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惡性高熱和肌肉萎縮症

Malignant Hyperthermia and Muscular Dystrophies

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Anesth Analg 2009 109: 1043-1048.

背景：據報導患有肌肉萎縮症（肌營養不良）的病人在全身麻醉時和麻醉後可能發生很多致命的併發症。作者對患有肌肉萎縮症的病人做了一項系統分析，旨在定義此類病人麻醉相關併發症的範疇，重點強調了惡性高熱的易感性。
方法：作者使用了多個搜尋引擎進行文獻檢索並對合適的文獻進行評價從而確定患肌肉萎縮症病人麻醉相關併發症。在所有肌肉萎縮症的類型中，Duchenne 型肌營養不良（DMD）和 Becker 型肌營養不良（BD）佔據了幾乎所有麻醉相關的報導。
結果：DMD 和 BD 病人麻醉相關併發症包括術中心力衰竭、吸入麻醉相關的橫紋肌溶解症（不用琥珀醯膽鹼）和琥珀醯膽鹼引起的橫紋肌溶解症及高鉀血症。
結論：與普通人群相比，並沒有發現 DMD 和 BD 病人增加了惡性高熱的易感性。但是，暴露於吸入麻醉藥的營養不良病人可能引起疾病相關的心臟併發症，或罕見的以橫紋肌溶解爲特徵的惡性高熱相似症狀。後者也可能發生在術後。琥珀醯膽鹼可以引起致命性高鉀血症，應避免用於 DMD 和 BD 患者。
（李潺 譯 陳傑 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.

勞累性熱病、運動性橫紋肌溶解症及惡性高熱之間的關係

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

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Anesth Analg 2009 109: 1065-1069.

勞累性熱病、運動性橫紋肌溶解症和惡性高熱（MH）具有相似病理生理學的綜合征。三者均有高代謝特徵，包括：三磷酸腺苷的高需求、氧化和代謝的加速、肌肉的機械應力，以及不受控制的細胞內鈣的增加。儘管沒有臨床的對照研究來支持其中的關係，但有證據表明預期外的熱/運動的不耐受和 MH 易感性相關。有多個病理報告和小樣本的臨床研究已經用於體外肌肉攣縮實驗和/或基因監測來證實這種關係。然而，這種方法存在問題，因爲這些研究在與麻醉有關係的臨床 MH 中證實有效，而不是與勞累性熱病或惡性高熱有關。然而，這些相互關係對某些 MH 易感的患者以及他們運動的能力可能有意義，同時對臨床醫生的治療和伴隨有原因不明勞累性熱病和運動疾的患者的麻醉具有重要意義。
（懷曉蓉 譯 陳傑 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 的鎮痛作用不影響其制動需求

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

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Anesth Analg 2009 109: 1111-1116.

背景：氧化亞氮（N_2O）作用於脊髓上去甲腎上腺素神經元產生鎮痛，但尚不清楚這一作用是否與氧化亞氮的制動作用有關。本研究作者擬檢驗如下假設：在脊髓上去甲腎上腺素神經元選擇性清除或初次接受 N_2O 且鎮痛缺乏的動物（naïve animals ）N_2O 最低肺泡麻醉濃度（MAC）不變。

方法：研究者測定了 70% N_2O 、一個 MACN_2O 或一個 MAC 異氟醚下在腦室內注射抗多巴胺-β 羥化酶共軛皂草素(SAP-DBH; $n = 7$)或對照抗體共軛皂草素($n = 5$)前或後甩尾潛伏期(TFL)和後爪退縮潛伏期(HPL) 。naïve animals 組($n = 8$) N_2O MAC 在吸入 N_2O25–45 min 後測定（鎮痛作用高峰期），並在 120–140 min （TFL 和 HPL 恢復到基礎值後）後再測定

結果：吸入 N_2O30min 後，TEL 和 HPL 值顯著升高，但在 120min 內回到基礎值。接受 SAP-DBH 大鼠 N_2O 鎮痛未達到效果。然而，N_2O 和異氟醚的 MAC 在 SAP-DBH 組和對照組間無顯著差異（N_2O 平均值±標準差：1.7 ± 0.1 vs 1.7 ± 0.2; 異氟醚：1.6 ± 0.2%vs 1.7 ± 0.2%）。naïve 動物的 N_2O 吸入 30min 與 120min 時 MAC 沒有差別（1.8 ± 0.1 vs 1.8 ± 0.2）。

結論：去除腦幹去甲腎上腺素能神經元或長期接觸 N_2O 將減弱其鎮痛效應，但不改變 MAC。N_2O 的制動作用的機制不依賴其鎮痛作用。

（陳毓雯 譯 陳傑 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.

頭戴式顯示器監測：全方位模擬器和部分任務訓練器的操作和安全性

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

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背景：頭戴式顯示器可以幫助麻醉醫師在任何時候都可以看到術中病人所監測的生命體征，即使麻醉醫師忙於操作或者無法看到監視器的時候。相關的麻醉文獻顯示使用 HMD（頭戴式顯示器）有其優點，但也有研究表明 HMD 會加重注意遲鈍（使用者很有可能因專注於顯示器而忽略眼前的其他事物），同時可能產生於焦距深度有關的知覺問題。本研究調查了兩種模擬器的差異。
方法：實驗一，研究戴上 HMD 是否會影響麻醉醫師發現病情的速度，以及 HMD 設置的不同焦距（近或遠）其結果是否會有差別。12 名麻醉醫師在三個自然場景中進行麻醉，這三個場景中均用模擬器模擬手術環境。在病人的監視器上或者手術室內出現 24 種不同的病情變化。實驗二，研究麻醉醫師由於進行操作而受到身體上的限制，使用 HMD 的麻醉醫師是否會比沒有使用 HMD 的麻醉醫師更快發現病情變化。12 名麻醉醫師在監測模擬病人生命體征的同時，要在部分任務訓練器上進行複雜的模擬臨床任務。所有的參與者在兩種場景中共會碰到 4 種不同的情況。
結果：實驗一表明帶上 HMD 或者調整設置的焦距都不會降低參與者發現病情變化的能力（包括發現病情情況的數量和時間）。總的來說，使用 HMD 後的參與者較只用標準監視器的時候會花更多的時間去看病人，更少的時間去看麻醉機、顯示器。參與者報告他們更喜歡近焦距的設置。實驗二顯示參與者使用 HMD 後發現其中 2 種病情變化的速度會更快，而另一種病情變化發現較慢。參與者使用 HMD 後去看麻醉機顯示器的頻率大大下降。使用 HMD 後，參與者表示他們沒這麼忙了，監測也更容易，而且他們相信發現異常情況變化的速度會更快。
結論：HMD 可以幫助麻醉醫師在身體受限的時候也能發現病情變化，而不是在身體不受限的時候。儘管沒有足夠的證據說明會加重不注意，但在航空方面發現，與 HMD 有關的知覺問題會影響到測試者是否能發現到情況的變化。麻醉醫師使用

HMD 後應該自我調整焦距，從而使眼睛的限制降到最低，同時也應該注意到一點，就是某些變化可能無法引起他們的注意。對於建立設計 HMD 的原則，評估其他型號的 HMD，及臨床應用方面還需進一步研究。
（張婷 譯 陳傑 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 紐約州麻醉引起的惡性高熱的流行率

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

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背景：惡性高熱是一種極易被誘因觸發的藥物遺傳學綜合症。在美國惡性高熱流行病學尚未很好的建立。本研究作者對紐約醫院的惡性高熱流行性進行評估。
方法：通過紐約 2001 年至 2005 年間的出院病人資料，使用第九版疾病的國際分類法，修改代碼 995.86，來識別由麻醉導致惡性高熱的病人。通過人口統計學和臨床症狀來評估惡性高熱的流行性。
結果：在這段研究期間紐約醫院的 12749125 位出院病人中，有 73 例發生麻醉引起的惡性高熱。近 3/4 患者爲男性、71%的病人爲急診入院患者。手術出院病人的惡性高熱患病率爲 0.96/100000（可信區間 95%，誤差 0.67-1.24），接受麻醉的出院病人中惡性高熱患病率爲 1.08/100000（可信區間 95%，誤差 0.75-1.41）。這項惡性高熱流行性的評估中男性是女性的 2.5~4.5 倍。
結論：在紐約州醫院手術後的病人中，麻醉引起的惡性高熱流行率約 1/100000。男性患惡性高熱的風險顯著大於女性。
（楊秋娟 譯 陳傑 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.

在病態肥胖的病人中阻塞性睡眠呼吸暫停不是困難插管的危險因素

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

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背景：我們普遍認爲病態肥胖症(MO)，阻塞性呼吸暫停(OSA)和頸圍是氣管插管的獨立危險因素。本研究中，作者試圖確定這些因素對於在經歷減肥手術的病人是否會增加困難插管風險。通過測定呼吸暫停低通氣指數(AHI)、性別、頸圍和體重指數(BMI)的因素來確定阻塞性呼吸暫停(OSA)和它的嚴重程度。
方法：所有已登記的病態肥胖症病人術前描記多導睡眠圖。阻塞性呼吸暫停的嚴重程度是通過呼吸暫停低通氣指數和美國麻醉醫師協會的阻塞性呼吸暫停嚴重等級來確定。全部病人用統一標準的麻醉藥及方法，包括使用傾斜位直接喉鏡法。
結果：有 180 位連續病例入選，140 位女性，40 位男性。阻塞性呼吸暫停的發生率爲 68%，平均體重指數是 49.4 kg/m^2，平均呼吸暫停低通氣指數是 31.3 (範圍, 0-135)。所有病人的氣管插管均未用急救氣道並由麻醉住院醫生完成。6 例病人嘗試了 3 次及 3 次以上插管才成功。困難插管的發生率爲 3.3%，困難喉鏡暴露（Cormack and Lehane 3 級或 4 級）的發生率爲 8.3%。頸圍和困難氣管插管無關(優勢比 1.02, 95% 可信區間 0.93-1.1)，阻塞性呼吸暫停和困難插管之間無顯著相關(*P* = 0.09)，體重指數和困難插管之間也無顯著相關(優勢比 0.99, 95%可信區間 0.92-1.06, *P* = 0.8)。插管的嘗試次數和體重指數 BMI (*P* = 0.8), 呼吸暫停低通氣指數 AHI (*P* = 0.82) 及頸圍 NC (*P* = 0.3)之間沒有關係。Mallampati 評級 III 或者更高預示著困難插管(*P* = 0.02)，男性也是如此(*P* = 0.02)。最後，Cormack and Lehane 分級與體重指數 BMI (*P* = 0.88)；呼吸暫停低通氣指數 AHI (*P* = 0.93)；阻塞性呼吸暫停 OSA (*P* = 0.6)無關。頸圍的增大與喉鏡暴露困難有關，但是和困難插管無關(*P* = 0.02)。
結論：病態肥胖症病人斜坡位減肥手術時，困難插管或者喉鏡暴露困難和阻塞性呼吸暫停, 體重指數, 或者頸圍這些因素的有無及嚴重程度無關。只有 Mallampati 評分爲 3 或者 4 和男性病人才預示著困難插管。
（陳靈科 譯 陳傑 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 提高使用大劑量腎上腺素 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：多種干預措施用於心肺復蘇（CPR）的研究，以優化藥物的使用、胸外按壓和通氣功能。沒有研究顯示呼氣末正壓（PEEP）對於 CPR 結果的作用。作者推測，由於呼氣末正壓可扭轉肺不張，降低肺血管阻力，並有可能改善心輸出量，因此，CPR 中使用 PEEP 將提高存活率。
方法：麻醉的 Sprague - Dawley 大鼠暴露於 1 分鐘窒息心臟驟停。復蘇程式爲標準化的胸部按壓、吸氧（Fio_2 1.0）和靜脈注射腎上腺素 30μg/kg（組 1）和 10μg/kg（組 2）。使用超聲心動圖評估左心功能（組 1），大鼠在 CPR 開始階段或者是整個復蘇過程中隨機接受 5cm H_2O PEEP 或 0 PEEP。存活定義爲初步復蘇後，自主迴圈恢復並持續 60min 或者 120min（組 2）。
結果：組間基礎情況無差異。在組 1，與 0 PEEP 相比較，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）。應用 5 cm H_2O PEEP (Fio_2 1.0)能夠增加左室舒張末期容積，全身氧供和功能殘氣量。呼氣末正壓的使用並沒有影響左室收縮功能或動脈血壓。其結果差異不是因爲氧合的增加，因爲其存活率依次爲 5 cm H_2O PEEP (Fio_2 1.0) ≥5 cm H_2O PEEP (Fio_2 0.21) >

zero PEEP (Fio_2 1.0)，然而其動脈血氧分壓的排序依次爲 5 cm H_2O PEEP (Fio_2 1.0) > 5 cm H_2O PEEP (Fio_2 0.21) ≈zero PEEP (Fio_2 1.0)。在另外一個組中，使用 10 μg/kg 腎上腺素，即使其存活率爲 100% ，PEEP 的益處仍有限。

結論：齧齒動物窒息心跳驟停模型，在 CPR 期間和之後持續呼氣末正壓通氣（5 cm H_2O），對於存活率有多方面的有益作用，但與吸入氧濃度無關，且無不良心血管效應。

（張蕾 譯 陳傑 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.

肥胖對於妊娠患者神經阻滯技術難度的影響：一項前瞻性、觀察研究

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

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背景：從業者常常假定肥胖會增加妊娠患者神經阻滯技術的難度，但是很少有人系統地調查過與硬膜外或蛛網膜下腔阻滯相關的危險因素。作者設計這項前瞻性研究來預測妊娠患者神經阻滯技術困難的因素。
方法：使用一前瞻性、觀察表，觀察妊娠患者神經阻滯困難的多項潛在的危險因素，包括體重指數，能夠觸摸到棘突的程度，最大的背彎曲度，脊柱側凸和從業人員的經驗。用兩種方法來評估神經阻滯麻醉的難點：1）到達所需間隙的進針深度；2)從皮膚進針到蛛網膜下腔注射或硬膜外導管放置妥善所需的時間。將這些資料代入一個廣義的負誤差二項式線性模型從而決定總的進針過程的預測。神經阻滯麻醉時間的預測取決於其麻醉操作時間的線性模型。生存模型用來說明主治醫生干預住院醫生過程中的偏差。
結果：研究了 427 例妊娠患者的神經阻滯麻醉程式，對於進針深度和操作時間，重要的預測難點是醫生觸摸病人骨標誌的能力和病人的背彎曲程度。肥胖，即體重指數不是一個獨立的預測因數。儘管如此，肥胖的確能預測觸摸骨標誌的能力和背的彎曲度。
結論：儘管肥胖可能會導致神經阻滯困難，但一些肥胖病人令人驚訝的很容易進行神經阻滯。當進行任何一例妊娠患者尤其是肥胖產婦的神經阻滯麻醉時，背彎曲度和骨標誌的觸摸能預測神經阻滯技術的難度。
（唐穎 譯 陳傑 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.

羥考酮與嗎啡在腹腔鏡子宮切除術後的病人靜脈自控鎮痛之比較

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

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背景：在這項研究中，作者研究了術後有內臟疼痛患者所需羥考酮和嗎啡的劑量，緩解疼痛的程度以及副作用。
方法：91 位行腹腔鏡子宮切除術的女患者在手術結束前接受羥考酮或嗎啡靜脈注射，並在術後 24 小時給予病人自控鎮痛術。
結果：羥考酮的累積消耗量比嗎啡少（13.3 ± 10.4 mg 比 22.0 ± 13.1 mg, P = 0.001）。使用羥考酮，術後第一小時的視覺類比量表評分顯著低，但術後 24 小時的鎮靜效果差，P=0.006。
結論：與嗎啡相比，羥考酮更適於緩解內臟疼痛，但不適用於鎮靜。
（鄒巧群 譯 陳傑 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.

內啡肽-1 和犬尿喹啉酸在大鼠炎症關節模型中的外周抗傷害效應

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

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背景：大量資料顯示，嗎啡和 n-甲基-d-天冬氨酸（NMDA）受體都位於外周水準，而作用於這些受體的藥物經局部給藥後，均可產生抗傷害作用，但是這些受體的內源性配體的抗傷害效應目前仍不明確。本次研究的目的在於確定內源性阿片肽，即內啡肽-1（EM1）和內源性 NMDA 受體拮抗劑犬尿喹啉酸（KYNA）的抗傷害效能，並在大鼠驗證關節模型中研究兩者在外周水準的相互作用。
方法：在大鼠右後足脛骨跗骨關節內注射角叉菜聚糖（300 μg/20μL）以產生機械性超敏反應。用 von Frey 細絲（0.064-110 g）評估其機械痛閾。將 EM1（30、100 和 200μg）、KYNA（30、100、200 和 400μg）以及以 1:1 的比例相混的兩者的混合液分別注入受感染的關節，並在給藥後 75min 時再次測定痛閾。
結果：向炎症關節注射 EM1 或 KYNA 對非炎症側的痛閾沒有影響。兩者的配體都能產生劑量依賴性的抗痛覺過敏效應，且最大劑量可導致延遲效應。相較於 KYNA（30%有效劑量[ED30]和 50%有效劑量[ED50]分別爲 204μg[可信區間{CI}：160-251]和 330μg[CI：280-407]），EM1（ED30 和 ED50 分別爲 112μg[CI：80-146]和 167μg[CI：135-220]）的抗傷害效能更強大。預先皮下應用納曲酮可抑制 EM1 的抗傷害效應。同時應用 EM1 和 KYNA 可產生增強的和/或延遲的抗傷害效應。混合液的 ED30 和 ED50 分別爲 141μg[CI：83-182]和 231μg[CI：190-293]，這與兩者理論上的疊加值（ED30 和 ED50 分別爲 145μg[CI：68-237]和 220μg[CI：144-230]）沒有明顯的差異，也就是說兩個配體的相互作用產生的是疊加效應。所有實施的治療均未產生任何併發症。
結論：外周給予內源性阿片類激動劑和 NMDA 受體拮抗劑的配體可能可以對抗炎性疼痛。由於這兩種藥物很少越過血腦屏障，因此局部使用不會產生中樞性的副作用。
（周姝婧 譯 陳傑 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.

大鼠通過舔（Licking）後爪可減少福馬林致痛實驗後脊髓背角胞外信號調節激酶的磷酸化

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：傷害性行爲可能減弱痛覺。最近研究表明胞外信號調節磷酸激酶(pERK)是由脊髓後角神經傷害性刺激引起的。作者研究福馬林實驗中脊髓後角的pERK是否受通過舔（licking）的影響。
方法：將 24 只成年大鼠分爲四組：對照組，福馬林致痛實驗組，活動受限對照組及活動受限福馬林致痛實驗組。福馬林致痛實驗組及活動受限福馬林致痛實驗組大鼠在其後爪皮下注射 10%福馬林。對照組與福馬林致痛實驗組小鼠放置在一乾淨塑膠小室中，而活動受限對照組及活動受限福馬林致痛實驗組大鼠置於一改良制動管狀小室中。所有大鼠在 25min 後處死。使用免疫組化技術卵白素生物素過氧化物酶方法檢測腰椎十二節段的 pERK 值。
結果：在活動受限福馬林致痛實驗中，患側淺層脊髓後角 pERK 陽性細胞數量顯著高於其它三組($P < 0.05$)。福馬林致痛實驗組的 pERK 表達與其他兩組並無顯著性差異。
結論：大鼠通過舔後爪減少福馬林致痛實驗中脊髓後角pERK的表達。這一發現表明通過舔後爪減弱了福馬林致痛實驗的疼痛。
（趙嫣紅 譯 陳傑 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.

下肢手術鞘內注射時布比卡因、左旋布比卡因以及羅呱卡因的半數有效劑量

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

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背景：鞘內麻醉普遍應用於下肢手術，布比卡因、左旋布比卡因以及羅呱卡因都已被用作鞘內藥物，但它們的相對效價目前還沒完全確定。在這項研究中，作者擬確定這三種局部麻醉藥用於下肢手術鞘內麻醉的半數有效劑量(ED_{50})，從而評估三者的相對效價。
方法：75 名腰硬聯合麻醉下行下肢手術的病人隨機分組，分別鞘內注射布比卡因或左旋布比卡因或羅呱卡因。局部麻醉藥的劑量使用升降冪貫分配法。每組第一個病人的劑量爲 8mg，劑量增加值設定爲 1mg。每組的序貫劑量由先前一個病人接受腰麻成功或失敗的結果來決定。鞘內注射後雙側 T12 冷感覺阻滯保留 20 分鐘，並且手術至少能順利進行至鞘內注射後 50min，其間不補充硬膜外麻醉藥物，則爲成功事件。用 Dixon and Massey 方法計算 ED_{50}。
結果：鞘內麻醉時布比卡因的 ED_{50} 爲 5.5mg，（95%的置信區間[CI]：4.90-6.10mg），左旋布比卡因的 ED_{50} 爲 5.68mg，（95%的 CI：4.92-6.44mg），羅呱卡因的 ED_{50} 爲 8.41mg，（95%的 CI：7.15-9.67mg）。左旋布比卡因與布比卡因麻醉相對效價比值是 0.97 (95%的 CI:：0.81-1.17)，羅呱卡因與布比卡因麻醉相對效價比值是 0.65 (95%的 CI： 0.54-0.80)，而羅呱卡因與左旋布比卡因麻醉相對效價比值是 0.68 (95%的 CI：0.55-0.84)。

結論：這項研究顯示，下肢手術鞘內麻醉時羅呱卡因的效價低於左旋羅呱卡因和布比卡因，而左旋羅呱卡因和布比卡因的效價相當。
（黃丹 譯 陳傑 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.

體外全血實驗發現抑制 XIII 因數會阻礙血凝形成，降低血凝塊穩定性並增加纖維蛋白溶解效應

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：在圍手術期進行血栓彈力圖的檢查又重新引起了人們的興趣。血栓彈力圖檢查結果的主要決定因素包括凝血因數濃度（各種酶原和纖維原）和血小板計數，因此血小板抑制劑會使得主要受凝血因數影響的檢測指標變得無意義。從而使得合併應用血小板抑制劑與否的檢測結果逐漸被用於評估和檢測血液製品替代治療的效果。在本次研究中，我們評估了 XIII 因數抑制劑配伍應用糖蛋白（GP）IIb/IIIa（血小板抑制）在全血血栓彈力圖中的效應，以及應用 XIII 因數抗體對常規檢測做一改進是否可用於檢測 XIII 因數缺乏症。
方法：正常全血會隨著非特異性抗體、抗 GPIIb/IIIa 抗體或者中性抗 XIII 因數抗體數量的增加而被孵化改變。樣本在經過組織因數啓動和血小板抑制的處理後進行分

析，得到全血血栓彈力圖。凝血時間、血凝塊形成時間、最大血凝塊穩定性以及 60 分鐘內血凝塊溶解程度被一式三份進行檢測。另外，25 個全血常規樣本分別用以下兩種方法來檢測 XIII 因數缺乏症：合併應用 XIII 因數抗體的新血栓彈力圖分析法和標準 XIII 因數檢測法，並將兩種方法進行比較。

結果：雖然 GPIIb/IIIa 抑制劑並沒有改變經血小板抑制的全血血栓彈力圖的檢測結果，但是 XIII 因數抑制劑卻明顯地降低了最大血凝塊穩定性（(P = 0.020），延長了血凝塊形成時間（P = 0.025）以及血凝塊溶解程度（P = 0.007），而凝血時間並未改變；並且在全血（而非血漿）中隨著抗體濃度的增加這一改變存在著封頂效應。應用這一血栓彈力圖檢測方法來發現 XIII 因數缺乏症，當檢測效能<70%時，敏感度爲 90%，且存在陰性預測值（受試者特徵性曲線下面積爲 0.803，P = 0.0015）；當檢測效能<60%時，敏感度和陰性預測值均爲 100%（受試者特徵性曲線下面積爲 0.84，P = 0.0037）。

結論：XIII 因數會明顯地影響血小板抑制劑啓動的全血血栓彈力圖結果。在分析出血患者，特別是接受促凝血治療的患者的血栓彈力圖結果的時候，應考慮到這一現象。由抗體介導的 XIII 因數抑制劑可被用於建立以血栓彈力圖爲基礎的檢驗方法來發現 XIII 因數缺乏症。

（單嘉琪譯 薛張綱校）

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.

肌強直和惡性高熱的易感性

The Myotonias and Susceptibility to Malignant Hyperthermia

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惡性高熱是一種骨骼肌遺傳性疾病，吸入麻醉藥可以觸發肌漿網內 Ca^{2+}的釋放從而導致機體高代謝、肌肉強直、橫紋肌溶解，嚴重可以致死。肌強直是一種疾病，它是由於與骨骼肌興奮收縮耦聯相關的基因突變所造成的，特殊的 DNA 序列重複造成諸如骨骼肌通道在內的許多蛋白質不穩定，從而影響的骨骼肌的興奮性。一般認爲有肌強直的病人增加了發展爲惡性高熱的風險。在這篇文章中，我們概述了肌肉興奮性及興奮收縮耦聯的生理過程，惡性高熱及肌強直的病理過程，並參考了和惡性高熱易感性相關的文獻。我們得出了如下結論，除了低鉀週期性麻痹外，其他肌病患者出現惡性高熱的幾率與普通人群相似。由於沒有低鉀週期性麻痹的病人發展爲惡性高熱的報導，儘管我們認爲這類病人的發病率很低，但是我們尚不能評估其發展惡性高熱的風險。

（陳珺珺譯 薛張綱校）

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.

長時間丙泊酚麻醉與血乳酸增加不相關

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

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背景：乳酸性酸中毒常被認爲是異丙酚輸注綜合征的先期標誌。此研究旨在探討長時間使用丙泊酚與揮發性吸入麻醉藥（VA）後血乳酸和 pH 值的變化情況。

方法：回顧術程長於 8 小時的擇期脊柱手術患者的麻醉記錄，從中獲取手術與人口統計學資料。根據麻醉時間（AT）（±30min）和出血量（BL）（±500ml），按 1：2 的比例，將接受丙泊酚麻醉的患者與接受 VA 麻醉的患者進行配比。

結果：在所回顧的 246 名患者中，共選取 50 名接受丙泊酚麻醉的患者（AT=10±2h，BL=1955±1409ml）與 100 名接受 VA 麻醉的患者（AT=10±1h，BL=1801±1543 毫升），其中分別有 40 和 72 名患者擁有完備的基線與麻醉後 8 小時血乳酸資料，故將其納入主要分析。丙泊酚組患者接受丙泊酚的平均劑量爲 8.8±2mg/kg/h。VA 組患者其年齡大於丙泊酚組患者（分別爲 58±12 歲與 51±15 歲，P=0.002），但兩者在性別、ASA 分級、術中血流動力學指標和血管升壓藥的使用等方面均無差異。8 小時後，與丙泊酚組相比，VA 組患者動脈血乳酸水準較基線值大幅增加（與基線值相比：丙泊酚組增幅爲 0.48±0.72mmol/L；VA 組增幅爲 1.2±1.2mmol/L，P=0.001）。

結論：在術程長於 8 小時的脊柱手術中，與輸注異丙酚相比，接受 VA 麻醉可使血乳酸水準大幅增加。但這一結論的確切機制和臨床意義仍需前瞻性研究進一步闡明。

（范羽譯 薛張綱校）

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.

異氟烷抑制SH-SY5Y細胞的環磷酸腺苷反應組件結合蛋白磷酸化以及鈣調蛋白易位至細胞核

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(鈣調蛋白)被鈣離子啓動後易位至細胞核，從而刺激CREB(環磷酸腺苷反應組件結合蛋白)磷酸化形成P-CREB(磷酸化環磷酸腺苷反應組件結合蛋白)。這一過程是新基因表達所必須經過的步驟，與長期的增效作用有關，同時也是記憶形成中非常重要的步驟。異氟烷可以影響記憶，因此我們想測試下它是否會干涉CaM易位至神經細胞細胞核的過程以及減弱P-CREB的合成作用。

方法：從人神經母細胞系培養出SH-SY5Y細胞，在KCl作用下去極化。使用西式印跡法、酶聯免疫吸附試驗和免疫細胞化學的方法檢測CREB的磷酸化作用。在去極化後經過分次的溶解離心檢測出CaM從胞質至胞核的易位的數量。CaM通過免疫細胞化學的方法定位，西式印跡和顯像法定量。在KCl去極化之前和過程中，將細胞分別暴露在異氟烷、異氟烷複合Bay K8644、尼群地平以及Ω-芋螺毒素GVIa中。

結果：細胞經KCl去極化後P-CREB的數量增加，峰值出現在去極化開始後30秒。尼群地平對這一過程具有抑制作用，Ω-芋螺毒素沒有明顯影響，異氟烷則是具有劑量依賴性作用。經L型鈣離子通道激動劑Bay K 8644預處理後，可以減弱異氟烷對P-CREB的抑制作用。經KCl去極化後CaM開始出現在細胞核內。尼群地平抑制CaM的易位，而異氟烷減弱這一作用，Bay K 8644預處理可以減輕異氟烷對CaM易位至細胞核的抑制作用。

結論：我們的資料顯示異氟烷可以抑制CaM的易位過程和P-CREB的形成。這很可能是由於異氟烷抑制了L型鈣離子通道對鈣離子的通透性。

（黃劍譯 薛張綱校）

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.

通過小兒中心靜脈注入系統對藥物輸注動力學的分析：量化達到設定劑量的延遲

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：小兒患者在重症監護病房和手術室經常需通過中心靜脈導管連續注射藥物。本研究通過一個標準小兒中心靜脈輸液系統的實驗模型設計給藥方案。
方法：評估標準的小兒 8 釐米，4 - f 雙腔導管給藥。一個注射泵通過一個三通開關連接導管的一側注射生理鹽水作爲流體載體。通過三通開關的另一個介面，第二個注射泵以 0.5ml/h 的恒定速率注入模型藥物亞甲基藍，並收集每分鐘的輸注量進行定量分析。我們 2ml/h 和 12ml/h 的總流量模擬 3kg 嬰兒的給藥量，並通過用模型藥灌注三通管道來模擬藥物注射停止後恢復，而不予灌注可模擬一個新的輸液。藥物泵裝置起始的表現被測量以估計對灌注起始時的貢獻。
結果：啓動一個新的藥物輸注模型時，導管末端藥物達到試驗設定濃度的時間通過達到靶濃度的一半的時間來測量。藥物輸注啓動時在總流量低時(t(50) = 23.5 +/- 2.1 min)要遠慢于總流量高時(t(50) = 15.7 +/- 2.9 min)。而預先用亞甲基藍灌注輸液管道可顯著縮短達到靶濃度的時間(low flow t(50) = 12.7 +/- 0.6 min, high flow t(50) = 5.2 +/- 0.8 min)。停止藥物泵後使用高的載體流速時雙腔導管末端藥物停止的時間(t(50) = 3 +/- 0.5 min)相比於使用低載體流速時(t(50) = 11.6 +/- 0.8 min)顯著縮短。藥物泵系統的啓動性能造成輸注啓動延遲。
結論：當前的小兒科護理設置的灌注技術在給與患者預期藥物劑量時可導致有意義的，未明確的，有潛在危險的延誤。總流量率，灌注輸液系統，流體路徑固定容積，以及輸液泵系統的啓動性能可能延遲達到藥物輸注靶速率。
（李瑩譯 薛張剛校）

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.

鎮痛分娩過程中麻醉相關的併發症的流行病學研究

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

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背景：鎮痛和分娩過程中與麻醉相關的併發症的流行病學資料對於評估產科麻醉的安全性及有效性是非常有必要的，但是這方面的資料是缺乏的。在我們的這項研究中，我們對紐約醫院中產婦進行大規模的研究，旨在調查麻醉相關的併發症的流行病學情況。

方法：使用醫療保健的費用，並應用國家住院病人資料庫資料，我們收集了紐約各大醫院從 2002 年至 2005 年產科病人的資料。根據第九版國際疾病分類法修訂版，我們收集了分娩鎮痛過程中出現麻醉相關併發症的病人資訊。我們統計了麻醉相關併發症發生的人口特徵及臨床特徵。並使用多因素回歸分析的方法評估了麻醉相關併發症的危險因素。

結果：總共調查了 957,471 名產婦，其中 4438 (0.46%)名至少出現了一項麻醉相關併發症。 其中併發症主要是椎管內麻醉相關的併發症（占 55%），其次是系統性症狀（占 43%），藥物過量及藥物副作用占 2%。多因素回歸分析揭示了五個麻醉相關併發症的危險因素：剖宮產(優勢比[OR] 2.51, 95%置信區間[CI] 2.36-2.68),農村地區(OR 1.33, 95% CI 1.21-1.46), Charlson-Deyo 同病指數≥1 (OR 1.47, 95% CI 1.28-1.69), 高加索人種(OR 1.37, 95% CI 1.24-1.52), 及擇期入院(OR 1.10, 95% CI 1.03-1.18)。麻醉相關併發症與平均住院天數增加一天相關（3.89 ± 3.69 [均數±標準差]天 vs 2.92 ± 2.38 天，分娩過程中沒有麻醉相關併發症,$P < 0.0001$） ，增加了孕產婦死亡率 22 倍(OR 22.26, 95% CI 11.20-44.24)。

結論：儘管分娩鎮痛過程中麻醉相關併發症的發生率較低，但是仍應引起重視，特別是那些行剖宮產的，生活在農村或有合併症的產婦。

（陳珺珺譯 薛張綱校）

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.

院外心肺復蘇的預測模型

A Prediction Model for Out-of-Hospital Cardiopulmonary Resuscitation

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背景：我們創建了一個預測模型來決定是否省去徒然的心肺復蘇從而節省資源。
方法：在這個析因分析中，我們評價成功心肺復蘇後神經恢復的預測參數。原始試驗爲單盲、隨機、前瞻性、對照、多中心臨床試驗。
結果：我們入組了 1166 例院前發生心跳驟停並予以高級生命支持的患者。其中 786 例患者（67.4%）在現場死亡，380 例患者（32.6%）被送往醫院。265 例患者（22.7%）在醫院死亡。115 例患者（9.8%）出院，其中的 92 例能被隨訪到。出院的 54%的患者（92 例中的 50 例）腦功能恢復良好。46%的病人爲意識不清或留有嚴重殘疾。
心室纖顫更有可能在神經功能恢復好的病人中發生（42/50=84.0%），而神經功能恢復差的病人更有可能發生了心跳驟停（9/42=21.4%）。運用 logistic 回歸分析，用一個評分來預測死亡的可能性。預測院內死亡其敏感度爲 99.8%（953/955），但其特異性爲 2.9%（3/104;閾值 0.5）。預測入院直到出院的生存其敏感度爲 99%（103/104），但其特異度爲 8%（72/955;閾值 0.99）。受試者工作曲線顯示可信區間爲 95%時曲線下面積爲 0.795(0.751-0.839)。
結論：對於院外心跳驟停的患者，該參數不能準確預測院內的生存。
（姚敏敏譯 薛張綱校）

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.

在擇期剖宮產患者中比較預先加用膠體和腰麻同時加用膠體的效果：一項隨機試驗

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

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背景：剖宮產手術中腰麻後低血壓十分常見。先前的研究顯示同時加用晶體液（在鞘內注射同時快速補液）比常規的腰麻前預先加入晶體液（在鞘內注射前補液）在預防低血壓方面更爲優越。預先加用膠體能獲得中心血容量的持久增加。我們假設：和晶體液比較，預先加用膠體可能比腰麻同時加膠體在減少腰麻導致的低血壓發生率上更爲有效。
方法：在這個雙盲研究中，178 個病人被隨機分配，分別在腰麻前 15-20 分鐘內給予 500ml 羥乙基澱粉（n=90）或者在腦脊液注藥的同時快速補入等量液體（n=88）。當收縮壓下降少於患者本身血壓的 80%和小於 100mm Hg，或者較輕微低血壓伴隨噁心嘔吐或眩暈時給予血管加壓素（麻黃堿或苯腎上腺素）。最主要的結果是低血壓的發生率（定義爲至少一個劑量血管加壓素的使用）。
結果：低血壓的發生率在兩組中間沒有明顯區別（預先加膠體組 68%，同時加膠體組 75%，兩者之間區別的 95%置信區間爲－6%－20%; P = 0.28），麻黃堿和苯腎上腺素的用量，和血管加壓素單位劑量的數量。嚴重低血壓（收縮壓小於 80 mm Hg）的發生率爲 16%在預先加膠體組，22%在同時加膠體組(P = 0.30)。兩者之間在噁心嘔吐發生率沒有區別。
結論：女性患者中在腰麻前給與膠體和在腰麻同時給與膠體，低血壓的發生率沒有區別。兩種方式作爲一項單獨的干預措施在預防低血壓方面都沒有效果。
（俞佳譯 薛張綱校）
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.

兔子中七氟醚預處理誘導通過細胞外信號調節激酶的啓動介導快速缺血耐受來對抗脊髓缺血再灌注損傷、

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

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Anesth Analg 2009 109: 1263-1272.
背景：七氟醚預處理對脊髓缺血/再灌注（I / R）保護作用是不清楚的。我們設計這個研究，調查是否七氟醚預處理可在短暫兔脊髓缺血模型中誘導快速缺血耐受，以及細胞外信號調節激酶（ERK）如何作用的。
方法：新西蘭白色雄性家兔隨機分爲三組來測試七氟醚預處理是否誘導快速缺血耐受。七氟醚組動物預處理吸入 3.7％七氟醚（1.0 最低肺泡麻醉濃度）混合 96％氧氣 30 分鐘，而氧氣組動物僅控制性吸入 96％氧氣 30 分鐘。假手術組接受了同樣的麻醉和手術的準備，但沒有預處理或脊髓的缺血/再灌注。評估 ERK 的啓動在七氟醚預處理中作用，兔隨機分爲 4 組。 U0126， ERK 的抑制劑，於預處理前 20 分鐘注入 U012+ O2 組和 U0126 +七氟醚組。同時在媒介物+ O2 組和媒介物+七氟醚組中靜脈注入二甲基亞砜。預處理後 1 小時，動物通過腹主動脈阻斷來導致脊髓

的 I / R。48 小時後所有動物通過再修改 Tarlov 評分標準，以及脊髓節段（腰）的組織病理學檢查，TUNEL 染色和免疫印跡磷- ERK1 / 2 來評估。
結果：七氟醚組動物比氧組有更高的神經系統得分和更多的正常運動神經元（各個比較均 P<0.01）。與媒介物+七氟醚組相比，U0126 +七氟醚組神經檢查結果更差，有功能的神經元更少，神經元凋亡更多，更顯著的 ERK1 / 2 的磷酸化的降低（各個比較均 P<0.01）。媒介物+ O2 組，U0126 + O2 組和 U0126 +七氟醚組之間沒有顯著差異。
結論：本研究表明，七氟醚預處理在實驗兔中誘導快速耐受脊髓缺血/再灌注且耐受可能是通過 ERK 的啓動介導的。這些資料表明，七氟醚預處理可能爲保護圍手術期脊髓缺血/再灌注提供新的處理的方法。
（張玥琪譯，薛張綱校）
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.

弗羅因德式完全佐劑誘發椎間盤炎的椎間盤源性下腰痛的動物模型

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

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背景： 雖然建立了大量的椎間盤(IVD)退變相關的下腰痛的動物模型，但是這些資料資料不足以對疼痛得出明確的結論。此研究的目的在於確定用弗羅因德式完全佐劑(CFA)注射入大鼠脊柱模擬人類椎間盤性疼痛的動物模型的可靠程度.
方法:向成年大鼠 L5-6 椎間盤內注射 10 微升 CFA 後,應用行爲學,組織學和生物化學方法研究椎間盤退變和疼痛的關係.通過分析一系列組織學變化觀察椎間盤退行性變。應用免疫組化，PCR 即時聚合酶鏈反應測定降鈣素基因相關多肽(CGRP)、前列腺素 E(PGE)和誘導型一氧化氮合酶(iNOS)，作爲疼痛發生的依據。此外椎間盤內 CGRP 免疫活性作爲神經內生長的間接證據。
結果：術後 7 周 CFA 組中可觀察到後續退變反應加強。組織學分析發現椎間盤退變但不伴隨周圍結構如神經根的破壞。椎間盤內注射 CFA 後 CGRP-免疫染色分析發現雙側後角和椎間盤免疫反應呈陽性。2 周和 4 周後脊髓背根神經節（DRG）中 CGRPmRNA 表達增加。2 周後 PGE 和 iNOS mRNA 的表達也顯著增加。CGRP 在異常疼痛的大鼠體內表達顯著高於在非異常疼痛大鼠體內的表達。
結論：椎間盤內 CFA 注射所誘導的慢性椎間盤退變導致的異常疼痛可能與疼痛性行爲和疼痛介質表達相關。在動物模型中 CGRP，PEG 和 iNOS 表達增加也與椎間盤和神經通路的信號傳導相關。本動物模型對於今後研究脊柱相關疼痛的病理生理及新的治療方法有意義。
（張釗譯 薛張綱校）
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.

大鼠鞘內注射嗎啡與馬普替林的協同效應

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

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背景:抗抑鬱藥物具有抑制去甲腎上腺素和/或 5-羥色胺重攝取的作用，常與阿片內聯合用於治療慢性疼痛。其增加鎮痛效果的機制尚未明確。我們使用大鼠熱撤離試驗比較鞘內注射嗎啡與非選擇性抗抑鬱藥阿米替林或選擇性抗抑鬱藥馬普替林、西他羅侖聯合使用時減弱傷害感受的效應。我們也觀察這些藥物間相互作用的可能機制。

方法：用七氟烷麻醉雄性 Wistar 大鼠，並分別向鞘內注射嗎啡、抗抑鬱藥或鹽水。給藥前後用熱撤離試驗評估減弱傷害感受效應。用最大可能效應百分比(MPE)表示撤離反應發生時間。爲了研究反應機制，所有動物均用非選擇性 α 2 受體阻滯劑育亨賓和非選擇性阿片類阻滯劑納洛酮預處理。同時用等輻射分析法評估固定比例注射馬普替林和嗎啡的藥理學相互作用。

結果：單一鞘內注射嗎啡 2 μg、阿米替林 125 μg、西他羅侖 144 μg、馬普替林 1.25 μg 分別產生 51.6% ± 8.9%, 10.3% ± 3.2%, 33.8% ± 5.2%和 48.5% ± 9.2% 的 MPE。嗎啡與阿米替林合用時減弱傷害感受效應增強至 91.3% ± 4.6% MPE，與馬普替林合用增強至 86.9% ± 9.2% MPE，而與西他羅侖合用無增強效果(40.6% ± 4.6% MPE)。與馬普替林聯合使用時嗎啡的減弱傷害感受時間增加四倍，由 120 分鐘增加至 480 分鐘，這一效應可被α-2 受體抑制劑育亨賓和阿片類 μ 受體拮抗劑納絡酮預處理逆轉。等輻射分析法證明了嗎啡和馬普替林的協調作用。

結論：選擇性去甲腎上腺素重攝取抑制劑通過α-2 和阿片類受體能明顯增加嗎啡減弱傷害性感受的強度和時間。選擇性 5-羥色胺抑制劑西他羅侖與嗎啡無此相互作用。

（朱蘭芳譯 薛張綱校）

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.

利多卡因中加入對乙醯氨基酚行靜脈區域阻滯的鎮痛效果

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

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背景：在這項研究中，我們評估了在利多卡因中加入對乙醯氨基酚用於靜脈區域阻滯時，感覺及運動阻滯的起效時間，止血帶疼痛和術後鎮痛效果。
方法：60 名行手部手術的病人隨機、盲法分爲 3 組。三組病人行靜脈區域鎮痛，均給予利多卡因 3 mg/kg ，用生理鹽水稀釋至 40ml。組 1 靜脈給予利多卡因加生理鹽水；組 2 給予利多卡因加 300mg 對乙醯氨基酚，生理鹽水稀釋至 40ml；組 3 給予利多卡因加對乙醯氨基酚 300mg。分別評估了手術期間感覺或運動阻滯起效時間、止血帶疼痛和鎮痛藥物使用情況。止血帶放氣後 1，2，4，6，12 和 24 小時

進行 VAS 評分，第一次使用鎮痛藥的時間，最初 24 小時鎮痛藥物使用的總量及副作用的情況。

結果：第二組運動阻滯起效時間較短，運動感覺恢復時間較長（$P < 0.05$）。組 2 手術期間 20, 30 和 40 分鐘 VAS 評分較低($P < 0.05$)。手術期間芬太尼使用總量分別爲 78 ± 12、58 ± 14、78 ± 11 μg；因止血帶疼痛需要使用芬太尼的病人數量分別爲 13 人、3 人和 9 人，組 2 人數明顯較少($P < 0.05$)。組 2 術後使用芬太尼的時間間隔較長（分別爲 15 ± 6, 25 ± 5, 15 ± 4 分鐘）($P < 0.05$)。組 2 手術麻醉的品質較高($P < 0.05$)。術後 VAS 評分和開始使用鎮痛藥物的時間在各組中相似；組 2 使用雙氯芬酸鈉的總量較少($P < 0.05$)。

結論：靜脈區域鎮痛時，利多卡因中加入對乙醯氨基酚可以降低止血帶疼痛，提高麻醉的品質，減少術後鎮痛藥物使用的量。

（陳珺珺譯 薛張綱校）

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.

體外迴圈對腦的微栓子數量和冠脈搭橋術後認知障礙發生率的影響

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：術後認知障礙（POCD）可能是冠脈搭橋術（CABG）後使患者虛弱的併發症。體外迴圈（CPB）中的腦微栓子被認爲是 POCD 一個重要發病因素。在這個研究中，我們驗證在中國人群中不進行 CPB 的手術是否可以減少腦栓子的數量和 CABG 術後 POCD 的發生率。
方法：227 例患者被選入這個前瞻性佇列研究。59 例患者進行了 CPB 下的 CABG 手術，168 例患者在不運用 CPB 下進行手術。腦微栓子通過雙側大腦中動脈經顱多普勒超聲檢查來持續檢測。一套神經心理學測試，包括七個試驗九個方面，在術前、術後 1 周和術後 3 月進行。POCD 用國際 POCD1 研究的定義來進行定義。
結果：在 CPB 下手術患者的腦微栓子總數量的中位數爲 430（範圍：155－2088），不進行 CPB 的患者爲 2（0－66）(P < 0.001)。術後 1 周(55.2%或 32/58[95%可信區間: 41.5%–68.3%]對 47.0%或 78/166[39.2%–54.9%], *P* = 0.283)和 3 月的 POCD 發生率(6.4%或 3/47 [1.3%–17.5%] 對 13.1%或 16/122[7.7%–20.4%], *P* = 0.214)，進行 CPB 和不進行 CPB 手術的患者之間沒有差異。年齡的增加和較短的術後住院時間與術後 1 周的認知障礙獨立相關。年齡增加和有糖尿病史與術後 3 月認知障礙獨立相關。CPB 或腦微栓子與 POCD 的發生沒有顯著的相關性。
結論：在中國人群中，CABG 手術中不使用 CPB 能顯著減少腦微栓子的數量，但不能減少術後 1 周和 3 月 POCD 的發生。CPB 和腦微栓子都不是與 POCD 獨立相關的風險因素。
（唐亮 譯 馬皓琳 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.

惡性高熱、共存失調和酶病：風險和管理選擇

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

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已觀察到患多種綜合症、酶病和兩者共存的病人出現與診斷惡性高熱一致的臨床症狀和異常實驗室試驗，因此提高了有相同診斷的其他病人的病因相關的可能性和已增高的圍術期風險。在本綜述中，我們檢查了被其他醫生認定爲潛在易患惡性高熱患者的可得到的已發表系列、病例報導和攣縮試驗的結果。對多數情況下，與惡性高熱易感性有病因相關的證據是很少的。本綜述總結了當支持或不支持相關性的證據不確定時的臨床管理的建議。
（王宏翻譯，馬皓琳，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.

神經肌肉阻滯不同程度地影響近最低肺泡有效濃度麻醉下的體動抑制和皮層活性

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

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背景：麻醉誘導的體動抑制和皮層活動的抑制是由解剖上分開但相互作用的中樞神經系統區域所控制的。因此，與抑制傷害刺激引起的體動反應相比，需要更大濃度的吸入麻醉藥以抑制皮層的活動。我們觀察了在近最低肺泡有效濃度的地氟烷麻醉過程中有傷害性刺激時，神經肌肉阻滯（NMB）產生的傳入輸入減弱對體動反應和用腦電雙頻指數（BIS）評估的皮層活動的影響。

方法：在 24 名健康的志願者中，用上下調節的方法和單獨的前臂方法評價 NMB 對用於體動抑制的地氟烷有效呼氣末肺泡濃度($EtDes_{50}$ 或 $MAC_{tetanus}$) 中位數的影響。 每個志願者以隨機次序連續地注射鹽水、美維庫銨和司可林，而在注射同樣藥物的前一個志願者的體動反應的基礎上來測定用不同藥物時的 EtDes 濃度。用非線性混合反應模型來評估 NMB 對基礎狀態下和有害刺激後 BIS 與 EtDes 濃度的對比關係的影響，而前額的 EMG(EMG_{BIS})對 BIS 的影響同樣也作爲模型中的一個變數。同時比較了應用不同藥物時有害刺激引起的心血管反應。

結果：司可林和美維庫銨顯著減少 $MAC_{tetanus}$(95%可信區間)。其中生理鹽水組爲 5.00% (4.85%–5.13%),而司可林和美維庫銨分別爲 4.05% (3.81%–4.29%) 和 3.84% (3.60%–4.08%)。無論應用何種藥物，傷害性刺激均明顯地增加了 BIS 的反應，儘管是最低限度的。司可林增加 BIS 值而不依賴於其對 EMG_{BIS}的影響。注射司可林增加了心血管系統的活性。有趣的是，儘管美維庫銨減弱了心血管對傷害刺激的反應，用 BIS 測定的皮層反應並沒有變化。

結論：在接近於 MAC 的麻醉期間，司可林和美維庫銨增加了對體動反應的抑制。所有用藥均會引起傷害性刺激產生的小但卻顯著的 BIS 值增加。而司可林增加 BIS 值並不依賴於傷害性刺激或 EMG_{BIS}。美維庫銨抑制了對傷害性刺激的自主神經反應。

（黃麗娜 譯 馬皓琳 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.

短暫接觸七氟烷後，幹細胞樣人內皮祖細胞顯示集落形成能力增強：吸入麻醉藥對血管生成細胞的預處理

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：內皮祖細胞在組織修復中起關鍵作用，因此在“再生醫學”中被用於細胞的替代治療。我們檢驗麻醉藥七氟烷是否會調製這些血管生成細胞的生長或動員。
方法：在離體模型中，從健康捐獻者的外周血中分離出來的單核細胞用七氟烷預處理（2 vol%，30 min 3 次，間隔予 30 min 空氣）。9 天后在培養中測定集落形成單位，並與同期配對的未處理對照進行比較。使用磁性細胞分選法，從人臍帶血中富集 CD133+/CD34+的內皮祖細胞，並用即時逆轉錄多聚酶鏈式反應對七氟烷處理或未處理細胞內血管內皮生長因數（VEGF）、VEGFR2（KDR）、粒細胞集落刺激因數（G-CSF）、STAT3、c-kit 以及 CXCR4 的表達進行測定。在一個採用交叉試驗設計的志願者研究中，我們使用外周血樣流式細胞儀，檢驗吸入七氟烷（呼氣

末濃度<1 vol%）是否動員內皮祖細胞從骨髓生態位區進入迴圈。同時測定血漿中 VEGF 和 G-CSF 的水準。
結果：單核細胞體外接觸七氟烷增強 CD133+/CD34+臍帶血細胞的集落形成能力並增加 VEGF mRNA 水準($P = 0.017$)。健康志願者吸入七氟烷並不改變迴圈中 CD133+/CD34+ 或 KDR+/CD34+內皮祖細胞的數量，但增加了集落形成單位的數量($P = 0.034$)，而血漿中 VEGF 和 G-CSF 的水準保持不變。
結論：七氟烷預處理促進了幹細胞樣人類內皮祖細胞的生長和增殖，因此可能被用於促進圍手術期血管癒合並支持細胞替代治療。
（黃施偉 譯，馬皓琳 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.

多輸注治療期間多路輸注裝置對體外給藥的影響

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

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背景：多路輸注裝置能夠同時輸注多種藥物，但也可能引起各投藥通路相互干擾，其原因是有效藥物的給藥速率的個體差異較大。我們在本研究中試圖闡明在多輸注治療期間多路輸注裝置屬性（死腔容量和抗反流閥[ARV]）對投藥的影響。
方法：對長度、死腔量以及有無 ARV 方面不同的輸注裝置進行評估。通過不同通路點同時輸注 3 種藥物，並通過紫外線分光亮度計分析流出液來得到其藥物濃度。評估以下參數來比較不同的輸注配置：(1)每單位時間輸入病人體內的藥物量、(2)輸注穩態（品質流速平臺）期間每單位時間輸入病人體內的平均藥量及(3) 流量變化效能——實驗暫態品質流率曲線下面積與對應的理論暫態品質流率曲線下面積的比值。
結果：無論流速如何變化，與高死腔量的輸注裝置（死腔量等於 6.16 mL 時流量變化效能 5.6% ± 8.2%）相比，低死腔量的輸注裝置明顯具有較高的流量變化效能（死腔量等於 0.046 mL 時開始輸注後 5 min 爲 53.0% ± 15.4%）。即使存在較大死腔量，具有 ARV 的輸注裝置明顯抬高品質流率平臺（從沒有 ARV 時理論平臺的 92.4% 增加至有 ARV 時的 99.3%）。
結論：多輸注治療引起投藥干擾（輸注滯後時間，返流，單次注射量）。採用非常低死腔量和具有 ARV 的輸注裝置可以降低這種干擾。
(江繼宏 譯 馬皓琳 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.

核心肌病與惡性高熱的風險

Core Myopathies and Risk of Malignant Hyperthermia

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在本文中我們分析了核心肌病，核心肌病與惡性高熱之間的聯繫已經有人提出了。我們討論了核心肌病的臨床特徵、基礎的遺傳缺陷、對細胞鈣代謝的後續影響以及對惡性高熱觸發的離體肌反應。我們詳細描述了中央核疾病、多小核疾病和線形體小枝肌病。我們還依據疾病侵犯的蛋白質對疾病加以分類，並分析了惡性高熱的風險，其中當鈣傳導蛋白質受侵犯時惡性高熱的風險性更高或者在理論上可能。
（薑旭暉 譯，馬皓琳 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.

開顱手術中手術室工作人員暴露於七氟醚的相關情況

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

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背景：這個研究中，我們最初的目的是探討開顱腫瘤切除術中，對於從手術部位散發的揮發性麻醉藥七氟醚的接觸量，神經外科醫生是否大於麻醉醫生。
方法：首先，我們測定了 35 例顱內腫瘤切除術從硬腦膜打開到關閉，從手術部位散發的七氟醚量。揮發性麻醉藥吸收劑被放置在三個測定部位：1)外科醫生呼吸區域、2)麻醉醫生呼吸區域、以及 3)離手術部位最遠的手術室角落。在第二個採樣系列中(包括 16 名病人)，第一個採樣系列中被放置在手術室最遠角落的吸收劑這次

被放置在病人的嘴邊 (5cm 以內)。被吸收劑吸附的七氟醚用色譜法由一名獨立的藥劑師進行定量測定。
結果：外科醫生呼吸區域吸收劑吸附的七氟醚量(0.24 ± 0.04 ppm)明顯低於麻醉醫生呼吸區域(1.40 ± 0.37 ppm)，且與手術室最遠角落吸收劑吸附的七氟醚量(0.25 ± 0.07 ppm)相當。吸收劑吸附的七氟醚量與手術切口大小沒有相關性，即使調整了手術時間這一變數，兩者也沒有相關性。在第二個採樣系列中，病人嘴邊的吸收劑吸附了最高量的七氟醚(1.54 ± 0.55 ppm)，其次是麻醉醫生呼吸區域(1.14 ± 0.43 ppm)和外科醫生呼吸區域(0.15 ± 0.05 ppm)。
結論：外科醫生呼吸區域最接近的手術部位並不是增加七氟醚暴露的來源。我們觀察到麻醉醫生在手術室環境中暴露於七氟醚更多，這有必要深入研究。
（張瑩譯 馬皓琳 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.

性別對咪達唑侖全身麻醉下正常受試者上呼吸道阻塞引起的代償性神經肌肉反應的影響

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：在睡眠和麻醉時解剖學改變（機械性能）或神經控制受擾（補償性神經肌肉反應）都可能危及上呼吸道通暢。麻醉時上呼吸道阻塞的病理生理學在男性和女性可能不同。近來，我們報導通過在咪達唑侖鎮靜時測量被動臨界閉合壓(P_{CRIT})和上游阻力(R_{US})，上呼吸道機械性能與自然非快眼動相睡眠相當。在這個研究中，我們比較了性別對咪達唑侖全身麻醉下上呼吸道阻塞引起的代償性神經肌肉反應的影響。
方法：研究 32 例受試者（14 名男性，18 名女性）。在咪達唑侖麻醉過程中建立壓力-流量關係來評估 P_{CRIT} 和 R_{US}。咪達唑侖麻醉誘導用咪達唑侖首量 0.07–0.08 mg/kg bolus，維持以 0.3–0.4 $\mu g \cdot kg^{-1} \cdot min^{-1}$ 的速度持續輸注，用 Ramsay（5 級）和 OAA/S（2 級）評分評估麻醉水準。監測多項睡眠生理檢查和血液動力學變數，同時記錄鼻腔壓力（通過面罩）、吸氣氣流（通過呼吸速度描記器）和頦舌肌肌電圖(EMG_{GG})。在 EMG_{GG} 衰減的被動條件下和 EMG_{GG} 增加的主動條件下得到 P_{CRIT}（分別爲被動 P_{CRIT} 和主動 P_{CRIT}）。在每例受試者計算主動 P_{CRIT} 和被動 P_{CRIT} 間的差($\Delta P_{CRIT\ P-A}$)來測定代償性神經肌肉反應。
結果：$\Delta P_{CRIT\ A-P}$ 在女性明顯大於男性(女性 4.6 ± 2.8 cm H_2O ，男性 2.2 ± 1.7 cm H_2O, $P < 0.01$)，提示對不依賴於覺醒狀態的上呼吸道阻塞有更大的代償性神經肌肉反應。
結論：我們證明咪達唑侖麻醉中對上呼吸道阻塞的不依賴於覺醒狀態的補償性神經肌肉反應在女性中部分維持，性別可能是麻醉中代償性反應強度的決定性因素。
（朱 慧譯 馬皓琳 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.

從實驗室及理論角度分析安氟醚的皮質電效應

The Electrocortical Effects of Enflurane: Experiment and Theory

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背景：高濃度的安氟醚能引起典型的腦電圖：包括週期性抑制與大而短的突發性癲癇樣放電(PEDs)交替。在本研究中，我們比較了這種活性的理論性電腦模型與從麻醉大鼠中獲得的真實局部場電位(LFP)資料。
方法：將 8 x 8 的高密度電極植入視覺皮層後，記錄大鼠在 0.5、1.0、1.5 及 2.0 倍最小肺泡麻醉濃度(MAC)的安氟醚麻醉時 LFP 及多元峰活性。將來自於新皮層動力學平均場模型的電腦類比與這些記錄進行比較。通過延長抑制性突觸後電位(IPSP)衰減時間常數模擬增加安氟醚濃度所產生的神經元效應。與新皮層的激發率相反，我們調整了興奮性突觸後電位(EPSP)的振幅。
結果：在麻醉大鼠中，安氟醚濃度的持續增加會始終引起 LFP 記錄中表現出抑制波型(>1.5 MAC)。多元鋒電位的平均速率從 2.54/s (0.5 MAC) 下降到 0.19/s (2.0 MAC)。在高 MAC 時，大多數的多元動作電位事件變得與 PED 同步。在理論模型中，IPSP 衰減時間的延長及活性依賴 EPSP 的調整所導致的輸出結果與從實驗資料中所獲得的形態相似。通過分析方程的本征值來測定模型中節律性暴發樣活動的傾向。
結論：使用新皮層動力的平均場理論來複製安氟醚麻醉大鼠 LFP 中所觀察到的 PED 圖型是可能的。該圖型需要作一些綜合調整：適當增加 IPSP 下的總面積，延長 IPSP 衰減時間，及對 EPSP 的振幅進行活性依賴的調整。
（裘毅敏譯，馬皓琳、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.

評估圍手術期使用普加巴林對預防和減輕腹腔鏡下膽囊切除術後肩痛的效果

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

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腹腔鏡手術後常發生肩痛（PLSP）。在這個安慰劑對照的研究中，我們評估了圍手術期用普加巴林 300 毫克間隔 12 h 給予兩個劑量來預防和減輕腹腔鏡下膽囊切除手術後 PLSP 的療效。術後 48 小時評價 PLSP 的發生率和嚴重度、術後鎮痛的需要及副作用。在兩個組中，PLSP 的總體發生率沒有明顯差異，且 PLSP 疼痛評分、第一次用鎮痛藥的時間，和累計酮咯酸消耗量在每個時間點是相似的。然而，普加巴林組在術後 2 小時過度鎮靜的發生率較高。
（彭中美 譯 馬皓琳 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.

小鼠坐骨神經中細胞外信號調節激酶啓動促進坐骨神經部分結紮後神經性疼痛

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種類型的疼痛中具有重要作用。但是對磷酸化細胞外信號調節激酶（pERK）在受傷的周圍神經局部的具體作用的認識還很少。在本文中，我們研究了在小鼠受損的坐骨神經損傷中 pERK 是否促進由坐骨神經部分結紮（PSL）誘發的神經性疼痛。
方法：小鼠行坐骨神經部分結紮；通過蛋白質印跡法和免疫組織化學方法測定坐骨神經中的 pERK1/2 (p44/42)。在 PSL 前 30min 於神經內注射 U0126（一種 ERK 激酶抑制劑）20 nmol/2 μL，在 PSL 後 1 天于神經周圍注射 20 nmol/10 μL。PSL 誘發的熱痛覺過敏和觸覺異常性疼痛分別由熱板縮爪試驗和 von Frey 試驗來測定。
結果：通過 Western blot 檢測，在假手術的小鼠，坐骨神經 pERK1/2 水準在第 1-14 天均穩定且與非手術者相同。在 PSL 手術後小鼠，pERK1/2 在 PSL 後 1 天顯著增高並且持續至第 3 天。通過免疫組織化學檢測，與假手術的坐骨神經相比， PSL 手術後的坐骨神經 pERK1/2 免疫反應性在 PSL 後 1 天顯著增高。通過雙重免疫染色顯示，在 PSL 後 1 天，坐骨神經中 pERK1/2 免疫反應性增強與許旺細胞標記物膠質纖維酸性蛋白（GFAP）共存，而非巨噬細胞的標記物 F4/80。在使用 U0126 處理能顯著減輕 PSL 後第 3，7 和 14 天的熱痛過敏，也能顯著減輕 PSL 後第 7 和第 14 天時的觸覺異常性疼痛。
結論：受損傷的周圍神經系統許旺細胞 ERK 啓動可能在神經性疼痛發展中具有重要作用。我們的結果提示 pERK 本身和 ERK 相關介質是治療神經性疼痛的潛在治療靶點。
（顏濤 譯， 馬皓琳 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.

脂肪乳劑可以改善從布比卡因引起的心搏驟停恢復，但對羅呱卡因或甲呱卡因引起的心搏驟停無效

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

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背景：局麻藥的心臟毒性與其親油性顯著相關。最近，已有研究顯示持續輸注脂肪乳劑是一個有希望能處理局麻藥引起的心搏驟停的方法。根據其可能的作用機制，所謂的“脂質沉積”作用可能決定於局麻藥的親油性。在這項研究中，我們探討脂質的作用是否隨著所給局麻藥的不同而不同。
方法：在離體的大鼠心臟中，分別給予等效劑量的布比卡因、羅呱卡因和甲呱卡因引起心搏驟停，然後用或不用脂肪乳劑進行心臟灌注($0.25\ mL \cdot kg^{-1} \cdot min^{-1}$)。然後，評定從開始灌注到發生第一次心臟活動恢復的時間，及到心率和心率－收縮壓乘積恢復的時間。
結果：在所有的小組中，脂肪乳劑對任何心臟活動恢復時間沒有影響。給予脂質卻可以顯著縮短布比卡因引起的心臟毒性中的心率和心率－收縮壓乘積恢復時間，但是對羅呱卡因和甲呱卡因引起的心臟毒性沒有影響。

結論：這些資料顯示脂肪乳劑對局麻藥引起的心臟毒性的影響主要決定於所給的局麻藥本身。我們得出結論，局麻藥的親油性顯著影響脂肪乳劑處理心搏驟停的有效性。
（黃佳佳譯，馬皓琳 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.

星狀神經節阻滯後分形心血管動力和壓力反射敏感性的狀態

Fractal Cardiovascular Dynamics and Baroreflex Sensitivity After Stellate Ganglion Block

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背景：研究現實星狀神經節阻滯可降低壓力反射的敏感性。本研究主要目的爲確定心率和收縮壓變異性分形動力學（自體相似波動模式的動態改變）是否均參與了星狀神經節阻滯後壓力反射敏感性降低的機制。
方法：16 名健康年輕志願者參與了本次研究。採用 1%甲呱卡因 6mL 間隔 1~1[illegible] 月對志願者行左或右星狀神經節阻滯。阻滯前、阻滯後 30、60、90 和 120 分鐘進行心率和收縮壓變異形頻譜分析。每次頻譜分析後即刻採用直立傾斜試驗評估壓力反射敏感性。
結果：經直立傾斜試驗評估，右側或左側星狀神經節阻滯後 30 分鐘壓力反射敏感性顯著降低（分別爲 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, $P < 0.01$）。反映波動自體相似性程度的分形斜率在右側或左側星狀神經節阻滯後 30 分鐘顯著增加（右星狀神經節阻滯－心率；–1.08 ± 0.30

到 –1.62 ± 0.22, $P < 0.01$；右星狀神經節阻滯－收縮壓；–1.30 ± 0.80 到 –2.40 ± 0.80, $P < 0.05$；左星狀神經節阻滯－收縮壓；–1.20 ± 0.40 到–2.13 ± 0.50, $P < 0.05$）。心率變異性分析顯示分形斜率在左星狀神經節阻滯後沒有改變。
結論：分形斜率的增加提示心率和收縮壓變異性的複雜性消失（保持複雜行爲的狀態），這是星狀神經節阻滯後壓力反射敏感性降低的機制之一。
（周雅春 譯 馬皓琳 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.