊酚与挥发性吸入麻醉药（VA）后血乳酸和 pH 值的变化情况。

方法：回顾术程长于 8 小时的择期脊柱手术患者的麻醉记录，从中获取手术与人口统计学资料。根据麻醉时间（AT）（±30min）和出血量（BL）（±500ml），按 1：2 的比例，将接受丙泊酚麻醉的患者与接受 VA 麻醉的患者进行配比。

结果：在所回顾的 246 名患者中，共选取 50 名接受丙泊酚麻醉的患者（AT=10±2h，BL=1955±1409ml）与 100 名接受 VA 麻醉的患者（AT=10±1h，BL=1801±1543 毫升），其中分别有 40 和 72 名患者拥有完备的基线与麻醉后 8 小时血乳酸数据，故将其纳入主要分析。丙泊酚组患者接受丙泊酚的平均剂量为 8.8±2mg/kg/h。VA 组患者其年龄大于丙泊酚组患者（分别为 58±12 岁与 51±15 岁，P=0.002），但两者在性别、ASA 分级、术中血流动力学指标和血管升压药的使用等方面均无差异。8 小时后，与丙泊酚组相比，VA 组患者动脉血乳酸水平较基线值大幅增加（与基线值相比：丙泊酚组增幅为 0.48±0.72mmol/L；VA 组增幅为 1.2±1.2mmol/L，P=0.001）。

结论：在术程长于 8 小时的脊柱手术中，与输注异丙酚相比，接受 VA 麻醉可使血乳酸水平大幅增加。但这一结论的确切机制和临床意义仍需前瞻性研究进一步阐明。

（范羽译 薛张纲校）

BACKGROUND: Lactic acidosis is considered an early sign of propofol infusion syndrome. In this study, we investigated the changes in lactate and pH with propofol versus volatile anesthesia (VA) of long duration.

METHODS: Demographic and intraoperative data were recorded retrospectively from the anesthesia records of patients who underwent elective spine surgery longer than 8 h. Propofol patients were matched 1:2 to VA patients, based on anesthesia time (AT) (±30 min) and blood loss (BL) (±500 mL).

RESULTS: Of 246 patients identified, 50 received propofol (AT = 10 ± 2 h, BL = 1955 ± 1409 mL) and were matched to 100 VA cases (AT = 10 ± 1 h, BL = 1801 ± 1543 mL), and of those, 40 and 72 patients, respectively, had complete lactate data at baseline and at 8 h after anesthesia and were included in the main analysis. The propofol group received 8.8 ± 2 mg · kg–1 · h–1 of propofol. The VA group age was older than the propofol group (58 ± 12 vs 51 ± 15 yr, respectively, P = 0.002), but there was no difference between the groups in gender, ASA grade, intraoperative hemodynamic variables, and use of vasopressors. After 8 h, the VA group had a larger increase in arterial lactate from baseline compared with the propofol group (change from baseline: propofol, 0.48 ± 0.72 mmol/L; VA, 1.2 ± 1.2 mmol/L, P = 0.001).

CONCLUSIONS: During prolonged spine surgery >8 h, VA was associated with higher serum lactate, when compared with propofol infusion. Prospective studies are needed to elucidate the exact mechanisms and clinical implications of this finding.

异氟烷抑制SH-SY5Y细胞的环磷酸腺苷反应元件结合蛋白磷酸化以及钙调蛋白易位至细胞核

Isoflurane inhibits cyclic adenosine monophosphate response element-binding protein phosphorylation and calmodulin translocation to the nucleus of SH-SY5Y cells.

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背景：CaM(钙调蛋白)被钙离子激活后易位至细胞核，从而刺激CREB(环磷酸腺苷反应元件结合蛋白)磷酸化形成P-CREB(磷酸化环磷酸腺苷反应元件结合蛋白)。这一过程是新基因表达所必须经过的步骤，与长期的增效作用有关，同时也是记忆形成中非常重要的步骤。异氟烷可以影响记忆，因此我们想测试下它是否会干涉CaM易位至神经细胞细胞核的过程以及减弱P-CREB的合成作用。

方法：从人神经母细胞系培养出SH-SY5Y细胞，在KCl作用下去极化。使用西式印迹法、酶联免疫吸附试验和免疫细胞化学的方法检测CREB的磷酸化作用。在去极化后经过分次的溶解离心检测出CaM从胞质至胞核的易位的数量。CaM通过免疫细胞化学的方法定位，西式印迹和显像法定量。在KCl去极化之前和过程中，将细胞分别暴露在异氟烷、异氟烷复合Bay K8644、尼群地平以及Ω-芋螺毒素GVIa中。

结果：细胞经KCl去极化后P-CREB的数量增加，峰值出现在去极化开始后30秒。尼群地平对这一过程具有抑制作用，Ω-芋螺毒素没有明显影响，异氟烷则是具有剂量依赖性作用。经L型钙离子通道激动剂Bay K 8644预处理后，可以减弱异氟烷对P-CREB的抑制作用。经KCl去极化后CaM开始出现在细胞核内。尼群地平抑制CaM的易位，而异氟烷减弱这一作用，Bay K 8644预处理可以减轻异氟烷对CaM易位至细胞核的抑制作用。

结论：我们的数据显示异氟烷可以抑制CaM的易位过程和P-CREB的形成。这很可能是由于异氟烷抑制了L型钙离子通道对钙离子的通透性。

（黄剑译 薛张纲校）

BACKGROUND: Calmodulin (CaM) activation by Ca(2+), its translocation to the nucleus, and stimulation of phosphorylation of cyclic adenosine monophosphate response element-binding protein (CREB) (P-CREB) are necessary for new gene expression and have been linked to long-term potentiation, a process important in memory formation. Because isoflurane affects memory, we tested whether isoflurane interfered with the translocation of CaM to the neuronal cell nucleus and attenuated the formation P-CREB.

METHODS: SH-SY5Y cells, a human neuroblastoma cell line, were cultured. Cells were depolarized with KCl and the phosphorylation of CREB examined by Western blotting, enzyme-linked immunosorbant assay, and immunocytochemistry. The translocation of CaM from the cytosol to the nucleus was also examined after

depolarization. Cells were depolarized and lysed and fractionated by centrifugation to determine the amount of CaM translocated to the nucleus. CaM was localized by immunocytochemistry and quantitated by Western blotting and imaging. Before and during KCl depolarization, cells were exposed to isoflurane, isoflurane plus BΩay K 8644, nitrendipine, and Ω-conotoxin GVIa, respectively.
RESULTS: P-CREB increased after KCl depolarization. The increase of P-CREB peaked at depolarization duration of 30 s. The increase in P-CREB formation was inhibited by nitrendipine, but not omega-conotoxin, and by isoflurane in a concentration-dependent fashion. Pretreatment with the L-type Ca(2+) channel agonist, Bay K 8644, attenuated the inhibition of P-CREB formation by isoflurane. CaM presence in the nucleus occurred after KCl depolarization. CaM translocation was inhibited by nitrendipine and attenuated by isoflurane. Bay K 8644 pretreatment decreased the isoflurane inhibition of CaM translocation to the nucleus.
CONCLUSIONS: Our data demonstrate that isoflurane inhibits CaM translocation and P-CREB formation. This most likely occurs through isoflurane inhibition of Ca(2+)entry through L-type Ca(2+) channels.

通过小儿中心静脉注入系统对药物输注动力学的分析：量化达到设定剂量的延迟

An Analysis of Drug Delivery Dynamics via a Pediatric Central Venous Infusion System: Quantification of Delays in Achieving Intended Doses

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背景：小儿患者在重症监护病房和手术室经常需通过中心静脉导管连续注射药物。本研究通过一个标准小儿中心静脉输液系统的实验模型设计给药方案。
方法：评估标准的小儿 8 厘米，4 - f 双腔导管给药。一个注射泵通过一个三通开关连接导管的一侧注射生理盐水作为流体载体。通过三通开关的另一个接口，第二个注射泵以 0.5ml/h 的恒定速率注入模型药物亚甲基蓝，并收集每分钟的输注量进行定量分析。我们 2ml/h 和 12ml/h 的总流量模拟 3kg 婴儿的给药量，并通过用模型药灌注三通管道来模拟药物注射停止后恢复，而不予灌注可模拟一个新的输液。药物泵装置起始的表现被测量以估计对灌注起始时的贡献。
结果：启动一个新的药物输注模型时，导管末端药物达到试验设定浓度的时间通过达到靶浓度的一半的时间来测量。药物输注启动时在总流量低时(t(50) = 23.5 +/- 2.1 min)要远慢于总流量高时(t(50) = 15.7 +/- 2.9 min)。而预先用亚甲基蓝灌注输液管道可显著缩短达到靶浓度的时间(low flow t(50) = 12.7 +/- 0.6 min, high flow t(50) = 5.2 +/- 0.8 min)。停止药物泵后使用高的载体流速时双腔导管末端药物停止的时间(t(50) = 3 +/- 0.5 min)相比于使用低载体流速时(t(50) = 11.6 +/- 0.8 min)显著缩短。药物泵系统的启动性能造成输注启动延迟。
结论：当前的小儿科护理设置的灌注技术在给与患者预期药物剂量时可导致有意义的，未明确的，有潜在危险的延误。总流量率，灌注输液系统，流体路径固定容积，以及输液泵系统的启动性能可能延迟达到药物输注靶速率。
（李莹译 薛张刚校）

BACKGROUND: Pediatric patients frequently receive continuous infusions of drugs via central venous catheters in the intensive care unit and the operating room. This study characterized drug delivery profiles in a quantitative laboratory model of a standard pediatric central venous infusion system.
METHODS: We evaluated drug delivery via a standard pediatric 8-cm, 4-F double-lumen catheter. One syringe pump infused normal saline as the carrier fluid through a limb of a Y-piece connected to the catheter's 22-gauge distal lumen. Through the other limb of the Y-piece, a second syringe pump infused methylene blue, the model drug, at a constant rate of 0.5 mL/h. The volume delivered was collected every minute for quantitative analysis. We compared 2 mL/h and 12 mL/h total flow rates to mimic volume delivery to a 3-kg infant, and priming of the Y-piece with the model drug, to mimic resumption of a stopped drug infusion, versus no priming, to mimic a new infusion.Drug pump system start-up performance was measured to estimate this factor's contribution to infusion onset profiles.
RESULTS: When initiating a new infusion of the model drug, the time to steady-state delivery at the catheter's end varied significantly among the studied scenarios as measured by the time to reach half of the targeted dose (t(50)). Onset of delivery with a low total flow was much slower (t(50) = 23.5 +/- 2.1 min) than with the high flow rate (t(50) = 15.7 +/- 2.9 min). Priming the drug limb of the connecting Y-piece with methylene blue substantially shortened the time to steady state (low flow t(50) = 12.7 +/- 0.6 min, high flow t(50) = 5.2 +/- 0.8 min).Time to cessation of drug delivery to the end of the catheter after stopping the drug pump was substantially shorter using the high carrier flow rate (t(50) = 3 +/- 0.5 min) compared with the low carrier flow rate (t(50) = 11.6 +/- 0.8 min).Drug pump system start-up performance contributed to onset delay.
CONCLUSIONS: Current infusion techniques in the pediatric care setting can result in significant, unrecognized, and potentially hazardous delays in achieving delivery of intended drug doses to the patient. Total flow rate, priming of the infusion system, the dead volume of the fluid path, and the start-up performance of the infusion pump system contribute to delays in achieving targeted rates of drug delivery.

镇痛分娩过程中麻醉相关的并发症的流行病学研究

Epidemiology of Anesthesia-Related Complications in Labor and Delivery, New York State, 2002-2005

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背景：镇痛和分娩过程中与麻醉相关的并发症的流行病学资料对于评估产科麻醉的安全性及有效性是非常有必要的，但是这方面的数据是缺乏的。在我们的这项研究中，我们对纽约医院中产妇进行大规模的研究，旨在调查麻醉相关的并发症的流行病学情况。

方法：使用医疗保健的费用，并应用国家住院病人数据库资料，我们收集了纽约各大医院从 2002 年至 2005 年产科病人的资料。根据第九版国际疾病分类法修订版，我们收集了分娩镇痛过程中出现麻醉相关并发症的病人信息。我们统计了麻醉相关并发症发生的人口特征及临床特征。并使用多因素回归分析的方法评估了麻醉相关并发症的危险因素。

结果：总共调查了 957,471 名产妇，其中 4438 (0.46%)名至少出现了一项麻醉相关并发症。 其中并发症主要是椎管内麻醉相关的并发症（占 55%），其次是系统性症状（占 43%），药物过量及药物副作用占 2%。多因素回归分析揭示了五个麻醉相关并发症的危险因素：剖宫产(优势比[OR] 2.51, 95%置信区间[CI] 2.36-2.68),农村地区(OR 1.33, 95% CI 1.21-1.46), Charlson-Deyo 同病指数≥1 (OR 1.47, 95% CI 1.28-1.69), 高加索人种(OR 1.37, 95% CI 1.24-1.52), 及择期入院(OR 1.10, 95% CI 1.03-1.18)。麻醉相关并发症与平均住院天数增加一天相关（3.89 ± 3.69 [均数±标准差]天 vs 2.92 ± 2.38 天，分娩过程中没有麻醉相关并发症,$P < 0.0001$） ，增加了孕产妇死亡率 22 倍(OR 22.26, 95% CI 11.20-44.24)。

结论：尽管分娩镇痛过程中麻醉相关并发症的发生率较低，但是仍应引起重视，特别是那些行剖宫产的，生活在农村或有合并症的产妇。

（陈珺珺译 薛张纲校）

BACKGROUND: Epidemiologic data on anesthesia-related complications occurring during labor and delivery are essential for measuring and evaluating the safety and quality of obstetric anesthesia care but are lacking. We aimed to fill this research gap by exploring the epidemiologic patterns and risk factors of anesthesia-related complications in a large sample of women giving birth in New York hospitals.

METHODS: Using the Healthcare Cost and Utilization Project State Inpatient Databases files, we identified all discharge records for labor and delivery from New York hospitals between 2002 and 2005. We then identified women who experienced any recorded anesthesia-related complication during labor and delivery as determined by International Classification of Diseases, Ninth Revision, Clinical Modification codes. The incidence of anesthesia-related complications was calculated by demographic and clinical characteristics. Multivariate logistic regression was performed to assess risk factors of anesthesia-related complications.

RESULTS: Of the 957,471 deliveries studied, 4438 (0.46%) had at least one anesthesia-related complication. The majority (55%) of anesthesia-related events occurring during labor and delivery were spinal complications, followed by systemic complications (43%) and overdose or adverse effects (2%). Multivariate logistic regression revealed five risk factors of anesthesia-related complications: cesarean delivery (odds ratio [OR] 2.51, 95% confidence interval [CI] 2.36-2.68), rural area (OR 1.33, 95% CI 1.21-1.46), Charlson-Deyo Comorbidity Index ≥1 (OR 1.47, 95% CI 1.28-1.69), Caucasian race (OR 1.37, 95% CI 1.24-1.52), and scheduled admission (OR 1.10, 95% CI 1.03-1.18). Anesthesia-related complications were associated with about a one-day increase in the average length of stay (3.89 ± 3.69 [mean ± sd] days vs 2.92 ± 2.38 days for deliveries without anesthesia-related complications, $P < 0.0001$) and a 22-fold increased risk of maternal mortality (OR 22.26, 95% CI 11.20-44.24).

CONCLUSION: The incidence of anesthesia-related complications during labor and delivery seems to be low but remains a cause of concern, particularly in women

undergoing cesarean delivery, living in rural areas, or having preexisting medical conditions.

院外心肺复苏的预测模型

A Prediction Model for Out-of-Hospital Cardiopulmonary Resuscitation

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背景：我们创建了一个预测模型来决定是否省去徒然的心肺复苏从而节省资源。

方法：在这个析因分析中，我们评价成功心肺复苏后神经恢复的预测参数。原始试验为单盲、随机、前瞻性、对照、多中心临床试验。

结果：我们入组了 1166 例院前发生心跳骤停并予以高级生命支持的患者。其中 786 例患者（67.4%）在现场死亡，380 例患者（32.6%）被送往医院。265 例患者（22.7%）在医院死亡。115 例患者（9.8%）出院，其中的 92 例能被随访到。出院的 54%的患者（92 例中的 50 例）脑功能恢复良好。46%的病人为意识不清或留有严重残疾。

心室纤颤更有可能在神经功能恢复好的病人中发生（42/50=84.0%），而神经功能恢复差的病人更有可能发生了心跳骤停（9/42=21.4%）。运用 logistic 回归分析，用一个评分来预测死亡的可能性。预测院内死亡其敏感度为 99.8%（953/955），但其特异性为 2.9%（3/104;阈值 0.5）。预测入院直到出院的生存其敏感度为 99%（103/104），但其特异度为 8%（72/955;阈值 0.99）。受试者工作曲线显示可信区间为 95%时曲线下面积为 0.795(0.751-0.839)。

结论：对于院外心跳骤停的患者，该参数不能准确预测院内的生存。

（姚敏敏译 薛张纲校）

BACKGROUND: We created a prediction model to be used in cardiopulmonary resuscitation (CPR) attempts as a decision tool to omit futile CPR attempts and to save resources.

METHODS: In this post hoc analysis, we assessed predictive parameters for neurological recovery after successful CPR. The original study was designed as a blinded, randomized, prospective, controlled, multicenter clinical trial.

RESULTS: We identified 1166 prehospital cardiac arrest patients being treated with advanced cardiac life support. Seven hundred eighty-six of 1166 patients (67.4%) died at the scene and 380 of 1166 (32.6%) were brought to the hospital. Two hundred sixty-five of 1166 patients (22.7%) died in the hospital. One hundred fifteen of 1166 (9.8%) were discharged from the hospital and 92 of the 115 patients (80%) could be followed-up. Good cerebral performance was regained by 54% of discharged patients (50 of 92 patients). In 46% of patients (42/92), unconsciousness or severe disability remained. Ventricular fibrillation was more likely to have occurred in patients with good neurological recovery (42/50 = 84.0%), whereas asystole was more likely in patients with

poor neurological recovery (9/42 = 21.4%). A score was developed to predict the probability of death using logistic regression analysis. Predicting death in the hospital revealed a sensitivity of 99.8% (953/955), but only a specificity of 2.9% (3/104; threshold 0.5). Predicting survival until discharge from the hospital revealed a sensitivity of 99% (103/104), but only a specificity of 8% (72/955; threshold 0.99). A receiver operating characteristic curve yielded an area under the curve of 0.795 (0.751-0.839) at a confidence interval of 95%.
CONCLUSION: For out-of-hospital patients with cardiac arrest, parameters documented in the field did not allow accurate prediction of hospital survival.

在择期剖宫产患者中比较预先加用胶体和腰麻同时加用胶体的效果：一项随机试验

A Randomized Trial Comparing Colloid Preload to Coload During Spinal Anesthesia for Elective Cesarean Delivery

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背景：剖宫产手术中腰麻后低血压十分常见。先前的研究显示同时加用晶体液（在鞘内注射同时快速补液）比常规的腰麻前预先加入晶体液（在鞘内注射前补液）在预防低血压方面更为优越。预先加用胶体能获得中心血容量的持久增加。我们假设：和晶体液比较，预先加用胶体可能比腰麻同时加胶体在减少腰麻导致的低血压发生率上更为有效。
方法：在这个双盲研究中，178 个病人被随机分配，分别在腰麻前 15-20 分钟内给予 500ml 羟乙基淀粉（n=90）或者在脑脊液注药的同时快速补入等量液体（n=88）。当收缩压下降少于患者本身血压的 80%和小于 100mm Hg，或者较轻微低血压伴随恶心呕吐或眩晕时给予血管加压素（麻黄碱或苯肾上腺素）。最主要的结果是低血压的发生率（定义为至少一个剂量血管加压素的使用）。
结果：低血压的发生率在两组中间没有明显区别（预先加胶体组 68%，同时加胶体组 75%，两者之间区别的 95%置信区间为－6%－20%; P = 0.28），麻黄碱和苯肾上腺素的用量，和血管加压素单位剂量的数量。严重低血压（收缩压小于 80 mm Hg）的发生率为 16%在预先加胶体组，22%在同时加胶体组(P = 0.30)。两者之间在恶心呕吐发生率没有区别。
结论：女性患者中在腰麻前给与胶体和在腰麻同时给与胶体，低血压的发生率没有区别。两种方式作为一项单独的干预措施在预防低血压方面都没有效果。
（俞佳译 薛张纲校）
BACKGROUND: Hypotension after spinal anesthesia for cesarean delivery is common. Previous studies have demonstrated that a
crystalloid fluid "coload" (rapid administration of a fluid bolus starting at the time of intrathecal injection) is superior to the conventional crystalloid preload (fluid

administered before the intrathecal injection) for preventing hypotension. Colloid preload provides a sustained increase in central blood volume. We hypothesized that, in contrast to crystalloid, a colloid preload may be more effective than colloid coload for reducing the incidence of spinal anesthesia-induced hypotension.
METHODS: In this double-blind study, 178 patients were randomly assigned to receive a preload of 500 mL of hydroxyethyl starch over a period of 15–20 min before initiation of spinal anesthesia (n = 90) or an identical fluid bolus of hydroxyethyl starch starting at the time of identification of cerebrospinal fluid (n = 88). Vasopressors (ephedrine or phenylephrine) were administered if systolic arterial blood pressure decreased less than 80% of the baseline pressure and <100 mm Hg, or with smaller decreases in blood pressure if accompanied by nausea, vomiting, or dizziness. The primary outcome was the incidence of hypotension (defined as the administration of at least one dose of vasopressor).
RESULTS: There was no significant difference between the groups in the incidence of hypotension (68% in preload group and 75% in coload group, 95% confidence interval of difference –6%–20%; P = 0.28), doses of ephedrine and phenylephrine, and number of vasopressor unit doses. The incidence of severe hypotension (systolic blood pressure <80 mm Hg) was 16% in the preload group and 22% in the coload group (P = 0.30). There were no differences in the incidence of nausea and/or vomiting, or neonatal outcome between the groups.
CONCLUSION: There was no difference in the incidence of hypotension in women who received colloid administration before the initiation of spinal anesthesia compared with at the time of initiation of anesthesia. Both modalities are inefficient as single interventions to prevent hypotension.

兔子中七氟醚预处理诱导通过细胞外信号调节激酶的激活介导快速缺血耐受来对抗脊髓缺血再灌注损伤、

Sevoflurane Preconditioning Induces Rapid Ischemic Tolerance Against Spinal Cord Ischemia/Reperfusion Through Activation of Extracellular Signal-Regulated Kinase in Rabbits

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背景：七氟醚预处理对脊髓缺血/再灌注（I / R）保护作用是不清楚的。我们设计这个研究，调查是否七氟醚预处理可在短暂兔脊髓缺血模型中诱导快速缺血耐受，以及细胞外信号调节激酶（ERK）如何作用的。
方法：新西兰白色雄性家兔随机分为三组来测试七氟醚预处理是否诱导快速缺血耐受。七氟醚组动物预处理吸入 3.7％七氟醚（1.0 最低肺泡麻醉浓度）混合 96％氧气 30 分钟，而氧气组动物仅控制性吸入 96％氧气 30 分钟。假手术组接受了同样的麻醉和手术的准备，但没有预处理或脊髓的缺血/再灌注。评估 ERK 的激活在七氟醚预处理中作用，兔随机分为 4 组。 U0126， ERK 的抑制剂，于预处理前 20 分钟注入 U012+ O2 组和 U0126 +七氟醚组。同时在媒介物+ O2 组和媒介物+七氟醚组中静脉注入二甲基亚砜。预处理后 1 小时，动物通过腹主动脉阻断来导致脊髓

的 I / R。48 小时后所有动物通过再修改 Tarlov 评分标准，以及脊髓节段（腰）的组织病理学检查，TUNEL 染色和免疫印迹磷- ERK1 / 2 来评估。
结果：七氟醚组动物比氧组有更高的神经系统得分和更多的正常运动神经元（各个比较均 P<0.01）。与媒介物+七氟醚组相比，U0126 +七氟醚组神经检查结果更差，有功能的神经元更少，神经元凋亡更多，更显著的 ERK1 / 2 的磷酸化的降低（各个比较均 P<0.01）。媒介物+ O2 组，U0126 + O2 组和 U0126 +七氟醚组之间没有显著差异。
结论：本研究表明，七氟醚预处理在实验兔中诱导快速耐受脊髓缺血/再灌注且耐受可能是通过 ERK 的激活介导的。这些数据表明，七氟醚预处理可能为保护围手术期脊髓缺血/再灌注提供新的处理的方法。
（张玥琪译，薛张纲校）

BACKGROUND: The protective effect of sevoflurane preconditioning against spinal cord ischemia/reperfusion (I/R) is unclear. We designed this study to investigate whether sevoflurane preconditioning could induce rapid ischemic tolerance to the spinal cord in a rabbit model of transient spinal cord ischemia and how the role of extracellular signal-regulated kinase (ERK) is involved.
METHODS: To test whether preconditioning with sevoflurane induces rapid ischemic tolerance, New Zealand White male rabbits were randomly assigned to three groups. Animals in the Sev group received preconditioning with 3.7% sevoflurane (1.0 minimum alveolar anesthetic concentration) in 96% oxygen for 30 min, whereas animals in the O_2 group serving as controls inhaled only 96% oxygen for 30 min. The Sham group received the same anesthesia and surgical preparation but no preconditioning or spinal cord I/R. To evaluate the role of ERK activation in sevoflurane preconditioning, rabbits were randomly assigned to four groups. U0126, an ERK inhibitor, was administered IV 20 min before the beginning of preconditioning in the U0126 + O_2 and U0126 + Sev groups. Dimethylsulfoxide was administered IV at the same time in the vehicle + O_2 and vehicle + Sev groups. At 1 h after preconditioning, the animals were subjected to spinal cord I/R induced by infrarenal aorta occlusion. All animals were assessed at 48 h after reperfusion with modified Tarlov criteria, and the spinal cord segments (L5) were harvested for histopathological examination, TUNEL staining, and Western blot of phosphor-ERK1/2.
RESULTS: The animals in the Sev group had higher neurological scores and more normal motor neurons than those in the O_2 group ($P < 0.01$ for each comparison). Compared with vehicle + Sev group, the U0126 + Sev group had worse neurological outcomes, fewer viable neurons, more apoptotic neurons, and significantly decreased ERK1/2 phosphorylation ($P \leq 0.01$ for each comparison). There were no significant differences in the outcomes among vehicle + O_2, U0126 + O_2, and U0126 + Sev groups.
CONCLUSIONS: This study demonstrates that sevoflurane preconditioning induces rapid tolerance to spinal cord I/R in rabbits, and the tolerance is possibly mediated through the activation of ERK. These data suggest that sevoflurane preconditioning might provide a new practical method for protecting perioperative spinal cord I/R.

弗罗因德式完全佐剂诱发椎间盘炎的椎间盘源性下腰痛的动物模型

Complete Freund's adjuvant-induced intervertebral discitis as an animal model for discogenic low back pain.

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背景： 虽然建立了大量的椎间盘(IVD)退变相关的下腰痛的动物模型，但是这些数据资料不足以对疼痛得出明确的结论。此研究的目的在于确定用弗罗因德式完全佐剂(CFA)注射入大鼠脊柱模拟人类椎间盘性疼痛的动物模型的可靠程度.
方法:向成年大鼠 L5-6 椎间盘内注射 10 微升 CFA 后,应用行为学,组织学和生物化学方法研究椎间盘退变和疼痛的关系.通过分析一系列组织学变化观察椎间盘退行性变。应用免疫组化，PCR 实时聚合酶链反应测定降钙素基因相关多肽(CGRP)、前列腺素 E(PGE)和诱导型一氧化氮合酶(iNOS)，作为疼痛发生的依据。此外椎间盘内 CGRP 免疫活性作为神经内生长的间接证据。
结果：术后 7 周 CFA 组中可观察到后续退变反应加强。组织学分析发现椎间盘退变但不伴随周围结构如神经根的破坏。椎间盘内注射 CFA 后 CGRP-免疫染色分析发现双侧后角和椎间盘免疫反应呈阳性。2 周和 4 周后脊髓背根神经节（DRG）中 CGRPmRNA 表达增加。2 周后 PGE 和 iNOS mRNA 的表达也显著增加。CGRP 在异常疼痛的大鼠体内表达显著高于在非异常疼痛大鼠体内的表达。
结论：椎间盘内 CFA 注射所诱导的慢性椎间盘退变导致的异常疼痛可能与疼痛性行为和疼痛介质表达相关。在动物模型中 CGRP，PEG 和 iNOS 表达增加也与椎间盘和神经通路的信号传导相关。本动物模型对于今后研究脊柱相关疼痛的病理生理及新的治疗方法有意义。
（张钊译 薛张纲校）
BACKGROUND: Although numerous animal models for low back pain associated with intervertebral disk (IVD) degeneration have been proposed, insufficient data have been provided to make any conclusions regarding pain. Our aim in this study was to determine the reliability of complete Freund's adjuvant (CFA) injection into the rat spine as an animal model representing human discogenic pain.
METHODS: We studied IVD degenerative changes with pain development after a 10-microL CFA injection into the L5-6 IVD of adult rats using behavioral, histologic, and biochemical studies. Serial histologic changes were analyzed to detect degenerative changes. Expression of calcitonin gene-related peptide (CGRP), prostaglandin E (PGE), and inducible nitric oxide synthase (iNOS) were determined using immunohistochemistry or real-time polymerase chain reaction as support data for pain development. In addition, CGRP immunoreactivity (ir) at the IVD was considered indirect evidence of neural ingrowth into the IVD.
RESULTS: There was a significant increase of the hindpaw withdrawal response in the CFA group until 7 wk postoperatively ($P < 0.05$). Histologic analyses revealed progressive degenerative changes of the disks without any damage in adjacent structures, including nerve roots. In the CGRP-ir staining study, the bilateral dorsal horns and IVD had positive ir after intradiscal CFA injection. CGRP mRNA expression was increased in the dorsal root ganglion (DRG) at 2 and 4 wk, whereas PGE and iNOS mRNAs were markedly increased at 2 wk. The increment of CGRP expression was higher in allodynic rats compared with nonallodynic rats.

CONCLUSION: Intradiscal CFA injection led to chronic disk degeneration with allodynia, which was suggested by pain behavior and expression of pain-related mediators. The increment of CGRP, PGE, and iNOS also suggest pain-related signal processing between the IVD and the neural pathway in this animal model. This animal model may be useful for future research related to the pathophysiology and development of novel treatment for spine-related pain.

大鼠鞘内注射吗啡与马普替林的协同效应

The Synergistic Interaction Between Morphine and Maprotiline After Intrathecal Injection in Rats

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背景:抗抑郁药物具有抑制去甲肾上腺素和/或 5-羟色胺重摄取的作用，常与阿片内联合用于治疗慢性疼痛。其增加镇痛效果的机制尚未明确。我们使用大鼠热撤离试验比较鞘内注射吗啡与非选择性抗抑郁药阿米替林或选择性抗抑郁药马普替林、西他罗仑联合使用时减弱伤害感受的效应。我们也观察这些药物间相互作用的可能机制。

方法：用七氟烷麻醉雄性 Wistar 大鼠，并分别向鞘内注射吗啡、抗抑郁药或盐水。给药前后用热撤离试验评估减弱伤害感受效应。用最大可能效应百分比(MPE)表示撤离反应发生时间。为了研究反应机制，所有动物均用非选择性 α 2 受体阻滞剂育亨宾和非选择性阿片类阻滞剂纳洛酮预处理。同时用等辐射分析法评估固定比例注射马普替林和吗啡的药理学相互作用。

结果：单一鞘内注射吗啡 2 μg、阿米替林 125 μg、西他罗仑 144 μg、马普替林 1.25 μg 分别产生 51.6% ± 8.9%, 10.3% ± 3.2%, 33.8% ± 5.2%和 48.5% ± 9.2% 的 MPE。吗啡与阿米替林合用时减弱伤害感受效应增强至 91.3% ± 4.6% MPE，与马普替林合用增强至 86.9% ± 9.2% MPE，而与西他罗仑合用无增强效果(40.6% ± 4.6% MPE)。与马普替林联合使用时吗啡的减弱伤害感受时间增加四倍，由 120 分钟增加至 480 分钟，这一效应可被α-2 受体抑制剂育亨宾和阿片类 μ 受体拮抗剂纳络酮预处理逆转。等辐射分析法证明了吗啡和马普替林的协调作用。

结论：选择性去甲肾上腺素重摄取抑制剂通过α-2 和阿片类受体能明显增加吗啡减弱伤害性感受的强度和时间。选择性 5-羟色胺抑制剂西他罗仑与吗啡无此相互作用。

（朱兰芳译 薛张纲校）

BACKGROUND: Antidepressant drugs act as potent inhibitors of norepinephrine and/or serotonin reuptake and are widely used with opioids for the treatment of chronic pain. The mechanism of this increased analgesic action is unclear. We compared the anti nociceptive effects of the intrathecal administration of morphine with that of a nonselective (amitriptyline) or selective (maprotiline or citalopram) antidepressant drug

using the thermal withdrawal test in rats. We also investigated the possible mechanisms involved in the interactions of these drugs.
METHODS: Male Wistar rats were anesthetized with sevoflurane and administered morphine and antidepressant drugs, or saline, through intrathecal injection. The antinociceptive effect was evaluated using the thermal withdrawal test before and after drug administration. The time for the withdrawal reaction was expressed as percentage of maximum possible effect (MPE). Animals were also pretreated with yohimbine (a nonselective alpha2-adrenergic antagonist) and naloxone (a nonselective opioid antagonist) for mechanism of action studies. Pharmacologic interaction was evaluated using isobolographic analysis of simultaneous administration of fixed proportions of maprotiline and morphine.
RESULTS: Single intrathecal administration of morphine (2 μg), amitriptiline (125 μg), citalopram (144 μg), and maprotiline (1.25 μg) produced 51.6% ± 8.9%, 10.3% ± 3.2%, 33.8% ± 5.2%, and 48.5% ± 9.2% MPE, respectively. The antinociceptive effect of morphine was increased when combined with amitriptyline (91.3% ± 4.6% MPE) and maprotiline (86.9% ± 9.2% MPE) but not with citalopram (40.6% ± 4.6% MPE). Coadministration of maprotiline increased the antinociceptive duration of morphine by 4-fold (from 120 to 480 min), which was reversed by pretreatment with the α_2-adrenoceptor inhibitor, yohimbine, and the mu-type opioid receptor antagonist, naloxone. Isobolographic analysis demonstrated a synergistic interaction between morphine and maprotiline.
CONCLUSIONS: Selective norepinephrine reuptake inhibitors can significantly increase the intensity and duration of morphine antinociceptive activity via both α_2-adrenergic and opioid receptors. This interaction was not observed with the selective serotonin inhibitor, citalopram.

利多卡因中加入对乙酰氨基酚行静脉区域阻滞的镇痛效果

The Analgesic Effect of Paracetamol When Added to Lidocaine for Intravenous Regional Anesthesia

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背景：在这项研究中，我们评估了在利多卡因中加入对乙酰氨基酚用于静脉区域阻滞时，感觉及运动阻滞的起效时间，止血带疼痛和术后镇痛效果。
方法：60 名行手部手术的病人随机、盲法分为 3 组。三组病人行静脉区域镇痛，均给予利多卡因 3 mg/kg ，用生理盐水稀释至 40ml。组 1 静脉给予利多卡因加生理盐水；组 2 给予利多卡因加 300mg 对乙酰氨基酚，生理盐水稀释至 40ml；组 3 给予利多卡因加对乙酰氨基酚 300mg。分别评估了手术期间感觉或运动阻滞起效时间、止血带疼痛和镇痛药物使用情况。止血带放气后 1，2，4，6，12 和 24 小时

进行 VAS 评分，第一次使用镇痛药的时间，最初 24 小时镇痛药物使用的总量及副作用的情况。

结果：第二组运动阻滞起效时间较短，运动感觉恢复时间较长（$P < 0.05$）。组 2 手术期间 20, 30 和 40 分钟 VAS 评分较低($P < 0.05$)。手术期间芬太尼使用总量分别为 78 ± 12、58 ± 14、78 ± 11 μg；因止血带疼痛需要使用芬太尼的病人数量分别为 13 人、3 人和 9 人，组 2 人数明显较少($P < 0.05$)。组 2 术后使用芬太尼的时间间隔较长（分别为 15 ± 6, 25 ± 5, 15 ± 4 分钟）($P < 0.05$)。组 2 手术麻醉的质量较高($P < 0.05$)。术后 VAS 评分和开始使用镇痛药物的时间在各组中相似；组 2 使用双氯芬酸钠的总量较少($P < 0.05$)。

结论：静脉区域镇痛时，利多卡因中加入对乙酰氨基酚可以降低止血带疼痛，提高麻醉的质量，减少术后镇痛药物使用的量。

（陈珺珺译 薛张纲校）

BACKGROUND: In this study, we evaluated the effect of paracetamol on sensory and motor block onset time, tourniquet pain, and postoperative analgesia, when added to lidocaine in IV regional anesthesia (IVRA).

METHODS: Sixty patients undergoing hand surgery were randomly and blindly divided into three groups. All groups received IVRA lidocaine (3 mg/kg) diluted with saline to a total volume of 40 mL. Group 1 received IVRA lidocaine plus IV saline, Group 2 received IVRA lidocaine and paracetamol (300 mg) admixture plus IV saline, and Group 3 received IVRA lidocaine plus IV paracetamol (300 mg). Sensory and motor block onset time, tourniquet pain, and analgesic use were assessed during operation. After tourniquet deflation, visual analog scale (VAS) scores at 1, 2, 4, 6, 12, and 24 h, the time to first analgesic requirement, total analgesic consumption in first 24 h, and side effects were noted.

RESULTS: Onset of motor block was shorter and recovery of motor and sensory block was significantly longer in Group 2 ($P < 0.05$). Intraoperative VAS scores at intraoperative 20, 30, and 40 min were significantly lower in Group 2 ($P < 0.05$). Intraoperative fentanyl consumption (78 ± 12, 58 ± 14, 78 ± 11 μg, respectively) and the number of patients who required fentanyl for tourniquet pain (13 patients, 3 patients, 9 patients, respectively) were significantly less in Group 2 ($P < 0.05$). Time to postoperative fentanyl administration was also prolonged (15 ± 6, 25 ± 5, 15 ± 4 min, respectively) in Group 2 ($P < 0.05$). The quality of surgical anesthesia was better in Group 2 ($P < 0.05$). Postoperative VAS scores and time of initial analgesic requirement were similar among groups; however, the total amount of diclophenac use was less in Group 2 ($P < 0.05$).

CONCLUSION: The addition of paracetamol during IVRA with lidocaine decreased tourniquet pain, increased anesthesia quality, and decreased postoperative analgesic consumption.

体外循环对脑的微栓子数量和冠脉搭桥术后认知障碍发生率的影响

The Effects of Cardiopulmonary Bypass on the Number of Cerebral Microemboli and the Incidence of Cognitive Dysfunction After Coronary Artery Bypass Graft Surgery

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背景：术后认知障碍（POCD）可能是冠脉搭桥术（CABG）后使患者虚弱的并发症。体外循环（CPB）中的脑微栓子被认为是 POCD 一个重要发病因素。在这个研究中，我们验证在中国人群中不进行 CPB 的手术是否可以减少脑栓子的数量和 CABG 术后 POCD 的发生率。
方法：227 例患者被选入这个前瞻性队列研究。59 例患者进行了 CPB 下的 CABG 手术，168 例患者在不运用 CPB 下进行手术。脑微栓子通过双侧大脑中动脉经颅多普勒超声检查来持续检测。一套神经心理学测试，包括七个试验九个方面，在术前、术后 1 周和术后 3 月进行。POCD 用国际 POCD1 研究的定义来进行定义。
结果：在 CPB 下手术患者的脑微栓子总数量的中位数为 430（范围：155－2088），不进行 CPB 的患者为 2（0－66）(P < 0.001)。术后 1 周(55.2%或 32/58[95%可信区间: 41.5%–68.3%]对 47.0%或 78/166[39.2%–54.9%], *P* = 0.283)和 3 月的 POCD 发生率(6.4%或 3/47 [1.3%–17.5%] 对 13.1%或 16/122[7.7%–20.4%], *P* = 0.214)，进行 CPB 和不进行 CPB 手术的患者之间没有差异。年龄的增加和较短的术后住院时间与术后 1 周的认知障碍独立相关。年龄增加和有糖尿病史与术后 3 月认知障碍独立相关。CPB 或脑微栓子与 POCD 的发生没有显著的相关性。
结论：在中国人群中，CABG 手术中不使用 CPB 能显著减少脑微栓子的数量，但不能减少术后 1 周和 3 月 POCD 的发生。CPB 和脑微栓子都不是与 POCD 独立相关的风险因素。
（唐亮 译 马皓琳 李士通 校）

BACKGROUND: Postoperative cognitive dysfunction (POCD) can be a debilitating complication after coronary artery bypass graft (CABG) surgery. Cerebral microemboli during cardiopulmonary bypass (CPB) are believed to be an important etiologic factor of POCD. In this study, we examined whether avoidance of CPB with "off-pump" surgery reduces the number of cerebral microemboli and the incidence of POCD after CABG surgery in Chinese population.
METHODS: Two hundred twenty-seven patients were enrolled in this prospective cohort study. Fifty-nine patients underwent CABG surgery with CPB and 168 underwent off-pump surgery. Cerebral microemboli were measured continuously with bilateral transcranial Doppler ultrasonography of the middle cerebral arteries. A neuropsychological test battery that included seven tests with nine subscales was administered at baseline, as well as at 1 wk and 3 mo after surgery. POCD was defined using the international study of POCD1 definition.
RESULTS: The median total number of cerebral microemboli for the case was 430 (range: 155–2088) in patients undergoing surgery with CPB and 2 (0–66) in the off-pump patients (*P* < 0.001). There were no differences in the incidence of POCD between the patients having surgery with or without CPB either at 1 wk (55.2% or 32 of 58 patients [95% confidence interval: 41.5%–68.3%] vs 47.0% or 78 of 166 patients [39.2%–54.9%], *P* = 0.283) or 3 mo (6.4% or 3 of 47 patients [1.3%–17.5%] vs 13.1% or 16 of 122 of

patients [7.7%–20.4%], $P = 0.214$) after surgery. Increasing age and shorter duration of postoperative hospital stay were independently associated with cognitive dysfunction at 1 wk after surgery. Increasing age and a history of diabetes mellitus were independently associated with cognitive dysfunction 3 mo after surgery. CPB or cerebral microemboli were not significantly related to the occurrence of POCD.
CONCLUSIONS: In Chinese population, avoidance of CPB during CABG surgery significantly decreased the number of cerebral microemboli, but it did not decrease the incidence of POCD at either 1 wk or 3 mo after CABG. Neither CPB nor cerebral microemboli was independently associated with the risk of POCD.

恶性高热、共存失调和酶病：风险和管理选择

Malignant Hyperthermia, Coexisting Disorders, and Enzymopathies: Risks and Management Options

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已观察到患多种综合症、酶病和两者共存的病人出现与诊断恶性高热一致的临床症状和异常实验室试验，因此提高了有相同诊断的其他病人的病因相关的可能性和已增高的围术期风险。在本综述中，我们检查了被其他医生认定为潜在易患恶性高热患者的可得到的已发表系列、病例报道和挛缩试验的结果。对多数情况下，与恶性高热易感性有病因相关的证据是很少的。本综述总结了当支持或不支持相关性的证据不确定时的临床管理的建议。
（王宏翻译，马皓琳，李士通校正）
Clinical episodes and abnormal laboratory tests compatible with a diagnosis of malignant hyperthermia have been observed in patients with a diversity of syndromes, enzymopathies, and coexisting disorders thereby raising the likelihood of causal associations and heightened perioperative risk in others carrying a shared diagnosis. In the present review, we survey available published series, case reports, and the results of contracture testing in patients identified by others to be potentially predisposed to malignant hyperthermia. For most conditions, evidence for a causal relationship with malignant hyperthermia susceptibility is weak. The review concludes with suggestions for clinical management when evidence for or against an association is uncertain.

神经肌肉阻滞不同程度地影响近最低肺泡有效浓度麻醉下的体动抑制和皮层活性

Neuromuscular Block Differentially Affects Immobility and Cortical Activation at Near–Minimum Alveolar Concentration Anesthesia

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背景：麻醉诱导的体动抑制和皮层活动的抑制是由解剖上分开但相互作用的中枢神经系统区域所控制的。因此，与抑制伤害刺激引起的体动反应相比，需要更大浓度的吸入麻醉药以抑制皮层的活动。我们观察了在近最低肺泡有效浓度的地氟烷麻醉过程中有伤害性刺激时，神经肌肉阻滞（NMB）产生的传入输入减弱对体动反应和用脑电双频指数（BIS）评估的皮层活动的影响。

方法：在 24 名健康的志愿者中，用上下调节的方法和单独的前臂方法评价 NMB 对用于体动抑制的地氟烷有效呼气末肺泡浓度($EtDes_{50}$ 或 $MAC_{tetanus}$) 中位数的影响。 每个志愿者以随机次序连续地注射盐水、美维库铵和司可林，而在注射同样药物的前一个志愿者的体动反应的基础上来测定用不同药物时的 EtDes 浓度。用非线性混合反应模型来评估 NMB 对基础状态下和有害刺激后 BIS 与 EtDes 浓度的对比关系的影响，而前额的 EMG(EMG_{BIS})对 BIS 的影响同样也作为模型中的一个变量。同时比较了应用不同药物时有害刺激引起的心血管反应。

结果：司可林和美维库铵显著减少 $MAC_{tetanus}$(95%可信区间)。其中生理盐水组为 5.00% (4.85%–5.13%),而司可林和美维库铵分别为 4.05% (3.81%–4.29%) 和 3.84% (3.60%–4.08%)。无论应用何种药物，伤害性刺激均明显地增加了 BIS 的反应，尽管是最低限度的。司可林增加 BIS 值而不依赖于其对 EMG_{BIS}的影响。注射司可林增加了心血管系统的活性。有趣的是，尽管美维库铵减弱了心血管对伤害刺激的反应，用 BIS 测定的皮层反应并没有变化。

结论：在接近于 MAC 的麻醉期间，司可林和美维库铵增加了对体动反应的抑制。所有用药均会引起伤害性刺激产生的小但却显著的 BIS 值增加。而司可林增加 BIS 值并不依赖于伤害性刺激或 EMG_{BIS}。美维库铵抑制了对伤害性刺激的自主神经反应。

（黄丽娜 译 马皓琳 李士通 校）

BACKGROUND: Anesthesia-induced immobility and cortical suppression are governed by anatomically separate, but interacting, areas of the central nervous system. Consequently, larger volatile anesthetic concentrations are required to suppress cortical activation than to abolish movement in response to noxious stimulation. We examined the effect of decreased afferent input, as produced by neuromuscular block (NMB), on immobility and cortical activation, as measured by Bispectral index (BIS) of the electrocardiogram, in the presence of noxious stimulation during approximately minimum alveolar concentrations (MACs) of desflurane anesthesia.

METHODS: The effect of NMB on the median effective end-tidal concentration of desflurane ($EtDes_{50}$, or $MAC_{tetanus}$) for immobility was estimated using the up-and-down method and isolated forearm technique in 24 healthy volunteers. Each volunteer sequentially received saline, mivacurium, and succinylcholine in a randomized order, while EtDes concentration during each of the treatments was determined based on the

movement response of the previous volunteer on the same treatment. Nonlinear mixed-effects modeling was used to evaluate the effect of NMB on BIS versus EtDes concentration relationship at baseline and after noxious stimulation, while the frontal electromyogram (EMG_{BIS}) effect on BIS was also modeled as a covariate. Cardiovascular responses to noxious stimulation were compared across treatments.
RESULTS: Succinylcholine and mivacurium significantly reduced $MAC_{tetanus}$ (95% confidence interval) from 5.00% (4.85%–5.13%), during saline, to 4.05% (3.81%–4.29%) and 3.84% (3.60%–4.08%), respectively. Noxious stimulation significantly, although minimally, increased BIS response during all treatments. Succinylcholine increased BIS independently of an effect on EMG_{BIS}. Succinylcholine administration increased cardiovascular activity. Interestingly, although cardiovascular reaction to the noxious event was ablated by mivacurium, cortical response, as determined by BIS, was retained.
CONCLUSIONS: Both succinylcholine and mivacurium enhanced immobility during near-MAC anesthesia. All treatments were associated with a small, although significant, BIS increase in response to noxious stimulation, whereas succinylcholine increased BIS independently of noxious stimulation or EMG_{BIS}. Mivacurium suppressed autonomic response to a noxious event.

短暂接触七氟烷后，干细胞样人内皮祖细胞显示集落形成能力增强：吸入麻醉药对血管生成细胞的预处理

Stem Cell-Like Human Endothelial Progenitors Show Enhanced Colony-Forming Capacity After Brief Sevoflurane Exposure: Preconditioning of Angiogenic Cells by Volatile Anesthetics

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背景：内皮祖细胞在组织修复中起关键作用，因此在“再生医学”中被用于细胞的替代治疗。我们检验麻醉药七氟烷是否会调制这些血管生成细胞的生长或动员。
方法：在离体模型中，从健康捐献者的外周血中分离出来的单核细胞用七氟烷预处理（2 vol%，30 min 3 次，间隔予 30 min 空气）。9 天后在培养中测定集落形成单位，并与同期配对的未处理对照进行比较。使用磁性细胞分选法，从人脐带血中富集 CD133+/CD34+的内皮祖细胞，并用实时逆转录多聚酶链式反应对七氟烷处理或未处理细胞内血管内皮生长因子（VEGF）、VEGFR2（KDR）、粒细胞集落刺激因子（G-CSF）、STAT3、c-kit 以及 CXCR4 的表达进行测定。在一个采用交叉试验设计的志愿者研究中，我们使用外周血样流式细胞仪，检验吸入七氟烷（呼气

末浓度<1 vol%）是否动员内皮祖细胞从骨髓生态位区进入循环。同时测定血浆中 VEGF 和 G-CSF 的水平。
结果：单核细胞体外接触七氟烷增强 CD133+/CD34+脐带血细胞的集落形成能力并增加 VEGF mRNA 水平($P = 0.017$)。健康志愿者吸入七氟烷并不改变循环中 CD133+/CD34+ 或 KDR+/CD34+内皮祖细胞的数量，但增加了集落形成单位的数量($P = 0.034$)，而血浆中 VEGF 和 G-CSF 的水平保持不变。
结论：七氟烷预处理促进了干细胞样人类内皮祖细胞的生长和增殖，因此可能被用于促进围手术期血管愈合并支持细胞替代治疗。
（黄施伟 译，马皓琳 李士通 校）

BACKGROUND: Endothelial progenitor cells play a pivotal role in tissue repair, and thus are used for cell replacement therapies in "regenerative medicine." We tested whether the anesthetic sevoflurane would modulate growth or mobilization of these angiogenic cells.
METHODS: In an *in vitro* model, mononuclear cells isolated from peripheral blood of healthy donors were preconditioned with sevoflurane (3 times 30 min at 2 vol% interspersed by 30 min of air). Colony-forming units were determined after 9 days in culture and compared with time-matched untreated control. Using magnetic cell sorting, CD133+/CD34+ endothelial progenitors were enriched from human umbilical cord blood, and vascular endothelial growth factor (VEGF), VEGFR2 (KDR), granulocyte colony-stimulating factor (G-CSF), STAT3, c-kit, and CXCR4 expressions were determined in sevoflurane-treated and untreated cells by real-time reverse transcriptase polymerase chain reaction. In a volunteer study with crossover design, we tested whether sevoflurane inhalation (<1 vol% end-tidal concentration) would mobilize endothelial progenitor cells from the bone marrow niche into the circulation using flow cytometry of peripheral blood samples. VEGF and G-CSF plasma levels were also measured.
RESULTS: *In vitro* sevoflurane exposure of mononuclear cells enhanced colony-forming capacity and increased VEGF mRNA levels in CD133+/CD34+ cord blood cells ($P = 0.017$). Sevoflurane inhalation in healthy volunteers did not alter the number of CD133+/CD34+ or KDR+/CD34+ endothelial progenitors in the circulation, but increased the number of colony-forming units ($P = 0.034$), whereas VEGF and G-CSF plasma levels remained unchanged.
CONCLUSIONS: Sevoflurane preconditioning promotes growth and proliferation of stem cell-like human endothelial progenitors. Hence, it may be used to promote perioperative vascular healing and to support cell replacement therapies.

多输注治疗期间多路输注装置对体外给药的影响

Impact of Multiaccess Infusion Devices on *In Vitro* Drug Delivery During Multi-Infusion Therapy

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背景：多路输注装置能够同时输注多种药物，但也可能引起各投药通路相互干扰，其原因是有效药物的给药速率的个体差异较大。我们在本研究中试图阐明在多输注治疗期间多路输注装置属性（死腔容量和抗反流阀[ARV]）对投药的影响。
方法：对长度、死腔量以及有无 ARV 方面不同的输注装置进行评估。通过不同通路点同时输注 3 种药物，并通过紫外线分光光度计分析流出液来得到其药物浓度。评估以下参数来比较不同的输注配置：(1)每单位时间输入病人体内的药物量、(2)输注稳态（质量流速平台）期间每单位时间输入病人体内的平均药量及(3) 流量变化效能——实验瞬时质量流率曲线下面积与对应的理论瞬时质量流率曲线下面积的比值。
结果：无论流速如何变化，与高死腔量的输注装置（死腔量等于 6.16 mL 时流量变化效能 5.6% ± 8.2%）相比，低死腔量的输注装置明显具有较高的流量变化效能（死腔量等于 0.046 mL 时开始输注后 5 min 为 53.0% ± 15.4%）。即使存在较大死腔量，具有 ARV 的输注装置明显抬高质量流率平台（从没有 ARV 时理论平台的 92.4% 增加至有 ARV 时的 99.3%）。
结论：多输注治疗引起投药干扰（输注滞后时间，返流，单次注射量）。采用非常低死腔量和具有 ARV 的输注装置可以降低这种干扰。
(江继宏 译 马皓琳 李士通 校）

BACKGROUND: Multiaccess infusion sets allow multiple simultaneous infusions but may induce interference in drug delivery resulting from large variations in the delivery rate of potent drugs. In this study, we sought to understand the influence of multiaccess infusion device properties (dead space volume and antireflux valve [ARV]) on drug delivery during multi-infusion therapy.
METHODS: Infusion sets differing in length, dead space volume, and presence of an ARV were assessed. Three drugs were infused simultaneously through different access points, and their concentrations were obtained using UV spectrophotometric analysis of the effluent. Different infusion configurations were compared by assessing (1) the amount of drug delivered to the patient per unit of time, (2) the mean amount of drug delivered to the patient per unit of time during the steady-state infusion (mass flow rate plateau), and (3) flow change efficiency calculated from the ratio of the area under the experimental instant mass flow rate curve to the area corresponding to theoretical instant mass flow rate curve.
RESULTS: Infusion sets with lower dead space volumes offered significantly higher flow change efficiency (53.0% ± 15.4% with a dead space volume equal to 0.046 mL 5 min after the start of infusion) than infusion sets with higher dead space volume (5.6% ± 8.2% with a dead space volume equal to 6.16 mL), whatever the flow rate changes. Even in case of large dead space volumes, the presence of an ARV significantly increased the mass flow rate plateau (from 92.4% to 99.3% of the theoretical plateau without and with the presence of an ARV, respectively).

CONCLUSIONS: Multi-infusion therapy induces perturbation in drug delivery. These perturbations (lag time, backflow, and bolus) could be reduced by using infusion sets including very low dead space volume and an ARV.

核心肌病与恶性高热的风险

Core Myopathies and Risk of Malignant Hyperthermia

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在本文中我们分析了核心肌病，核心肌病与恶性高热之间的联系已经有人提出了。我们讨论了核心肌病的临床特征、基础的遗传缺陷、对细胞钙代谢的后续影响以及对恶性高热触发的离体肌反应。我们详细描述了中央核疾病、多小核疾病和线形体小枝肌病。我们还依据疾病侵犯的蛋白质对疾病加以分类，并分析了恶性高热的风险，其中当钙传导蛋白质受侵犯时恶性高热的风险性更高或者在理论上可能。（姜旭晖 译，马皓琳 李士通 校）

In this article, we analyze myopathies with cores, for which an association to malignant hyperthermia (MH) has been suggested. We discuss the clinical features, the underlying genetic defects, subsequent effects on cellular calcium metabolism, and *in vitro* muscle responses to MH triggers. We describe in detail central core disease, multiminicore disease, and nemaline rod myopathy. We categorize the diseases according to the affected proteins and discuss the risk for MH, which is high or theoretically possible when the calcium-conducting proteins are affected.

开颅手术中手术室工作人员暴露于七氟醚的相关情况

The Relative Exposure of the Operating Room Staff to Sevoflurane During Intracerebral Surgery

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背景：这个研究中，我们最初的目的是探讨开颅肿瘤切除术中，对于从手术部位散发的挥发性麻醉药七氟醚的接触量，神经外科医生是否大于麻醉医生。
方法：首先，我们测定了 35 例颅内肿瘤切除术从硬脑膜打开到关闭，从手术部位散发的七氟醚量。挥发性麻醉药吸收剂被放置在三个测定部位：1)外科医生呼吸区域、2)麻醉医生呼吸区域、以及 3)离手术部位最远的手术室角落。在第二个采样系列中(包括 16 名病人)，第一个采样系列中被放置在手术室最远角落的吸收剂这次

被放置在病人的嘴边 (5cm 以内)。被吸收剂吸附的七氟醚用色谱法由一名独立的药剂师进行定量测定。
结果：外科医生呼吸区域吸收剂吸附的七氟醚量(0.24 ± 0.04 ppm)明显低于麻醉医生呼吸区域(1.40 ± 0.37 ppm)，且与手术室最远角落吸收剂吸附的七氟醚量(0.25 ± 0.07 ppm)相当。吸收剂吸附的七氟醚量与手术切口大小没有相关性，即使调整了手术时间这一变量，两者也没有相关性。在第二个采样系列中，病人嘴边的吸收剂吸附了最高量的七氟醚(1.54 ± 0.55 ppm)，其次是麻醉医生呼吸区域(1.14 ± 0.43 ppm)和外科医生呼吸区域(0.15 ± 0.05 ppm)。
结论：外科医生呼吸区域最接近的手术部位并不是增加七氟醚暴露的来源。我们观察到麻醉医生在手术室环境中暴露于七氟醚更多，这有必要深入研究。
（张莹译 马皓琳 李士通校）

BACKGROUND: Our primary aim in this study was to investigate whether escape of the volatile anesthetic sevoflurane from the surgical site during craniotomy for tumor resection increases the exposure of the neurosurgeon to the anesthetic when compared with the anesthesiologist.
METHODS: Initially, the release of sevoflurane from the surgical site was measured during 35 tumorectomies starting from opening to closure of the dura. Volatile anesthetic absorbers were placed at three detection sites: 1) the surgeon's breathing zone, 2) the anesthesiologist's breathing zone, and 3) the farthest corner of the operation room. In the second sampling series that included 16 patients, the detector that had been in the corner of the operating room in the first series was now placed in the vicinity of the patient's mouth (within 5 cm). Sevoflurane captured by the absorbers was quantified by an independent chemist using chromatography.
RESULTS: Absorbers in the surgeon's breathing zone (0.24 ± 0.04 ppm) captured a significantly lower amount of sevoflurane compared with absorbers in the anesthesiologist's breathing zone (1.40 ± 0.37 ppm) and comparable with that in the farthest corner of the operation room (0.25 ± 0.07 ppm). There was no correlation between the amount of absorbed sevoflurane and the size of craniotomy window, even when adjusting for the variation in duration of surgery. In the second series of sampling, absorbers in the proximity of the patient's mouth captured the highest amount of sevoflurane (1.54 ± 0.55 ppm), followed by the anesthesiologist's (1.14 ± 0.43 ppm) and the surgeon's (0.15 ± 0.05 ppm) breathing zones.
CONCLUSIONS: The close proximity of the surgeon's breathing zone to the craniotomy window does not appear to be a source of increased exposure to sevoflurane. The observed higher exposure of the anesthesiologist to sevoflurane in the operating room environment warrants further exploration.

性别对咪达唑仑全身麻醉下正常受试者上呼吸道阻塞引起的代偿性神经肌肉反应的影响

The Effect of Gender on Compensatory Neuromuscular Response to Upper Airway Obstruction in Normal Subjects Under Midazolam General Anesthesia

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背景：在睡眠和麻醉时解剖学改变（机械性能）或神经控制受扰（补偿性神经肌肉反应）都可能危及上呼吸道通畅。麻醉时上呼吸道阻塞的病理生理学在男性和女性可能不同。近来，我们报道通过在咪达唑仑镇静时测量被动临界闭合压(P_{CRIT})和上游阻力(R_{US})，上呼吸道机械性能与自然非快眼动相睡眠相当。在这个研究中，我们比较了性别对咪达唑仑全身麻醉下上呼吸道阻塞引起的代偿性神经肌肉反应的影响。
方法：研究 32 例受试者（14 名男性，18 名女性）。在咪达唑仑麻醉过程中建立压力-流量关系来评估 P_{CRIT} 和 R_{US}。咪达唑仑麻醉诱导用咪达唑仑首量 0.07–0.08 mg/kg bolus，维持以 0.3–0.4 $\mu g \cdot kg^{-1} \cdot min^{-1}$ 的速度持续输注，用 Ramsay（5 级）和 OAA/S（2 级）评分评估麻醉水平。监测多项睡眠生理检查和血液动力学变量，同时记录鼻腔压力（通过面罩）、吸气气流（通过呼吸速度描记器）和颏舌肌肌电图(EMG_{GG})。在 EMG_{GG} 衰减的被动条件下和 EMG_{GG} 增加的主动条件下得到 P_{CRIT}（分别为被动 P_{CRIT} 和主动 P_{CRIT}）。在每例受试者计算主动 P_{CRIT} 和被动 P_{CRIT} 间的差($\Delta P_{CRIT\ P-A}$)来测定代偿性神经肌肉反应。
结果：$\Delta P_{CRIT\ A-P}$ 在女性明显大于男性(女性 4.6 ± 2.8 cm H_2O ，男性 2.2 ± 1.7 cm H_2O, $P < 0.01$)，提示对不依赖于觉醒状态的上呼吸道阻塞有更大的代偿性神经肌肉反应。
结论：我们证明咪达唑仑麻醉中对上呼吸道阻塞的不依赖于觉醒状态的补偿性神经肌肉反应在女性中部分维持，性别可能是麻醉中代偿性反应强度的决定性因素。
（朱 慧译 马皓琳 李士通校）

BACKGROUND: Upper airway patency may be compromised during sleep and anesthesia by either anatomical alterations (mechanical properties) or disturbances in the neural control (compensatory neuromuscular responses). The pathophysiology of upper airway obstruction during anesthesia may differ between men and women. Recently, we reported that the upper airway mechanical properties were comparable with those found during natural nonrapid eye movement sleep, as evaluated by measurements of passive critical closing pressure (P_{CRIT}) and upstream resistance (R_{US}) during midazolam sedation. In this study, we compared the effects of gender on compensatory neuromuscular responses to upper airway obstruction during midazolam general anesthesia.
METHOD: Thirty-two subjects (14 men and 18 women) were studied. We constructed pressure-flow relationships to evaluate P_{CRIT} and R_{US} during midazolam anesthesia. The midazolam anesthesia was induced with an initial dose of midazolam (0.07–0.08 mg/kg bolus) and maintained by midazolam infusion (0.3–0.4 $\mu g \cdot kg^{-1} \cdot min^{-1}$), and the level of anesthesia was assessed by Ramsay score (Level 5) and Observer's Assessment of Alertness/Sedation score (Level 2). Polysomnographic and hemodynamic variables were monitored while nasal pressure (via mask), inspiratory air flow (via pneumotachograph), and genioglossal electromyograph (EMG_{GG}) were recorded. P_{CRIT} was obtained in both

the passive condition, under conditions of decreased EMG_{GG} (passive P_{CRIT}), and in an active condition, whereas EMG_{GG} was increased (active P_{CRIT}). The difference between the active P_{CRIT} and passive P_{CRIT} ($\Delta P_{CRIT\ P-A}$) was calculated in each subject to determine the compensatory neuromuscular response.
RESULTS: The difference between the active P_{CRIT} and passive P_{CRIT} ($\Delta P_{CRIT\ A-P}$) was significantly greater in women than in men (4.6 ± 2.8 cm H_2O and 2.2 ± 1.7 cm H_2O, respectively; $P < 0.01$), suggesting greater compensatory neuromuscular response to upper airway obstruction independent of arousal.
CONCLUSION: We demonstrate that the arousal-independent compensatory neuromuscular responses to upper airway obstruction during midazolam anesthesia were partially maintained in women, and that gender may be a major determinant of the strength of compensatory responses during anesthesia.

从实验室及理论角度分析安氟醚的皮质电效应

The Electrocortical Effects of Enflurane: Experiment and Theory

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背景：高浓度的安氟醚能引起典型的脑电图：包括周期性抑制与大而短的突发性癫痫样放电(PEDs)交替。在本研究中，我们比较了这种活性的理论性计算机模型与从麻醉大鼠中获得的真实局部场电位(LFP)数据。
方法：将 8 x 8 的高密度电极植入视觉皮层后，记录大鼠在 0.5、1.0、1.5 及 2.0 倍最小肺泡麻醉浓度(MAC)的安氟醚麻醉时 LFP 及多元峰活性。将来自于新皮层动力学平均场模型的电脑模拟与这些记录进行比较。通过延长抑制性突触后电位(IPSP)衰减时间常数模拟增加安氟醚浓度所产生的神经元效应。与新皮层的激发率相反，我们调整了兴奋性突触后电位(EPSP)的振幅。
结果：在麻醉大鼠中，安氟醚浓度的持续增加会始终引起 LFP 记录中表现出抑制波型(>1.5 MAC)。多元锋电位的平均速率从 2.54/s (0.5 MAC) 下降到 0.19/s (2.0 MAC)。在高 MAC 时，大多数的多元动作电位事件变得与 PED 同步。在理论模型中，IPSP 衰减时间的延长及活性依赖 EPSP 的调整所导致的输出结果与从实验数据中所获得的形态相似。通过分析方程的本征值来测定模型中节律性暴发样活动的倾向。
结论：使用新皮层动力的平均场理论来复制安氟醚麻醉大鼠 LFP 中所观察到的 PED 图型是可能的。该图型需要作一些综合调整：适当增加 IPSP 下的总面积，延长 IPSP 衰减时间，及对 EPSP 的振幅进行活性依赖的调整。
（裘毅敏译，马皓琳、李士通校）

BACKGROUND: High concentrations of enflurane will induce a characteristic electroencephalogram pattern consisting of periods of suppression alternating with large short paroxysmal epileptiform discharges (PEDs). In this study, we compared a theoretical computer model of this activity with real local field potential (LFP) data obtained from anesthetized rats.
METHODS: After implantation of a high-density 8 x 8 electrode array in the visual cortex, the patterns of LFP and multiunit spike activity were recorded in rats during 0.5, 1.0, 1.5, and 2.0 minimum alveolar anesthetic concentration (MAC) enflurane anesthesia. These recordings were compared with computer simulations from a mean field model of neocortical dynamics. The neuronal effect of increasing enflurane concentration was simulated by prolonging the decay time constant of the inhibitory postsynaptic potential (IPSP). The amplitude of the excitatory postsynaptic potential (EPSP) was modulated, inverse to the neocortical firing rate.
RESULTS: In the anesthetized rats, increasing enflurane concentrations consistently caused the appearance of suppression pattern (>1.5 MAC) in the LFP recordings. The mean rate of multiunit spike activity decreased from 2.54/s (0.5 MAC) to 0.19/s (2.0 MAC). At high MAC, the majority of the multiunit action potential events became synchronous with the PED. In the theoretical model, prolongation of the IPSP decay time and activity-dependent EPSP modulation resulted in output that was similar in morphology to that obtained from the experimental data. The propensity for rhythmic seizure-like activity in the model could be determined by analysis of the eigenvalues of the equations.
CONCLUSION: It is possible to use a mean field theory of neocortical dynamics to replicate the PED pattern observed in LFPs in rats under enflurane anesthesia. This pattern requires a combination of a moderately increased total area under the IPSP, prolonged IPSP decay time, and also activity-dependent modulation of EPSP amplitude.

评估围手术期使用普加巴林对预防和减轻腹腔镜下胆囊切除术后肩痛的效果

An Evaluation of Perioperative Pregabalin for Prevention and Attenuation of Postoperative Shoulder Pain After Laparoscopic Cholecystectomy

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腹腔镜手术后常发生肩痛（PLSP）。在这个安慰剂对照的研究中，我们评估了围手术期用普加巴林 300 毫克间隔 12 h 给予两个剂量来预防和减轻腹腔镜下胆囊切除手术后 PLSP 的疗效。术后 48 小时评价 PLSP 的发生率和严重度、术后镇痛的需要及副作用。在两个组中，PLSP 的总体发生率没有明显差异，且 PLSP 疼痛评分、第一次用镇痛药的时间，和累计酮咯酸消耗量在每个时间点是相似的。然而，普加巴林组在术后 2 小时过度镇静的发生率较高。
（彭中美 译 马皓琳 李士通 校）
Postlaparoscopic shoulder pain (PLSP) frequently follows laparoscopic surgery. In this placebo-controlled study, we evaluated the efficacy of two perioperative doses of

pregabalin 300 mg 12 h apart for preventing and attenuating PLSP after laparoscopic cholecystectomy. The frequency and severity of PLSP, need for postoperative rescue analgesia, and side effect profiles were assessed for 48 h postoperatively. In both groups, the overall incidence of PLSP did not differ significantly, and the pain score for PLSP, time to first rescue analgesia, and cumulative ketorolac consumption were similar at each timepoint. However, the 2-h postoperative incidence of oversedation was higher with pregabalin.

小鼠坐骨神经中细胞外信号调节激酶激活促进坐骨神经部分结扎后神经性疼痛

Activation of Extracellular Signal-Regulated Kinase in Sciatic Nerve Contributes to Neuropathic Pain After Partial Sciatic Nerve Ligation in Mice

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背景：分裂原活化蛋白激酶族在多种类型的疼痛中具有重要作用。但是对磷酸化细胞外信号调节激酶（pERK）在受伤的周围神经局部的具体作用的认识还很少。在本文中，我们研究了在小鼠受损的坐骨神经损伤中 pERK 是否促进由坐骨神经部分结扎（PSL）诱发的神经性疼痛。
方法：小鼠行坐骨神经部分结扎；通过蛋白质印迹法和免疫组织化学方法测定坐骨神经中的 pERK1/2 (p44/42)。在 PSL 前 30min 于神经内注射 U0126（一种 ERK 激酶抑制剂）20 nmol/2 μL，在 PSL 后 1 天于神经周围注射 20 nmol/10 μL。PSL 诱发的热痛觉过敏和触觉异常性疼痛分别由热板缩爪试验和 von Frey 试验来测定。
结果：通过 Western blot 检测，在假手术的小鼠，坐骨神经 pERK1/2 水平在第 1-14 天均稳定且与非手术者相同。在 PSL 手术后小鼠，pERK1/2 在 PSL 后 1 天显著增高并且持续至第 3 天。通过免疫组织化学检测，与假手术的坐骨神经相比， PSL 手术后的坐骨神经 pERK1/2 免疫反应性在 PSL 后 1 天显著增高。通过双重免疫染色显示，在 PSL 后 1 天，坐骨神经中 pERK1/2 免疫反应性增强与许旺细胞标记物胶质纤维酸性蛋白（GFAP）共存，而非巨噬细胞的标记物 F4/80。在使用 U0126 处理能显著减轻 PSL 后第 3，7 和 14 天的热痛过敏，也能显著减轻 PSL 后第 7 和第 14 天时的触觉异常性疼痛。
结论：受损伤的周围神经系统许旺细胞 ERK 激活可能在神经性疼痛发展中具有重要作用。我们的结果提示 pERK 本身和 ERK 相关介质是治疗神经性疼痛的潜在治疗靶点。
（颜涛 译， 马皓琳 李士通 校）

BACKGROUND: The mitogen-activated protein kinase family plays an important role in several types of pain. However, the detailed role of phosphorylated extracellular signal-regulated kinase (pERK) in the region of injured peripheral nerve is poorly understood. In this study, we investigated whether pERK in injured sciatic nerve contributes to neuropathic pain induced by partial sciatic nerve ligation (PSL) in mice.
METHODS: Mice received PSL; pERK1/2 (p44/42) in sciatic nerve was measured by both Western blotting and immunohistochemistry. U0126 (an ERK kinase inhibitor) was

injected twice, an intraneural injection (20 nmol/2 μL) 30 min before PSL, and a perineural injection (20 nmol/10 μL) on Day 1 after PSL. Thermal hyperalgesia and tactile allodynia induced by PSL were evaluated by the thermal paw withdrawal test and the von Frey test, respectively.
RESULTS: As measured by Western blotting, in sham-operated mice, the levels of pERK1/2 in sciatic nerve were constant and the same as those in naive mice across Days 1-14. In PSL-operated mice, a significant increase in pERK1/2 was observed on Day 1 after PSL and persisted until Day 3. As measured by immunohistochemistry, immunoreactivity of pERK1/2 in PSL-operated sciatic nerve was markedly increased in comparison with that in sham-operated sciatic nerve on Day 1 after PSL. In the sciatic nerve on Day 1 after PSL, as indicated by double immunostaining, the increased immunoreactivity of pERK1/2 was colocalized with glial fibrillary acidic protein (GFAP), a marker of Schwann cells, but not F4/80, a marker of macrophages. PSL-induced thermal hyperalgesia was significantly attenuated by treatment with U0126 on Days 3, 7, and 14 after PSL. The PSL-induced tactile allodynia was also significantly attenuated by treatment with U0126 on Days 7 and 14 after PSL.
CONCLUSION: Activation of ERK in Schwann cells of the injured peripheral nervous system may play an important role in the development of neuropathic pain. Our results suggest that pERK itself and ERK-related mediators are potential therapeutic targets for the treatment of neuropathic pain.

脂肪乳剂可以改善从布比卡因引起的心搏骤停恢复，但对罗哌卡因或甲哌卡因引起的心搏骤停无效

Lipid Emulsion Improves Recovery from Bupivacaine-Induced Cardiac Arrest, but Not from Ropivacaine- or Mepivacaine-Induced Cardiac Arrest

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背景：局麻药的心脏毒性与其亲油性显著相关。最近，已有研究显示持续输注脂肪乳剂是一个有希望能处理局麻药引起的心搏骤停的方法。根据其可能的作用机制，所谓的“脂质沉积”作用可能决定于局麻药的亲油性。在这项研究中，我们探讨脂质的作用是否随着所给局麻药的不同而不同。
方法：在离体的大鼠心脏中，分别给予等效剂量的布比卡因、罗哌卡因和甲哌卡因引起心搏骤停，然后用或不用脂肪乳剂进行心脏灌注($0.25\ mL \cdot kg^{-1} \cdot min^{-1}$)。然后，评定从开始灌注到发生第一次心脏活动恢复的时间，及到心率和心率－收缩压乘积恢复的时间。
结果：在所有的小组中，脂肪乳剂对任何心脏活动恢复时间没有影响。给予脂质却可以显著缩短布比卡因引起的心脏毒性中的心率和心率－收缩压乘积恢复时间，但是对罗哌卡因和甲哌卡因引起的心脏毒性没有影响。

结论：这些数据显示脂肪乳剂对局麻药引起的心脏毒性的影响主要决定于所给的局麻药本身。我们得出结论，局麻药的亲油性显著影响脂肪乳剂处理心搏骤停的有效性。

（黄佳佳译，马皓琳 李士通 校）

BACKGROUND: Cardiac toxicity significantly correlates with the lipophilicity of local anesthetics (LAs). Recently, the infusion of lipid emulsions has been shown to be a promising approach to treat LA-induced cardiac arrest. As the postulated mechanism of action, the so-called "lipid sink" effect may depend on the lipophilicity of LAs. In this study, we investigated whether lipid effects differ with regard to the administered LAs.
METHODS: In the isolated rat heart, cardiac arrest was induced by administration of equipotent doses of bupivacaine, ropivacaine, and mepivacaine, respectively, followed by cardiac perfusion with or without lipid emulsion (0.25 mL · kg^{-1} · min^{-1}). Subsequently, the times from the start of perfusion to return of first heart activity and to recovery of heart rate and rate-pressure product (to 90% of baseline values) were assessed.
RESULTS: In all groups, lipid infusion had no effects on the time to the return of any cardiac activity. However, recovery times of heart rate and rate-pressure product (to 90% of baseline values) were significantly shorter with the administration of lipids in bupivacaine-induced cardiac toxicity, but not in ropivacaine- or mepivacaine-induced cardiac toxicity.
CONCLUSIONS: These data show that the effects of lipid infusion on LA-induced cardiac arrest are strongly dependent on the administered LAs itself. We conclude that lipophilicity of LAs has a marked impact on the efficacy of lipid infusions to treat cardiac arrest induced by these drugs.

星状神经节阻滞后分形心血管动力和压力反射敏感性的状态

Fractal Cardiovascular Dynamics and Baroreflex Sensitivity After Stellate Ganglion Block

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背景：研究现实星状神经节阻滞可降低压力反射的敏感性。本研究主要目的为确定心率和收缩压变异性分形动力学（自体相似波动模式的动态改变）是否均参与了星状神经节阻滞后压力反射敏感性降低的机制。
方法：16 名健康年轻志愿者参与了本次研究。采用 1%甲哌卡因 6mL 间隔 1~1[illegible]月对志愿者行左或右星状神经节阻滞。阻滞前、阻滞后 30、60、90 和 120 分钟进行心率和收缩压变异形频谱分析。每次频谱分析后即刻采用直立倾斜试验评估压力反射敏感性。
结果：经直立倾斜试验评估，右侧或左侧星状神经节阻滞后 30 分钟压力反射敏感性显著降低（分别为 1.26 ± 0.18 到 0.46 ± 0.08 bpm/mm Hg, $P < 0.05$ 和 1.17 ± 0.35 到 0.51 ± 0.13 bpm/min, $P < 0.01$）。反映波动自体相似性程度的分形斜率在右侧或左侧星状神经节阻滞后 30 分钟显著增加（右星状神经节阻滞－心率；–1.08 ± 0.30

到 –1.62 ± 0.22, $P < 0.01$；右星状神经节阻滞－收缩压；–1.30 ± 0.80 到 –2.40 ± 0.80, $P < 0.05$；左星状神经节阻滞－收缩压；–1.20 ± 0.40 到–2.13 ± 0.50, $P < 0.05$）。心率变异性分析显示分形斜率在左星状神经节阻滞后没有改变。
结论：分形斜率的增加提示心率和收缩压变异性的复杂性消失（保持复杂行为的状态），这是星状神经节阻滞后压力反射敏感性降低的机制之一。
（周雅春 译 马皓琳 李士通 校）
BACKGROUND: It has been shown that stellate ganglion block can attenuate baroreflex sensitivity. Our primary purpose in this study was to determine whether fractal dynamics (dynamic change of self-similar fluctuation patterns) of not only heart rate but also systolic blood pressure variability are involved in attenuation of baroreflex sensitivity after stellate ganglion block.
METHODS: Sixteen young, healthy volunteers entered the study. Spectral analysis of heart rate and systolic blood pressure variability was performed before and 30, 60, 90, and 120 min after either right or left stellate ganglion block, separated by a 1 to 1½-mo interval, with 6 mL of 1% mepivacaine. Shortly after each spectral analysis, baroreflex sensitivity was assessed with the head-up tilt test.
RESULTS: Baroreflex sensitivity, assessed by the head-up tilt test, was significantly attenuated at 30 min after either right or left stellate ganglion block (1.26 ± 0.18 to 0.46 ± 0.08 bpm/mm Hg, $P < 0.05$ and 1.17 ± 0.35 to 0.51 ± 0.13 bpm/min, $P < 0.01$, respectively). Fractal slopes reflecting the degree of self-similarity of fluctuations were significantly increased at 30 min after either right or left stellate ganglion block (right stellate ganglion block—heart rate; –1.08 ± 0.30 to –1.62 ± 0.22, $P < 0.01$; right stellate ganglion block—systolic blood pressure; –1.30 ± 0.80 to –2.40 ± 0.80, $P < 0.05$; left stellate ganglion block—systolic blood pressure; –1.20 ± 0.40 to –2.13 ± 0.50, $P < 0.05$). Fractal slope did not change after left stellate ganglion block with heart rate variability analysis.
CONCLUSIONS: Loss of complexity (status of being complex behavior) of both heart rate and systolic blood pressure variability, indicated by increased fractal slopes, is one mechanism in attenuating baroreflex sensitivity after stellate ganglion block.