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Anesth Analg February 2010 110:630-634; published ahead of print December 2, 2009, doi:10.1213/ANE.0b013e3181c76a33

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體外迴圈中血液濾過不能降低心臟手術後的房顫發生率

Hemofiltration During Cardiopulmonary Bypass Does Not Decrease the Incidence of Atrial Fibrillation After Cardiac Surgery

William J. Mauermann, MD, Gregory A. Nuttall, MD, David J. Cook, MD, Andrew C. Hanson, BS, Darrell R. Schroeder, MS, and William C. Oliver, MD
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Anesth Analg 2010 110: 329-334.

背景：心臟手術後有20%-50%的病人發生房顫並與增加的併發症的發病率和死亡率有關。據報導，皮質類固醇可減少術後房顫的發生率，可能與它可以減輕手術及體外迴圈（CPB）引起炎症有關。作者假設，在體外迴圈中的血液濾過可能減輕炎症，可能會降低心臟手術後房顫的發生率。
方法：這一雙盲，安慰劑對照的在體外迴圈心臟手術圍術期時類固醇治療和血液濾過對術後機械通氣期間的影響因素進行評估的回顧性分析。在這項研究中，192例心臟手術患者隨機分為1到3組：對照組（安慰劑），體外迴圈中過濾血液組，類固醇治療組。記錄新發房顫的證據。房顫定義為具有心電圖證據的房顫或患者臨床診斷的房顫。
結果：192例參加試驗的患者，3例不符合實驗規定和4例慢性房顫史被排除。共有185例患者原始資料。60例（32%）有心臟手術後新發房顫。各組間房顫發生無差異（對照組21%；類固醇組41%；血液濾過組36%，P = 0.057）。年齡是房顫發生的唯一危險因素（房顫患者的平均年齡，65.4 ± 10.1歲，無房顫患者61.4 ± 11.5歲，P = 0.024）。當年齡，生活方式和存在或不存在慢性阻塞性肺疾病經多變數分析，組間差異不明顯（P = 0.108）。
結論：圍手術期皮質類固醇或體外迴圈使用血液過濾並未減少心臟手術後房顫的發生率。在常規的使用之前，圍術期類固醇應用防止術後房顫發生的有效性和安全性需更深入的評估。
(張磊 譯 陳傑 校)
BACKGROUND: Atrial fibrillation (AF) occurs in 20%–50% of patients after cardiac surgery and is associated with increased morbidity and mortality. Corticosteroids are reported to decrease the incidence of postoperative AF, presumably by attenuating inflammation caused by surgery and cardiopulmonary bypass (CPB). We hypothesized that hemofiltration during CPB, which may attenuate inflammation, might decrease the incidence of AF after cardiac surgery.
METHODS: This was a retrospective review of patients previously enrolled in a double-blind, placebo-controlled trial evaluating the effects of perioperative steroid therapy and hemofiltration during CPB on duration of postoperative mechanical ventilation. In that study, 192 patients undergoing cardiac surgery were randomized to 1 of 3 groups: controls (placebo), hemofiltration during CPB, or perioperative steroid therapy. Patient records were reviewed to determine the incidence of new onset AF defined as any electrocardiogram evidence of AF or AF diagnosed by the patients' clinicians.
RESULTS: Of the 192 enrolled patients, 3 were excluded for protocol violations and 4 were excluded for history of chronic AF. Data from 185 patients from the original study were available for review. Sixty patients (32%) had new onset AF after cardiac surgery. There was no difference among groups in the incidence of AF (control group, 21%; steroid group, 41%; hemofiltration group, 36%; *P* = 0.057 among groups). The only risk factor for the development of AF was age (mean age of patients with AF, 65.4 ± 10.1 yr vs patients without AF, 61.4 ± 11.5 yr; *P* = 0.024). When age, procedure type, and presence or absence of chronic obstructive pulmonary disease were controlled for in multivariate analysis, the difference among study groups remained nonsignificant (*P* = 0.108).

CONCLUSIONS: Perioperative corticosteroids or the use of hemofiltration during CPB did not decrease the incidence of AF after cardiac surgery. Further studies evaluating the efficacy and safety of perioperative corticosteroids for prevention of postoperative AF are warranted before their routine use can be recommended.

心臟手術病人，大劑量氨甲環酸與臨床非缺血性癲癇發作有關

High-Dose Tranexamic Acid Is Associated with Nonischemic Clinical Seizures in Cardiac Surgical Patients

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Anesth Analg 2010 110: 350-353.

背景：在 2 個獨立的中心，作者觀察到經歷重大心臟手術的病人，術後癲癇發作的發生率有顯著的增加（從 1.3%到 3.8%）。這些事件從一般臨床應用治療的角度說，與抑肽酶撤出後大劑量氨甲環酸（血栓素）應用的時間上相一致。本研究進行了回顧性分析，研究心臟手術後血栓素的使用是否和驚厥發生有關。
方法：作者對 24 例圍手術期癲癇發作的病人進行了深入的研究，對其中的 11 例進行腦電波記錄，所有病人均行神經系統評估和腦成像研究。
結果：24 例患者中的 21 例無新的腦缺血損傷的證據，3 例患者可能由於缺血性腦損傷。所有癲癇發作的病人沒有永久性的神經異常，所有的發生癲癇的 24 名病人接受了大劑量的血栓素治療（61 to 259 mg/kg），平均年齡為 69.9 歲。24 例中 21 例為心內直視術而非冠脈搭橋手術。除了一例外均在體外迴圈下手術。無明確的腦缺血，代謝，或高溫導致癲癇發作的證據。
結論：研究結果表明，在體外迴圈下心內直視術老年患者中使用高劑量血栓素與臨床易感癲癇發作有一定的聯繫。
(劉世文 譯 陳傑 校)

BACKGROUND: In 2 separate centers, we observed a notable increase in the incidence of postoperative convulsive seizures from 1.3% to 3.8% in patients having undergone major cardiac surgical procedures. These events were temporally coincident with the initial use of high-dose tranexamic acid (TXA) therapy after withdrawal of aprotinin from general clinical usage. The purpose of this review was to perform a retrospective analysis to examine whether there was a relation between TXA usage and seizures after cardiac surgery.
METHODS: An in-depth chart review was undertaken in all 24 patients who developed perioperative seizures. Electroencephalographic activity was recorded in 11 of these patients, and all patients had a formal neurological evaluation and brain imaging studies.
RESULTS: Twenty-one of the 24 patients did not have evidence of new cerebral ischemic injury, but seizures were likely due to ischemic brain injury in 3 patients. All patients with seizures did not have permanent neurological abnormalities. All 24 patients with seizures received high doses of TXA intraoperatively ranging from 61 to 259 mg/kg, had a mean age of 69.9 years, and 21 of

24 had undergone open chamber rather than coronary bypass procedures. All but one patient were managed using cardiopulmonary bypass. No evidence of brain ischemic, metabolic, or hyperthermia-induced causes for their seizures was apparent.
CONCLUSION: Our results suggest that use of high-dose TXA in older patients in conjunction with cardiopulmonary bypass and open-chamber cardiac surgery is associated with clinical seizures in susceptible patients.

骨內輸液：針對麻醉醫生的小兒領域應用的綜述

Intraosseous Infusions: A Review for the Anesthesiologist with a Focus on Pediatric Use
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骨內輸液(IO)用於不能迅速建立外周靜脈的急診重症嬰幼兒。儘管骨內輸液在上述情況下是有效的，但它在急救室外的搶救或復蘇中的應用仍有限。然而這項技術的重點在於培訓急診室或嬰幼兒重症監護室的醫護人員如何應用，因此也體現出其在圍手術期中應用的局限性。 當在手術室內不能成功建立外周靜脈通路時，中心靜脈置管、外科血管切開等方式往往能成功建立安全的血管通路。但是這些方式花費大量時間。對有條件的患兒進行骨內輸液，可望使所有的嬰幼兒麻醉醫生的工作變得快速而便捷。本文著力於介紹骨內輸液的發展，回顧骨髓腔解剖結構，探討其在圍手術期中的應用價值以及分析其併發症。
(葉樂 譯 陳傑 校)
Intraosseous (IO) access is used most frequently for emergency care of critically ill infants and children when IV access cannot be rapidly achieved. Despite its efficacy in such situations, applications outside of the emergency room or resuscitation scenario have been limited. Furthermore, although the technique is emphasized in the teaching of those caring for critically ill infants and children in the emergency room or critical care setting, there is limited emphasis on its potential use in the perioperative setting. When peripheral venous access cannot be achieved in the operating room, alternative means of securing vascular access such as central line placement or surgical cutdown are generally successful; however, these techniques may be time consuming. Anyone providing anesthesia care for infants and children may want to become facile with the use of IO infusions for selected indications. We present the history of IO infusions, review the anatomy of the bone marrow space, discuss the potential role of IO infusions in the perioperative period, and analyze its adverse effect profile.

麻醉和老年人大腦

Anesthesia and the Old Brain
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Anesth Analg 2010 110: 421-426.

圍手術期老年患者的認知功能可能出現長期的嚴重後果。在這篇特別的文章中，作者總結了麻醉因素在其中的所起作用和證據。證據這一點需進一步的研究，尤其是在人類。

(舒慧剛 譯 陳傑 校)
The perioperative period may have long-term consequences on cognitive function in the elderly patient. In this special article, we summarize the rationale and evidence that the anesthetic *per se* is a contributor. The evidence at this point is considered suggestive and further research is needed, especially in humans.

阿爾茨海默病轉基因小鼠與同窩出生的非轉基因小鼠相比有著更大的異氟醚最低肺泡有效濃度

Transgenic Alzheimer Mice Have a Larger Minimum Alveolar Anesthetic Concentration of Isoflurane than Their Nontransgenic Littermates

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Anesth Analg 2010 110: 438-441.

背景：超過 12%的人在年齡超過 65 歲後會得阿爾茨海默氏症。由於目前對於此類患者麻醉時揮發性麻醉劑的需要量沒什麼研究，研究者測定了年輕和年老的遭受阿爾茨海默氏病威脅的轉基因小鼠的異氟醚的最低肺泡有效濃度（APP23 與瑞典雙突變雜合的小鼠）。為了區分轉基因模型的非特異性因素的影響與阿爾茨海默病的影響，研究者另加了一組與實驗組相比除了沒有阿爾茨海默氏症致病基因外其他基因構成都相同的瑞典雙突變體（與 APP51/16 雜合的小鼠）。
方法：測定 60 只（每組 10 只）小鼠的 MAC 值：4 月大和 18 月大的 APP23 雜合小鼠及其未經基因改造的野生同窩小鼠，18 月大的 APP51/16 雜合小鼠及其野生同窩小鼠。異氟醚與氧氣或空氣混合後進行麻醉誘導。異氟醚的吸入濃度維持在 1.0%和 2.0%之間，並記錄傷害性刺激引起體動反應的情況。用非配對 t 檢驗的方法對 MAC 值進行比較。
結果：18 月大的 APP23 雜合小鼠的 MAC 為 1.67 ± 0.09，比野生同窩小鼠的 MAC 值高 9%（1.53 ± 0.14，P = 0.020）。而 APP51/16 雜合小鼠的 MAC 比野生同窩小鼠低（1.32 ± 0.14 ，1.48 ± 0.13，P = 0.037）。所有的野生同窩小鼠和年輕的 APP23 小鼠有著類似 MAC 值。
結論： 老年 APP23雜合小鼠異氟醚 MAC 值的增加似乎與阿爾茨海默氏症引起的機體變化有關。
(丁俊雲 譯 陳傑 校)
BACKGROUND: More than 12% of all people older than 65 yr have Alzheimer's disease. Because nothing is known about changes in demand of volatile anesthetics in this disease, we determined minimum alveolar anesthetic concentration (MAC) values of isoflurane in young and aged transgenic mice at risk of developing Alzheimer's disease (heterozygote APP23 mice with the "Swedish double mutation"). To differentiate between unspecific effects of the transgenic model and specific Alzheimer effects, we additionally evaluated MAC values in mice with the same genetic construct but without the Alzheimer's disease-causing Swedish double mutation (heterozygote APP51/16 mice).
METHODS: MAC was determined in 60 mice (10 per group): heterozygote APP23 mice and their wild type littermates at the age of 4 and 18 mo, respectively, and heterozygote APP51/16

mice and their wild type littermates at the age of 18 mo. Anesthesia was induced with isoflurane in oxygen/air. The concentration of inhaled isoflurane varied between 1.0 and 2.0 Vol%, and the motor reaction to toeclamping was recorded. Means of the MAC values were compared with an unpaired *t*-test.
RESULTS: The MAC of 18-mo-old heterozygote APP23 mice was 1.67 ± 0.09, i.e., 9% larger than the MAC of their wild type littermates (1.53 ± 0.14; $P = 0.020$). Heterozygote APP51/16 mice had a lower MAC than their wild type littermates (1.32 ± 0.14 vs 1.48 ± 0.13; $P = 0.037$). All wild type groups and young heterozygote APP23 mice had comparable MAC values.
CONCLUSIONS: The increased MAC value in aged heterozygote APP23 mice seems to be attributable to changes related to Alzheimer's disease.

揮發性芳香族麻醉藥對人類$\alpha_4\beta_2$神經元煙鹼受體的抑制作用依賴於藥物疏水性

Inhibition of Human $\alpha_4\beta_2$ Neuronal Nicotinic Acetylcholine Receptors by Volatile Aromatic Anesthetics Depends on Drug Hydrophobicity

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Anesth Analg 2010 110: 455-460.

背景：苯等揮發性芳香族化合物是全身麻醉藥，吸入後會在傷害性刺激下產生遺忘，催眠和鎮靜作用。雖然這些化合物不應用於臨床，但它們被頻繁地用於商業產品，如溶劑和家用清潔用品，並被濫用於吸入性毒品。揮發性芳香族麻醉劑是有用的藥理學工具，可在假定的全麻靶目標上探索化學結構和藥物效應之間的關係。神經元煙鹼（乙醯膽鹼）受體廣泛表達於大腦的配體門控離子通道，被認為在學習和記憶方面發揮重要作用。在這項研究中，作者驗證了這個假設：芳香族麻醉劑可逆性抑制$\alpha_4\beta_2$神經元乙醯膽鹼受體功能，作者同時試圖確定受體抑制作用與結構的相關性。
方法：電生理學技術用於量化揮發性芳香族麻醉劑，由 1 mM Ach 探出，並調整人類乙醯膽鹼受體在爪蟾卵母細胞中的表達。
結果：本研究中使用的所有的揮發性芳香族麻醉劑可逆性抑制了$\alpha_4\beta_2$乙醯膽鹼受體，從 0.00091 大氣壓下的 1,2 二氟苯到 0.045 大氣壓下的六氟苯的 IC50 值。除六氟苯外，這些化合物 IC_{50} 值低於最低有效肺泡濃度。抑制效應與化合物的 cation-π結合能力並不相關（R2 的= 0.48，P = 0.059）。然而，抑制強度與辛醇/氣分配係數有良好的相關性（R2 = 0.87，P = 0.0008）。
結論：揮發性芳香族麻醉藥能強效及可逆性地抑制人類$\alpha_4\beta_2$神經元乙醯膽鹼受體。這種抑制作用可能在產生遺忘方面起著作用。相對於 N -甲基- D -天冬氨酸受體，芳香族麻醉劑對$\alpha_4\beta_2$神經元乙醯膽鹼受體的抑制強度似乎依賴於藥物的疏水性，而不是靜電性能。這意味著，揮發性芳香族麻醉藥與$\alpha_4\beta_2$神經元乙醯膽鹼受體結合位點在性質上是疏水性，這與 N -甲基- D -天冬氨酸受體結合位點性質不同。
(唐穎 譯 陳傑 校)

BACKGROUND: Volatile aromatic compounds such as benzene are general anesthetics that cause amnesia, hypnosis, and immobility in response to noxious stimuli when inhaled. Although these compounds are not used clinically, they are frequently found in commercial items such as solvents and household cleaning products and are abused as inhalant drugs. Volatile aromatic

anesthetics are useful pharmacological tools for probing the relationship between chemical structure and drug activity at putative general anesthetic targets. Neuronal nicotinic acetylcholine (nACh) receptors are ligand-gated ion channels widely expressed in the brain, which are thought to play important roles in learning and memory. In this study, we tested the hypothesis that aromatic anesthetics reversibly inhibit $\alpha_4\beta_2$ neuronal nACh receptor function and sought to determine the structural correlates of receptor inhibition.
METHODS: Electrophysiological techniques were used to quantify the effects of 8 volatile aromatic anesthetics on currents elicited by 1 mM ACh and mediated by human $\alpha_4\beta_2$ nACh receptors expressed in *Xenopus* oocytes.
RESULTS: All of the volatile aromatic anesthetics used in this study reversibly inhibited $\alpha_4\beta_2$ nACh receptors with IC_{50} values ranging from 0.00091 atm for 1,2-difluorobenzene to 0.045 atm for hexafluorobenzene. With the exception of hexafluorobenzene, all of the compounds had IC_{50} values less than minimum alveolar concentration. Inhibitory potency correlated poorly with the cation-π binding energies of the compounds ($r^2 = 0.48$, $P = 0.059$). However, there was a good correlation between inhibitory potency and the octanol/gas partition coefficient ($r^2 = 0.87$, $P = 0.0008$).
CONCLUSIONS: Volatile aromatic anesthetics potently and reversibly inhibit human $\alpha_4\beta_2$ neuronal nACh receptors. This inhibition may play a role in producing amnesia. In contrast to *N*-methyl-d-aspartate receptors, the inhibitory potencies of aromatic anesthetics for $\alpha_4\beta_2$ neuronal nACh receptors seem to be dependent on drug hydrophobicity rather than electrostatic properties. This implies that the volatile aromatic anesthetic binding site in the $\alpha_4\beta_2$ neuronal nACh receptor is hydrophobic in character and differs from the nature of the binding site in *N*-methyl-d-aspartate receptors.

圍術期血糖調控的原則與臨床意義

Scientific Principles and Clinical Implications of Perioperative Glucose Regulation and Control

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大手術後高血糖非常普遍，並由多因素調控。常見因素包括圍術期代謝狀態、術中病人管理及手術引起的神經內分泌應激反應。急性胰島素抵抗也會引起圍術期高血糖，並且起到很大作用。高血糖與危重病人及手術病人的不良預後具有相關性。經調查多數“高血糖”的診斷過於隨便，而且其初始治療閾值也大相迥異。曾有研究表明接受強化血糖控制（IGC,目標血糖濃度<110 mg/dL）的危重及手術病人預後得以改善。這些結果被其他臨床領域借鑒，並且圍術期也廣泛採用強化血糖控制。然而，很少有研究證實圍術期血糖控制的意義。此外，因沒有（確定）適當的血糖治療目標值，也沒有闡明圍術期血糖控制的真正效能，最近這些前瞻性實驗並不能體現 IGC 的益處。執業醫師們需理解不同血糖測量技術的臨床意義。IGC 顯著增加高血糖的危險性，而這在危重病人並無相關性。最新收集資料表明：圍術期血糖應慎重地控制在<180 mg/dL，同時血糖控制應基於密切的血糖監測。
(鄒巧群 譯 陳傑 校)

Development of hyperglycemia after major operations is very common and is modulated by many factors. These factors include perioperative metabolic state, intraoperative management of the patient, and neuroendocrine stress response to surgery. Acute insulin resistance also develops perioperatively and contributes significantly to hyperglycemia. Hyperglycemia is associated with poor outcomes in critically ill and postsurgical patients. A majority of the investigations use the term "hyperglycemia" very loosely and use varying thresholds for initiating treatment. Initial studies demonstrated improved outcomes in critically ill, postsurgical patients who received intensive glycemic control (IGC) (target serum glucose <110 mg/dL). These results were quickly extrapolated to other clinical areas, and IGC was enthusiastically recommended in the perioperative period. However, there are few studies investigating the value of intraoperative glycemic control. Moreover, recent prospective trials have not been able to show the benefit of IGC; neither an appropriate therapeutic glycemic target nor the true efficacy of perioperative glycemic control has been fully determined. Practitioners should also appreciate technical nuances of various glucose measurement techniques. IGC increases the risk of hypoglycemia significantly, which is not inconsequential in critically ill patients. Until further specific data are accumulated, it is prudent to maintain glucose levels <180 mg/dL in the perioperative period, and glycemic control should always be accompanied by close glucose monitoring.

亨廷頓病病人的麻醉管理

Anesthetic Management of Patients with Huntington Disease

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Anesth Analg 2010 110: 515-523.

背景：亨廷頓病(HD)是一種罕見的常染色體顯性遺傳性疾病。以舞蹈症、肌張力減退、共濟失調、認知障礙和行為減退為症狀。在病例報導中顯示這類患者對於麻醉的異常反應，需提高對這類患者麻醉安全的關注度。
方法：作者檢索了 Mayo Clinic 中亨廷頓病病人接受全身麻醉的醫療記錄資料。主要是回顧麻醉技術、藥物使用及術後併發症。
結果：有 17 例全麻的 11 例基因確診的亨廷頓病病人。精神類藥物的使用非常廣泛，有 6 例使用抗精神病藥物，抗抑鬱藥使用 7 例，3 例使用苯二氮卓類藥物。司可林使用 7 例，非去極化肌松藥使用 11 例，均無不良反應。患者在麻醉誘導及維持期間均無異常反應。嚴重的併發症也未發生。
結論：與以前的病例報告比較，作者發現，亨廷頓病病人進行全身麻醉時無異常反應。但是，麻醉醫生應該認識到麻醉藥和這類病人經常使用的精神藥物之間的相互作用。另外，因為延髓功能障礙可能是這種疾病表現，所以應該採取相應的措施來降低誤吸的危險性。
(張蕾 譯 陳傑 校)
BACKGROUND: Huntington disease (HD) is a rare autosomal dominant disease with symptoms of chorea, dystonia, incoordination, cognitive decline, and behavioral difficulties. Abnormal responses to anesthesia have been reported in case reports and raised concerns regarding the safety of anesthesia in this patient population.

METHODS: We performed a computerized search of the Mayo Clinic medical records database searching for patients with HD who underwent general anesthesia. Medical records were reviewed for anesthetic technique, medications used, and postoperative complications.
RESULTS: We identified 11 patients with genetically confirmed HD who underwent 17 general anesthetics. Psychiatric medication use was common, with 6 patients using antipsychotics, 7 patients using antidepressants, and 3 patients using benzodiazepines. Succinylcholine was used in 7 anesthetics, and nondepolarizing neuromuscular blocking drugs in 11 anesthetics, all without adverse effects. Patients had normal responses to induction and maintenance of anesthesia without adverse effects. Serious postoperative complications did not occur.
CONCLUSION: Contrary to previous case reports, we found that patients with HD have normal responses to general anesthesia. However, the anesthesiologist should be aware of interactions between anesthetics and psychiatric medications frequently used by these patients. Measures should also be taken to minimize the risk of pulmonary aspiration because bulbar dysfunction may be a manifestation of this disease.

血管活性藥物對內毒素血症的大鼠腸道功能性毛細血管密度（FCD）的影響：活體顯微視頻分析

The Effects of Vasoactive Drugs on Intestinal Functional Capillary Density in Endotoxemic Rats: Intravital Video-Microscopy Analysis

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背景：應用血管活性藥物來恢復膿毒血症休克病人動脈血壓仍然是危重病醫學的基礎。然而，升壓藥會加重膿毒血症休克病人內臟的灌注不足而導致細菌侵入和內毒素血症。在這項研究中，作者比較了不同血管活性藥物對內毒素血症休克未行液體治療的大鼠，腸道微循環和組織氧合的影響。
方法：戊巴比妥麻醉後的 Wistar Kyoto 大鼠由大腸桿菌內毒素（2mg/kg 靜注）誘導至內毒素血症休克。通過持續注射血管活性藥物維持動脈血壓，這些藥物有腎上腺素、去甲腎上腺素、苯腎上腺素、多巴胺，還有多巴酚丁胺與去甲腎上腺素的合劑。使用活體視頻顯微鏡評估小腸肌層的功能性毛細血管密度。同時進行腸系膜靜脈血氣和乳酸分析。
結果：靜注腎上腺素、去甲腎上腺素和苯腎上腺素後，功能性毛細血管密度約下降 25%~60%。注射多巴胺、多巴酚丁胺、多巴酚丁胺與去甲腎上腺素的合劑沒有引起消化道功能性毛細血管密度的重大變化。此外，在注射苯腎上腺素後腸系膜靜脈乳酸含量增加，注射腎上腺素和去甲腎上腺素後其乳酸含量有升高趨勢，而注射多巴胺、多巴酚丁胺、多巴酚丁胺與去甲腎上腺素合劑後其乳酸含量沒有顯著升高。
結論：這項研究證實了感染性休克實驗模型中系統血流動力學和微循環兩者影響分離。此外，研究顯示多巴胺、多巴酚丁胺、多巴酚丁胺和去甲腎上腺素合劑對內毒素血症大鼠的腸肌層微循環有保護作用。
(楊秋娟 譯 陳傑 校)

BACKGROUND: The use of vasoactive drugs to restore arterial blood pressure in patients with septic shock remains a cornerstone of intensive care medicine. However, vasopressors can accentuate the hypoperfusion of the gut during septic shock, allowing bacterial translocation and endotoxemia. In this study, we compared the effects of different vasoactive drugs on intestinal microcirculation and tissue oxygenation, independent of the effects of fluid therapy, in a rat model of endotoxemic shock.
METHODS: Pentobarbital-anesthetized Wistar Kyoto rats were submitted to endotoxemic shock induced by *Escherichia coli* lipopolysaccharide (2 mg/kg IV). Arterial blood pressure was normalized by a continuous infusion of different vasoactive drugs, including epinephrine, norepinephrine, phenylephrine, dopamine, dobutamine, or a combination of dobutamine and norepinephrine. The functional capillary density (FCD) of the muscular layer of the small intestine was evaluated by intravital video-microscopy. Mesenteric venous blood gases and lactate concentrations were also analyzed.
RESULTS: FCD decreased by approximately 25% to 60% after the IV infusion of epinephrine, norepinephrine, and phenylephrine. Administration of dopamine, dobutamine, and the combination of dobutamine and norepinephrine did not induce significant alterations in gut FCD. In addition, the mesenteric venous lactate concentration increased in the presence of phenylephrine and showed a tendency to increase after the administration of epinephrine and norepinephrine, whereas there was no observable increase after the administration of dopamine, dobutamine, and the combination of dobutamine with norepinephrine.
CONCLUSION: This study confirms dissociation of the systemic hemodynamic and microvascular alterations in an experimental model of septic shock. Moreover, the results indicate that the use of dopamine, dobutamine, and dobutamine in combination with norepinephrine yields a protective effect on the microcirculation of the intestinal muscular layer in endotoxemic rats.

Meta 分析比較地氟醚和七氟醚拔管平均時間和變異率

Statistical Modeling of Average and Variability of Time to Extubation for Meta-Analysis Comparing Desflurane to Sevoflurane

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背景：理想的麻醉藥和麻醉方法是復蘇快（例如：從手術結束到拔管平均 5min）且變異度小（例如：總是 4-7min）。作者用 AIMS 資料研究如何模仿從手術結束到拔管的時間。應用 meta 分析測試那些資訊，比較使用地氟醚和七氟醚後的拔管時間。
方法：AIMS data 研究由 95 位外科醫生實施的 32,792 例手術，包括在手術室的氣管插管和拔管，以及使用麻醉揮發氣體。Meta 分析通過 2008 年 29 個隨機對照研究來比較應用七氟醚和地氟醚的拔管時間。方法和標準差百分比的不同應用隨機 meta 分析和 Bayesian 法來研究。
結果:拔管的時間用威布林分佈分析較正態分佈分析更適合。藥物選擇對平均值和標準差的影響幾乎一致，正像變異係數未變一樣（29 個研究中 26 個研究 $P > 0.10$）及差值變異係數變化無意義（七氟醚 – 地氟醚 = –1%, 95% 可信區間 [CI] –3% to 1%, $P = 0.22$)。地氟

醚減少拔管時間 25% (95% 可信區間 17%–32%, $P < 0.0001$)和標準差降低 21% (95%可信區間 16%–26%)。為評價長時間拔管的無形成本（例如：外科醫生無益的等待時間），作者認為 15% AIMS 案例拔管時間大於 15min。這些案例從出手術室到外科醫生進行下一台手術平均延長 4.9min (95% 可信區間 2.7–7.1 min, $P < 0.0001$)。平均時間和標準差減少 20%–25%將會減少拔管延長時間的發生率 71%–82% (95% 可信區間 68%–84%)。

結論：與七氟醚相比地氟醚能減少平均拔管時間和變異率 20%–25%。這些方面的主要的經濟價值是手術花費時間的減少。然而，應考慮拔管時間延長造成隨後工作延遲導致有關的難以確定的花費。地氟醚較七氟醚拔管時間平均值和不一致的減少可解釋和測定預期延長拔管發生率減少 75%。

(陳靈科 譯 陳傑 校)

BACKGROUND: The recovery profile of an ideal anesthetic or technique would be fast (e.g., mean of 5 min from end of surgery to extubation) with little variability (e.g., always 4–7 min). We used anesthesia information management system (AIMS) data to learn how to model the time from end of surgery to extubation. We applied that knowledge for meta-analyses of trials comparing extubation times after use of desflurane and sevoflurane.

METHODS: AIMS data studied were 32,792 cases performed by 95 surgeons that included tracheal intubation and extubation in the operating room (OR) and use of volatile anesthetic(s). Meta-analysis included the 29 randomized controlled trials through 2008 comparing extubation times with desflurane and sevoflurane. Percentage differences in means and standard deviations were studied using random effects meta-analysis and a Bayesian method.

RESULTS: Times to extubation were better fit by (skewed) Weibull distributions than by (symmetric) normal distributions. Drug choice had nearly equally proportional effects on the means and standard deviations of extubation times, as shown by unchanged coefficients of variation ($P > 0.10$ for 26 of 29 studies) and nonsignificant pooled difference in the coefficient of variation (sevoflurane – desflurane = –1%, 95% confidence interval [CI] –3% to 1%, $P = 0.22$). Applying these findings, desflurane reduced the mean extubation time by 25% (95% CI 17%–32%, $P < 0.0001$) and standard deviation by 21% (95% CI 16%–26%). To value the intangible costs (e.g., frustrated waiting surgeons) of prolonged extubation times, we considered the 15% of AIMS cases with times >15 min. These cases averaged 4.9 min longer times from out of the OR to the start of surgery of the surgeon's next case (95% CI 2.7–7.1 min, $P < 0.0001$). Reduction in the means and standard deviations by 20%–25% would likely reduce incidences of these prolonged extubation times by 71%–82% (95% CI 68%–84%).

CONCLUSIONS: Desflurane reduces the average extubation time and the variability of extubation time by 20%–25% relative to sevoflurane. The principal economic value of these end points is their reductions of direct (labor) costs of OR time. However, reductions in intangible costs of prolonged extubation are real, being associated with subsequent delays. Reductions in the average and variance of times to extubation can be interpreted and monitored in terms of corresponding expected 75% reductions in the incidences of prolonged extubation times by using desflurane relative to sevoflurane.

大麻隆對纖維肌痛病人其睡眠品質的作用：隨機對照試驗

The Effects of Nabilone on Sleep in Fibromyalgia: Results of a Randomized Controlled Trial

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背景：睡眠障礙會對許多有慢性疼痛的病人造成影響。曾有報導稱印度大麻可説明病人入睡。以廣泛的慢性疼痛和失眠症為主要特徵的纖維肌痛症也會引起睡眠障礙，因而本文主要討論研究了一種合成的大麻素，即大麻隆，在治療此種睡眠障礙過程中的安全性和有效性。
方法：作者應用隨機，雙盲，主動對照及交叉試驗的方法比較了大麻隆(睡前 0.5-1.0mg)和阿米替林（睡前 10-20mg）對於有慢性失眠症的纖維肌痛病人的不同作用。受試者各服用大麻隆和阿米替林這兩種藥物兩個星期，之後各有兩個星期的藥物洗脫期。主要的評價結果是睡眠品質，用失眠嚴重指數以及里茲睡眠評估問卷來測試。次要的評價結果包括疼痛，情緒，生活品質和副反應。
結果：31 個受試者加入了這個實驗，其中 29 個人完成了這項試驗（26 個女性，平均年齡 49.5 歲）。儘管大麻隆和阿米替林都能幫助睡眠，但是大麻隆比阿米替林的作用效果更好（失眠嚴重指數相差 3.2，95%CI=1.2-5.3）。大麻隆可以幫助病人好好地休息，在這方面比阿米替林稍稍優越（里茲睡眠評估問卷差別=0.5（0.0-1.0）），然而對於不眠症，大麻隆和阿米替林相比沒有多大差別（差值=0.3（-0.2-0.8））。我們沒有發現兩種藥對於疼痛，情緒和生活品質的影響。但是有輕到中度的副反應，而且大麻隆的副反應相較于阿米替林更頻繁。大麻隆最常見的副反應是頭昏眼花，噁心和口幹。
結論：大麻隆可有效提高纖維肌痛病人的睡眠品質，且耐受性好。作者認為每天睡前應給予低劑量的大麻隆，而不選擇阿米替林。還需進行更多的試驗來明確大麻隆這種藥物作用的持續時間以及長期使用這種藥物的安全性問題。
(張婷 譯 陳傑 校)

BACKGROUND: Sleep disorders affect many patients with chronic pain conditions. Cannabis has been reported by several patient populations to help sleep. We evaluated the safety and efficacy of nabilone, a synthetic cannabinoid, on sleep disturbance in fibromyalgia (FM), a disease characterized by widespread chronic pain and insomnia.
METHODS: We conducted a randomized, double-blind, active-control, equivalency crossover trial to compare nabilone (0.5–1.0 mg before bedtime) to amitriptyline (10–20 mg before bedtime) in patients with FM with chronic insomnia. Subjects received each drug for 2 wk with a 2-wk washout period. The primary outcome was sleep quality, measured by the Insomnia Severity Index and the Leeds Sleep Evaluation Questionnaire. Secondary outcomes included pain, mood, quality of life, and adverse events (AEs).
RESULTS: Thirty-one subjects were enrolled and 29 completed the trial (26 women, mean age 49.5 yr). Although sleep was improved by both amitriptyline and nabilone, nabilone was superior to amitriptyline (Insomnia Severity Index difference = 3.2; 95% confidence interval = 1.2–5.3). Nabilone was marginally better on the restfulness (Leeds Sleep Evaluation Questionnaire difference = 0.5 [0.0–1.0]) but not on wakefulness (difference = 0.3 [–0.2 to 0.8]). No effects on pain, mood, or quality of life were observed. AEs were mostly mild to moderate and were more frequent with nabilone. The most common AEs for nabilone were dizziness, nausea, and dry mouth.

CONCLUSIONS: Nabilone is effective in improving sleep in patients with FM and is well tolerated. Low-dose nabilone given once daily at bedtime may be considered as an alternative to amitriptyline. Longer trials are needed to determine the duration of effect and to characterize long-term safety.

創傷患者接受長期連續外周神經阻滯導管治療後其血清羅呱卡因濃度和局麻藥的全身毒性之間的關係

Serum Ropivacaine Concentrations and Systemic Local Anesthetic Toxicity in Trauma Patients Receiving Long-Term Continuous Peripheral Nerve Block Catheters

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背景：羅呱卡因是一種長效局麻藥，常用于外周神經阻滯及連續外周神經阻滯導管中。在 Walter Reed 部隊醫療中心，戰爭創傷患者疼痛治療方案的一部分就是接受連續外周神經阻滯導管。這些導管通常在原處留置數日至數周不等。在本次研究中，作者通過檢測患者血清中結合的和非結合的局麻藥的濃度，以評估隨著時間的推移，創傷患者羅呱卡因的血藥濃度水準。同時，還檢測了長時間接受羅呱卡因輸注患者的血清 α-酸糖蛋白的濃度。
方法：本次研究共有 15 位患者入組，其中 2 位因僅獲取到一個羅呱卡因濃度而被排除。在其餘 13 位患者中，2 位在入組時放置了外周神經阻滯導管，另外 11 位在入組前即已放置，且這些患者在首次測試局麻藥水準之前，已接受輸注 0.2%羅呱卡因 18-126 小時。0.2%羅呱卡因的輸注速度為 6-14ml/h，導管內單次給藥時羅呱卡因的濃度為 0.5%。在第 1、3、5、7 和 10 日及此後每 3 日一次測量局麻藥的血藥濃度，直到拔除所有導管，但患者並未全部接受所有檢測。
結果：在研究入組的 13 位患者中，共獲取了 59 個血樣本。為了控制急性疼痛，導管在原處留置的平均時間為 7 日（範圍：6-27 日）。患者入組後導管留置在原處的平均時間為 7 日（範圍：4-25 日）。每位患者平均獲取到 4 個血樣本（範圍：2-10 個樣本）。2 位患者的血清游離羅呱卡因濃度異常升高並超出之前認為的毒性範圍，且沒有明顯的下降的跡象。這兩位患者在抽取該血樣本之前約 24 小時內接受 0.5%羅呱卡因的單次給藥共 300mg。研究整個過程中羅呱卡因的平均濃度為 0.11mg/l（範圍：測不到至 0.63mg/l）。在研究開始後的第一周內，每位患者的血清羅呱卡因濃度的平均變化值為 0.00mg/l（範圍：-0.35-0.47mg/l）。
結論：儘管 2 位患者的血清游離羅呱卡因濃度異常升高並超出之前認為的毒性範圍，但是 Walter Reed 部隊醫療中心所使用的連續外周神經阻滯導管及其給予的局麻藥劑量並未在臨床上導致明顯的全身毒性。除了 1 位在入組前已接受羅呱卡因輸注外，其餘患者的羅呱卡因濃度和 α-酸糖蛋白的濃度之間沒有相關性。儘管如此，局麻藥輸注的總時間似乎並不影響藥物的游離濃度。

(張婷 譯 陳傑 校)

BACKGROUND: Ropivacaine is a long-acting local anesthetic used frequently for peripheral nerve blocks and continuous peripheral nerve block catheters. Combat trauma patients at Walter Reed Army Medical Center often receive continuous peripheral nerve block catheters as part of their pain regimen. These catheters remain *in situ* for several days to weeks. In this study, we evaluated the free ropivacaine drug levels over time in trauma patients by measuring the serum concentration of bound and unbound local anesthetic. The corresponding α_1-acid glycoprotein concentration in patients with prolonged ropivacaine infusions was also measured.

METHODS: Fifteen patients were enrolled in the study; 2 patients were excluded because only a single ropivacaine level was obtained. Of the remaining 13 patients in the study, 2 had peripheral nerve catheters placed at the time of enrollment; the remaining 11 patients had catheters placed before enrollment. These patients were already receiving 0.2% ropivacaine infusions for a period of 18–126 h before the first assessment of local anesthetic level. Catheters infused 0.2% ropivacaine at a rate of 6–14 mL/h; catheter boluses were administered with 0.5% ropivacaine. Local anesthetic blood concentrations were scheduled to be measured on Days 1, 3, 5, 7, and 10 and every 3 days thereafter until all catheters were removed, although not all patients underwent each assessment. Specimens were assayed using high-performance liquid chromatography for total and free serum ropivacaine concentrations. α_1-Acid glycoprotein was also measured.

RESULTS: Thirteen patients remained in the study, for a total of 59 blood samples. The median number of days catheters remained *in situ* for the duration of acute pain therapy was 7 days (range: 6–27 days). The median number of days catheters remained *in situ* after enrollment into the study was 7 days (range: 4–25 days). The median number of blood samples collected per patient was 4 (range: 2–10 samples). Two patients had isolated increased concentrations of free ropivacaine into a previously identified toxic range with no obvious mitigating factors; both patients had received a 300-mg bolus of 0.5% ropivacaine approximately 24 h before that blood collection. The median ropivacaine concentration over the length of the study was 0.11 mg/L (range: undetectable to 0.63 mg/L). During the first week of the study, the median change in ropivacaine concentration per patient was 0.00 mg/L (range: –0.35 to 0.47 mg/L).

CONCLUSION: Although 2 patients demonstrated isolated serum ropivacaine concentration spikes into a previously identified toxic range, continuous peripheral nerve block catheter management and local anesthetic doses as practiced at Walter Reed Army Medical Center did not result in clinically evident systemic ropivacaine toxicity. There was no correlation between free ropivacaine concentration and α_1-acid glycoprotein concentration except in patients who had already been receiving ropivacaine infusions before entering the study. Despite this lack of correlation, the total duration of local anesthetic infusion did not seem to influence the free concentration of the drug.

低溫體外迴圈複溫過程中的腦血流自動調節功能受損及其與中風的潛在聯繫

Impaired Autoregulation of Cerebral Blood Flow During Rewarming from Hypothermic Cardiopulmonary Bypass and Its Potential Association with Stroke

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背景：心臟手術患者術後的腦損傷與低溫體外迴圈（CPB）後的複溫相關。在本研究中，我們評估成人患者體外迴圈過程中先降溫後升溫是否導致腦血流—血壓自動調節的變化。
方法：在 127 位行體外迴圈心臟手術的成年患者中使用經顱多普勒超聲監測左右大腦中動脈的血流速度。11 位行體外迴圈的患者保持動脈流入溫度〉35°C 作為對照組。平均速度指數(Mx)計算為大腦中動脈血流速度慢波和平均動脈壓之間的移動的線性相關係數。腦血流—血壓自動調節功能完整時的 Mx 接近 0。Mx 隨腦血流—血壓自動調節功能受損程度的加重而增加，接近於 1。比較體外迴圈前（基礎值）、體外迴圈的降溫相和升溫相過程中及體外迴圈後的 Mx 的時間平均值。測定體外迴圈各個階段的 Mx 計數〉4.0 即提示腦血流的自動調節功能受損的患者人數。
結果：在降溫過程中，Mx（左，0.29 ± 0.18；右，0.28 ± 0.18[平均數±標準差]）較基礎值提高（左，0.17 ± 0.21; 右, 0.17 ± 0.20; *P* ≤0.0001）。體外迴圈升溫過程中的 Mx (左, 0.40 ± 0.19; 右，0.39 ± 0.19；*P* 0.0001)較基礎值（*P* ≤0.001）和降溫階段（*P* ≤0.0001）增加，提示腦血流的自動調節功能受損。體外迴圈後，Mx (左, 0.27 ± 0.20; 右, 0.28 ± 0.21) 較基礎值增高(左, *P* = 0.0004; 右, *P* = 0.0003), 與降溫過程無差別，但是較升溫過程降低(左, *P* ≤0.0001;右, *P*≤ 0.0005)。43 個患者（34%）在體外迴圈降溫過程中及 68 位患者（53%）在複溫過程中平均 Mx ≥0.4。11 位元溫度控制的患者中 9 位在整個體外迴圈過程中 Mx ≥0.4。在術後 7 位患者發生中風，1 例短暫腦缺血發作。所有發生中風的患者在複溫過程中的 Mx ≥0.4（P=0.015）。複溫時 Mx ≥0.4 的患者發生任何神經系統意外（中風或短暫腦缺血發作）的未調整優勢比為 6.57(95% 可信區間, 0.79～55.0, *P* < 0.08)。
結論：低溫體外迴圈會引起腦血流—血壓自動調節功能的損傷，而升溫過程會加重這種損傷。我們發現有腦血流自動調節受損證據的患者的中風發生率較高。升溫過程中壓力被動的腦血流狀態是否導致腦缺血損傷發生的危險有待進一步的研究。
(胡豔 譯 馬皓琳 李士通 校)

BACKGROUND: Patient rewarming after hypothermic cardiopulmonary bypass (CPB) has been linked to brain injury after cardiac surgery. In this study, we evaluated whether cooling and then rewarming of body temperature during CPB in adult patients is associated with alterations in cerebral blood flow (CBF)–blood pressure autoregulation.

METHODS: One hundred twenty-seven adult patients undergoing CPB during cardiac surgery had transcranial Doppler monitoring of the right and left middle cerebral artery blood flow velocity. Eleven patients undergoing CPB who had arterial inflow maintained at >35°C served as controls. The mean velocity index (Mx) was calculated as a moving, linear correlation coefficient between slow waves of middle cerebral artery blood flow velocity and mean arterial blood pressure. Intact CBF–blood pressure autoregulation is associated with an Mx that approaches 0. Impaired autoregulation results in an increasing Mx approaching 1.0. Comparisons of time-averaged Mx values were made between the following periods: before CPB (baseline), during the cooling and rewarming phases of CPB, and after CPB. The number of patients in each phase of CPB with an Mx >4.0, indicative of impaired CBF autoregulation, was determined.

RESULTS: During cooling, Mx (left, 0.29 ± 0.18; right, 0.28 ± 0.18 [mean ± sd]) was greater than that at baseline (left, 0.17 ± 0.21; right, 0.17 ± 0.20; $P \leq 0.0001$). Mx increased during the rewarming phase of CPB (left, 0.40 ± 0.19; right, 0.39 ± 0.19) compared with baseline ($P \leq 0.001$) and the cooling phase ($P \leq 0.0001$), indicating impaired CBF autoregulation. After CPB, Mx (left, 0.27 ± 0.20; right, 0.28 ± 0.21) was higher than at baseline (left, $P = 0.0004$; right, $P = 0.0003$), no different than during the cooling phase, but lower than during rewarming (left, $P \leq 0.0001$; right, $P \leq 0.0005$). Forty-three patients (34%) had an Mx ≥0.4 during the cooling phase of CPB and 68 (53%) had an average Mx ≥0.4 during rewarming. Nine of the 11 warm controls had an average Mx ≥0.4 during the entire CPB period. There were 7 strokes and 1 TIA after surgery. All strokes were in patients with Mx ≥0.4 during rewarming ($P = 0.015$). The unadjusted odds ratio for any neurologic event (stroke or transient ischemic attack) for patients with Mx ≥0.4 during rewarming was 6.57 (95% confidence interval, 0.79 to 55.0, $P < 0.08$).
CONCLUSIONS: Hypothermic CPB is associated with abnormal CBF–blood pressure autoregulation that is worsened with rewarming. We found a high rate of strokes in patients with evidence of impaired CBF autoregulation. Whether a pressure-passive CBF state during rewarming is associated with risk for ischemic brain injury requires further investigation.

左布比卡因對離體大鼠主動脈的直接影響與脂氧酶通道啟動和內皮源性一氧化氮釋放有關

The Direct Effect of Levobupivacaine in Isolated Rat Aorta Involves Lipoxygenase Pathway Activation and Endothelial Nitric Oxide Release

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背景:左布比卡因是一種長效局部麻醉藥,它臨床特點與消旋布比卡因類似，但是它有較大的安全範圍。在活體上，左布比卡因能產生劑量依賴性的血管收縮。本篇離體研究中，我們的目的是：研究花生四烯酸代謝通路在左布比卡因導致離體大鼠主動脈收縮中的作用及探討哪種內皮源性血管舒張劑調節左布比卡因誘導血管收縮。
方法：分離大鼠胸主動脈環並懸吊，用於記錄等長張力。在 10^{-6} 到 3×10^{-4} M 濃度範圍內，在以下三組實驗繪製累積左布比卡因劑量－反應曲線：1）無藥物預處理的主動脈環組；2）內皮剝除的主動脈環組，預處理用二鹽酸喹吖因（非特異性磷脂酶 A2 抑制劑：2×10^{-5}、4×10^{-5} M）、去甲二氫愈創木酸（NDGA）（脂氧合酶抑制劑：10^{-5}、3×10^{-5}M）、消炎痛（非特異環氧合酶抑制劑：10^{-5} M）、AA-861（5-脂氧合酶抑制劑：10^{-5}、5×10^{-5} M）、氟康唑（細胞色素 P450 環氧化酶抑制劑：10^{-5} M）、維拉帕米（10^{-5} M）或無鈣溶液；3）內皮完整的主動脈環組，預處理用 N^{ω}-硝基-L 精氨酸甲酯（L-NAME）（一氧化氮合成酶抑制劑： 5×10^{-5} M)、消炎痛、或氟康唑。在內皮剝除主動脈環組，評

估在每個濃度水準（10^{-4},、3 x 10^{-4}）左布比卡因誘導的收縮反應。 在使用 NDGA 和 AA-861 或不使用情況下，分別繪製出內皮剝除主動脈環中氯化鉀的劑量－反應曲線。在單獨使用左比卡因或左旋布比卡因加 AA-861 干預的血管平滑機細胞內，通過使用 Fluo-4 螢光進行 Ca^{2+}圖像分析來監測細胞內 Ca^{+2} 水準。

結果：左布比卡因使離體主動脈環產生緊張性收縮。在左布比卡因濃度為 10^{-4} M 時，該反應最大，在濃度為 3 x 10^{-4}M 時,反應逐漸減弱。左布比卡因誘導內皮剝除主動脈環收縮強度比內皮完整主動脈環收縮強度大。內皮剝除主動脈環組：二鹽酸喹吖因、NDGA、AA-861、維拉帕米和無鈣溶液都減弱了左布比卡因誘導的主動脈環收縮，消炎痛則在較小程度上減弱其收縮。內皮完整主動脈環組：L-NAME 增強了左布比卡因誘導的主動脈環收縮，消炎痛輕微減弱了其收縮。NDGA 和 AA-861 減弱了氯化鉀導致的主動脈環收縮。AA-861 減弱了左布比卡因導致的血管平滑肌細胞內鈣離子增加。

結論：資料表明左布比卡因誘導大鼠主動脈平滑肌收縮主要由啟動脂氧合酶途徑調節及部分由啟動環氧合酶途徑調節。另外，啟動脂氧合酶途徑似乎增加了鈣離子通過 L 型鈣通道的內流。內皮源性一氧化氮減弱了左布比卡因誘導的主動脈收縮。

(王海濤 譯 馬皓琳 李士通 校)

BACKGROUND: Levobupivacaine is a long-acting local anesthetic with a clinical profile similar to that of racemic bupivacaine but with a greater margin of safety. Levobupivacaine produces dose-dependent vasoconstriction *in vivo*. Our goal in this *in vitro* study was to investigate the role of pathways involved in arachidonic acid metabolism in the levobupivacaine-induced contraction of isolated rat aorta and to determine which endothelium-derived vasodilators are involved in the modulation of levobupivacaine-induced contraction.

METHODS: Rat thoracic aortic rings were isolated and suspended for isometric tension recording. Cumulative levobupivacaine dose-response curves over a range of 10^{-6} to 3 x 10^{-4} M were constructed in 1) aortic rings with no drug pretreatment; 2) endothelium-denuded rings pretreated with quinacrine dihydrochloride (nonspecific phospholipase A_2 inhibitor: 2 x 10^{-5}, 4 x 10^{-5} M), nordihydroguaiaretic acid (NDGA) (lipoxygenase inhibitor: 10^{-5}, 3 x 10^{-5} M), indomethacin (nonspecific cyclooxygenase inhibitor: 10^{-5} M), AA-861 (5-lipoxygenase inhibitor: 10^{-5}, 5 x 10^{-5} M), fluconazole (cytochrome P450 epoxygenase inhibitor: 10^{-5} M), verapamil (10^{-5} M), or calcium-free solution; and 3) endothelium-intact rings pretreated with N^{ω}-nitro-l-arginine methyl ester (L-NAME) (nitric oxide synthase inhibitor: 5 x 10^{-5} M), indomethacin, or fluconazole. Levobupivacaine-induced contractile response at each concentration (10^{-4}, 3 x 10^{-4} M) was assessed in endothelium-denuded rings. Dose-response curves for potassium chloride in endothelium-denuded rings were generated in the presence or absence of NDGA and AA-861. Intracellular Ca^{2+} levels were monitored by Ca^{2+} image analysis using Fluo-4 fluorescence in vascular smooth muscle cells treated with levobupivacaine alone or AA-861 plus levobupivacaine.

RESULTS: Levobupivacaine produced a tonic contraction in isolated rat aorta rings; this response was maximal at 10^{-4} M levobupivacaine and gradually attenuated at 3 x 10^{-4} M levobupivacaine. Levobupivacaine-induced contractions of endothelium-denuded rings were larger than those of endothelium-intact rings. Levobupivacaine-induced contraction of endothelium-denuded rings was attenuated by quinacrine dihydrochloride, NDGA, AA-861, verapamil, and calcium-free solution and, to a lesser extent, by indomethacin. L-NAME enhanced levobupivacaine-induced contraction of endothelium-intact rings and indomethacin slightly attenuated this contraction. NDGA and AA-861 attenuated the potassium chloride-

induced contraction. AA-861 attenuated the levobupivacaine-induced intracellular calcium increase in vascular smooth muscle cells.
CONCLUSIONS: Our data indicate that levobupivacaine-induced contraction of rat aortic smooth muscle is mediated mainly by activation of the lipoxygenase pathway and in part by activation of the cyclooxygenase pathway. In addition, activation of the lipoxygenase pathway seems to facilitate calcium influx via L-type calcium channels. Endothelial nitric oxide attenuates levobupivacaine-induced contraction.

圍手術期兒童晶體液和膠體液的管理：我們已經做到的和如何做到的？

Perioperative Crystalloid and Colloid Fluid Management in Children: Where Are We and How Did We Get Here?

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Holliday 和 Segar 在他們里程碑式的文獻中（Pediatrics 1957; 19:823-32）推薦了給予住院兒童腸外支援補液的速度和成分，這已經有 50 多年歷史了。在圍手術期對液體管理的實踐操作也多以此文獻為基礎。但小兒手術病人對葡萄糖、電解質和血管內容量的需求也許和最初的人群已大相徑庭，因此，使用由 Holliday 和 Segar 推薦的傳統低滲液體可能導致高血糖、低鈉血症等術後併發症。在術後使用等滲液還是低滲液還存在較大的爭論。我們討論兒童圍手術期液體管理的起源，綜述當前晶體液管理的選擇，並呈現有關在小兒患者使用膠體液的資訊。
(滕淩雅 譯 馬皓琳 李士通 校)
It has been more than 50 yr since the landmark article in which Holliday and Segar (Pediatrics 1957;19:823–32) proposed the rate and composition of parenteral maintenance fluids for hospitalized children. Much of our practice of fluid administration in the perioperative period is based on this article. The glucose, electrolyte, and intravascular volume requirements of the pediatric surgical patient may be quite different than the original population described, and consequently, use of traditional hypotonic fluids proposed by Holliday and Segar may cause complications, such as hyperglycemia and hyponatremia, in the postoperative surgical patient. There is significant controversy regarding the choice of isotonic versus hypotonic fluids in the postoperative period. We discuss the origins of perioperative fluid management in children, review the current options for crystalloid fluid management, and present information on colloid use in pediatric patients.

比較丙泊酚和右美托咪啶靜脈鎮靜作用：在中樞和自主神經系統作用的一項隨機、交叉研究

A Comparison of Propofol and Dexmedetomidine for Intravenous Sedation: A Randomized, Crossover Study of the Effects on the Central and Autonomic Nervous Systems

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我們比較了志願者在心理應激期間丙泊酚(PROP)和右美托咪啶(DEX)對自主神經興奮性和主觀感覺的作用。在一項交叉設計中，25 名受試者接受 PROP 和 DEX 滴定直到腦電雙頻指數達 75~80。記錄心率、心率變異性、α—唾液澱粉酶（客觀指標）和面部焦慮量表（主觀指標）。詢問受試者兩種鎮靜劑中的喜好。客觀指標在兩組中有類似的改變。面部焦慮指數僅在 PROP 組中減少，且受試者更青睞 PROP。鎮靜劑量的丙泊酚對抑制焦慮情感比 DEX 更有效。
(唐李雋 譯 馬皓琳 李士通 校)

We compared, in volunteers, the effect of propofol (PROP) and dexmedetomidine (DEX) sedation on autonomic nervous activities and subjective feelings during psychological stresses. In a crossover design, 25 subjects received PROP and DEX titrated to a bispectral index value of 75 to 85. Heart rate, heart rate variability, and salivary α-amylase (objective indices) and a faces anxiety scale (subjective index) were assessed. Subjects were asked their preference between 2 sedatives. Objective indices showed similar changes in both groups. The faces anxiety scale decreased only in the PROP group and subjects preferred PROP. Propofol more effectively suppressed anxious feelings compared with DEX during sedation.

麻醉藥性能以外的方面：異氟醚對腦細胞死亡、神經發生和長時間的認知功能的影響

Beyond Anesthetic Properties: The Effects of Isoflurane on Brain Cell Death, Neurogenesis, and Long-Term Neurocognitive Function

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麻醉藥物會引起新生大鼠的腦細胞死亡和長時間的認知功能障礙。最近，人類資料也顯示生命早期施行麻醉可能引起認知功能損害。腦細胞死亡和神經認知功能功能下降的聯繫尚不確切。可以想像的是，除了腦細胞死亡以外，有其它機制可促使新生兒麻醉導致神經認知損害。在一系列的研究中，顯示暴露於異氟醚會導致出生後 7 天的大鼠明顯的高碳酸血症，暴露於異氟醚和二氧化碳 4 小時會引起腦細胞死亡。然而，暴露於異氟醚 1 小時並不足以引起腦細胞死亡。而且，僅僅暴露於異氟醚 4 小時，而不是暴露於異氟醚 1 小時或 2 小時或暴露於二氧化碳 4 小時，會引起海馬功能損害，這一現象引起了關於麻醉誘導的腦細胞死亡與神經認知功能障礙之間聯繫的疑問。在發育期和成人齒狀回的神經發生對海馬功能尤其是學習和記憶能力是重要的。γ-氨基丁酸可以調節發育期和成熟大腦的增生和神

經元分化。吸入麻醉藥是γ-氨基丁酸能的，可能因此會影響神經發生，這可能是介導麻醉引起非成熟大鼠的神經認知功能下降的另一機制。明白這一機制，將有助於引導目的在於界定人類中這一問題領域的臨床試驗，並可能會得出預防性的和治療性的策略。
(黃麗娜 譯 馬皓琳 李士通 校)

Anesthetic drugs cause brain cell death and long-term neurocognitive dysfunction in neonatal rats. Recently, human data also suggest that anesthesia early in life may cause cognitive impairment. The connection between cell death and neurocognitive decline is uncertain. It is conceivable that mechanisms other than brain cell death contribute to neurocognitive outcome of neonatal anesthesia. In a series of experiments, we demonstrate that isoflurane exposure causes significant hypercarbia in postnatal day 7 rats and that exposure to isoflurane or carbon dioxide for 4 h provoked brain cell death. However, 1 h of isoflurane exposure was not sufficient to cause brain cell death. Moreover, only 4 h of isoflurane exposure, but not 1 or 2 h of exposure or 4 h of carbon dioxide, led to impaired hippocampal function, questioning the association between anesthesia-induced brain cell death and neurocognitive dysfunction. Neurogenesis both in the developing and adult dentate gyrus is important for hippocampal function, specifically learning and memory. γ-Amino-butyric-acid regulates proliferation and neuronal differentiation both in the developing and the adult brain. Inhaled anesthetics are γ-amino-butyric-acid-ergic and may therefore affect neurogenesis, which could be an alternative mechanism mediating anesthesia-induced neurocognitive decline in immature rats. Understanding the mechanism will help guide clinical trials aiming to define the scope of the problem in humans and may lead to preventive and therapeutic strategies.

氨茶鹼對志願者意識喪失、BIS 值、異丙酚需要量以及地氟醚最低肺泡有效濃度的影響

The Effect of Aminophylline on Loss of Consciousness, Bispectral Index, Propofol Requirement, and Minimum Alveolar Concentration of Desflurane in Volunteers

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背景：腺苷是催眠類的神經遞質，而在臨床上作為支氣管擴張劑的氨茶鹼，能在中樞神經系統對抗腺苷此作用。因此，我們檢驗了此假說，即氨茶鹼延遲了丙泊酚麻醉患者的意識喪失（LOC）同時加快了意識恢復（ROC），並且增加了地氟醚的最低有效肺泡濃度（MAC）值。

方法：在這個雙盲交叉研究中，志願者在不同日期裡被隨機分到氨茶鹼組或鹽水對照組中。在研究日，先予 6 mg/kg 氨茶鹼靜脈推注，然後以 1.5 mg · kg^{-1} · h^{-1} 速度泵入維持 24 小時。在泵入氨茶鹼或鹽水 1 小時後，以 20 mg/min 的速度給予丙泊酚 200mg。持續監測

BIS 值及達到 LOC 和 ROC 的時間。當志願者從異丙酚麻醉中恢復後，以七氟醚全麻誘導，並以地氟醚維持麻醉。用 Dixon 的“上下”法來測定在反復的強直電刺激後每個志願者的 MAC 值。
結果：八名志願者均完成了兩組研究。氨茶鹼組達到 LOC 所需時間較鹽水組延長（均值±標準差）(7.7 ± 2.03 min 比 5.1 ± 0.75 min，*P* = 0.011)。氨茶鹼組達到 LOC 的總異丙酚用量更大（2.2 ± 0.9 比 1.4 ± 0.4 mg/kg, *P* = 0.01），而達到 ROC 所需時間更短（6.18 ± 3.96 比 12.2 ± 4.73 min, *P* = 0.035）。最低 BIS 值氨茶鹼組更大（51 ± 15 比 38 ± 9, *P* = 0.034），而兩組 MAC 值無明顯差異。
結論：氨茶鹼削弱了異丙酚的鎮靜作用，但對強直電刺激法檢測的地氟醚 MAC 值無影響。
BACKGROUND: Adenosine is a soporific neuromodulator; aminophylline, which is clinically used as a bronchodilator, antagonizes the action of adenosine in the central nervous system. Thus, we tested the hypothesis that aminophylline delays loss of consciousness (LOC) and speeds recovery of consciousness (ROC) with propofol anesthesia, and that aminophylline increases the minimum alveolar concentration (MAC) of desflurane.
METHODS: In this double-blind crossover study, volunteers were randomized to either aminophylline or saline on different days. Aminophylline 6 mg/kg was given IV, followed by 1.5 $mg \cdot kg^{-1} \cdot h^{-1}$ throughout the study day. After 1 h of aminophylline or saline administration, propofol 200 mg was given at a rate of 20 mg/min. The bispectral index was continuously monitored, as were times to LOC and ROC. After recovery from propofol, general anesthesia was induced with sevoflurane and subsequently maintained with desflurane. The Dixon "up-and-down" method was used to determine MAC in each volunteer after repeated tetanic electrical stimulation.
RESULTS: Eight volunteers completed both study days. Time to LOC was prolonged by aminophylline compared with saline (mean ± sd) (7.7 ± 2.03 min vs 5.1 ± 0.75 s, respectively, *P* = 0.011). The total propofol dose at LOC was larger with aminophylline (2.2 ± 0.9 vs 1.4 ± 0.4 mg/kg, *P* = 0.01), and the time to ROC was shorter (6.18 ± 3.96 vs 12.2 ± 4.73 min, *P* = 0.035). The minimum bispectral index was greater with aminophylline (51 ± 15 vs 38 ± 9, *P* = 0.034). There was no difference in MAC.
CONCLUSION: Aminophylline decreases the sedative effects of propofol but does not affect MAC of desflurane as determined by tetanic electrical stimulation.

脈動式染色光密度測定法和吲哚花青綠血漿消除用於 ASA I-II 級病人

Pulse Dye Densitometry and Indocyanine Green Plasma Disappearance in ASA Physical Status I-II Patients

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背景：吲哚花青綠血漿消除率（ICG-PDR）被用於評估肝功能。雖然 ICG-PDR <18%/min 通常被認為出現了肝功能衰竭，但 ICG 清除率並不太適合用於定義健康人群肝功能，區

分正常肝功能和肝功能衰竭的清晰的界限值還沒有被描繪。我們因此在另一健康患者人群定義 ICG 清除率。另外，我們評估了無創性檢測 ICG-PDR(經皮在手指和鼻部脈動式染色光密度測定法[PDD])，並與同時完成的有創性 ICG-PDR（在動脈血）測定進行比較。
方法：無肝臟疾病體征，並擇期非肝臟手術患者靜脈注射 10 mg ICG 後通過 PDD(DDG-2001, Nihon Kohden, 東京, 日本) 檢測 ICG-PDR。一組病人收集動脈血，比較有創性 ICG 檢測與 PDD。使用 Bland-Altman 分析比較兩種測定方法。我們研究和報導的關於鑒別性使用 ICG-PDR 評估肝功能衰竭的研究的結果被用於構建受者作用特徵曲線。
結果：41 名患者參加研究：33 名用於手指探頭，8 名用於鼻部探頭。在該人群中非侵入性 ICG-PDR 的均數±標準差是 23.1% ± 7.9%/min (*n* = 41)，範圍在 9.7% 到 43.2%/min。相對於動脈血檢測，用 PDD 檢測的 ICG-PDR 與在動脈血中測得的值之間的偏倚（±2 sd，一致限）在手指探頭是 1.6%/min (–5.2%～ 8.3%/min)，在鼻部探頭是–6.0%/min (–15.5% ～ 3.4%/min)。
結論：沒有肝功能衰竭人群中的 ICG-PDR 值範圍都低於 18%/min，這被引用為肝功能衰竭的臨界值。這一臨界值需要被重新考慮。另外，我們的結論是 ICG 濃度可通過 PDD 充分地無侵入性地檢測。
(王宏翻譯，馬皓琳、李士通校)
BACKGROUND: Indocyanine green plasma disappearance rate (ICG-PDR) is used to evaluate hepatic function. Although hepatic failure is generally said to occur with an ICG-PDR <18%/min, ICG disappearance rate is poorly defined in the healthy population, and a clear cutoff value of ICG-PDR that discriminates between normal hepatic function and hepatic failure has not yet been described. We therefore defined the ICG disappearance rate in an otherwise healthy patient population. In addition, we evaluated the noninvasive measurement of ICG-PDR (transcutaneously by pulse dye densitometry [PDD] at the finger and the nose) and compared these with the simultaneously performed invasive measurements of ICG-PDR (in arterial blood).
METHODS: In patients without signs of liver disease, scheduled for elective nonhepatic surgery, 10 mg ICG was administered IV and ICG-PDR measured by PDD (DDG-2001, Nihon Kohden, Tokyo, Japan). In a subset of patients, arterial blood samples were gathered to compare PDD with invasive ICG measurements. Methods were compared using Bland-Altman analysis. The results of our study and reported studies on discriminative use of ICG-PDR in assessing liver failure were used to construct receiver operating characteristic curves.
RESULTS: Forty-one patients were studied: 33 using the finger probe and 8 using the nose probe. The mean ± sd noninvasive ICG-PDR in this patient population is 23.1% ± 7.9%/min (*n* = 41) with a range of 9.7% to 43.2%/min. Bias (±2 sd, limits of agreement) for ICG-PDR measured by PDD compared with those measured in arterial blood were 1.6%/min (–5.2% to 8.3%/min) for the finger probe and –6.0%/min (–15.5% to 3.4%/min) for the nose probe.
CONCLUSION: ICG-PDR values in a population without liver failure ranged well below 18%/min, cited as the cutoff value for hepatic failure. This cutoff value needs reconsideration. In addition, we conclude that the ICG concentration is adequately determined noninvasively by PDD.

無纖維支氣管鏡輔助下的 Univent 管置管

Placement of the Univent Tube Without Fiberoptic Bronchoscope Assistance

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背景：在此項研究中，我們通過與纖維支氣管鏡（FOB）引導下或者根據廠商推薦的盲插技術放置 Univent 管（Fuji 系統，日本，東京）比較，評估在聽診（AUS）或者發光探條（LS）輔助下放置 Univent 管的可行性和準確性。
方法：80 位 ASA I–II 級擇期行胸科手術需單肺通氣的成人患者，根據放置 Univent 管方法的不同隨機分為 4 組：廠商推薦（MR）組（n=20）；FOB 組（n=20）; AUS 組（n=20）和 LS 組（n=20 ）。在麻醉誘導後，由同一位麻醉醫生應用一種上述方法使用直接硬質喉鏡進行 Univent 管插管並定位。然後由另外一位麻醉醫生使用 FOB 檢查導管位置。記錄導管定位成功所需的嘗試次數、阻塞套囊打氣所需空氣容量和插管時間以及單肺通氣的時間和潛在支氣管損傷併發症。
結果：AUS 組和 LS 組插管時間分別是 182 ± 42 秒和 176 ± 50 秒，比 FOB 組（278 ± 111 秒）和 MR 組（266 ± 127 秒）短（$P < 0.05$）。在 AUS、 LS 和 MR 組，第一次插管將支氣管阻塞套囊插入左主支氣管成功率分別是 100%、79%和 25%。MR 組的阻塞套囊放置嘗試次數和套囊打氣的空氣容量顯著高於 AUS 和 LS 組（$P < 0.05$）。平臥位時，MR 組的支氣管阻塞套囊位置滿意率為 14/20（70%)，顯著少於 FOB 組（18/20，90%）（$P < 0.05$）。AUS 組和 LS 組的支氣管阻塞套囊位置滿意率分別為 19/20（95%）和 16/20（80%）。病人側臥後，在 MR 組，支氣管阻塞套囊位置滿意率為 10/18（56%），顯著少於 FOB 組（17/19，89.5%）（$P < 0.05$）。在 AUS 和 LS 組，滿意率分別為 15/20(75%) 和 15/19（79%）。
結論：在 AUS 或 LS 輔助下放置 Univent 管是可行的，使用這兩種技術時，耗時均比使用 FOB 或者廠商推薦的方法放置時間短。
(沙歡歡 譯 馬皓琳 李士通校)

BACKGROUND: In this study, we evaluated the feasibility and accuracy of Univent tube (Fuji Systems, Tokyo, Japan) placement with the aid of auscultation (AUS) or as guided by a lighted stylet (LS) compared with placement guided by the fiberoptic bronchoscope (FOB) or the blind intubation technique as recommended by the manufacturer's guidelines.
METHODS: Eighty ASA physical status I–II adult patients requiring single-lung ventilation for elective thoracic surgery were randomly allocated into 4 groups according to the method used for Univent tube positioning: manufacturer-recommended (MR) group ($n = 20$); FOB group ($n = 20$); AUS group ($n = 20$); and LS group ($n = 20$). Tracheal placement of the Univent tube was accomplished with direct rigid laryngoscopy after anesthetic induction and was positioned by the same anesthesiologist using 1 of the above-described methods. Its position was then checked by another anesthesiologist with an FOB. The number of attempts required for successful tube positioning, the volume of air needed for blocker cuff inflation, and intubation times were recorded, as were the times for single-lung ventilation and the potential for bronchial injury.

RESULTS: The intubation time was 182 ± 42 s in the AUS group and 176 ± 50 s in the LS group, shorter than that in the FOB (278 ± 111 s) and MR (266 ± 127 s) ($P < 0.05$) groups. The success rate of bronchial blocker insertion into the left bronchus on the first attempt was 100% in the AUS group, 79% in the LS group, and 25% in the MR group. The number of blocker insertion attempts and the volume of air in the blocker cuff in the MR group were significantly higher than those in the AUS and LS ($P < 0.05$) groups. In the supine position, the number of acceptable bronchial blocker placements was 14 of 20 attempts (70%) in the MR group, significantly fewer than that in the FOB group (18 of 20, 90%) ($P < 0.05$). In the AUS and LS groups, the number of acceptable bronchial blocker placements was 19 of 20 (95%) and 16 of 20 (80%), respectively. After patients were turned to the lateral decubitus position, the number of acceptable bronchial blocker placements was 10 of 18 (56%) in the MR group, significantly fewer than that in the FOB group (17 of 19, 89.5%) ($P < 0.05$). In the AUS and LS groups, the number of acceptable bronchial blocker placements was 15 of 20 (75%) and 15 of 19 (79%), respectively.
CONCLUSIONS: The placement of the Univent tube with the aid of AUS or an LS is feasible, and both techniques require less time than placement aided by an FOB or as recommended by the manufacturer.

灌注壓對實驗性胃管模型的胃組織血流量的影響

The Effect of Perfusion Pressure on Gastric Tissue Blood Flow in an Experimental Gastric Tube Model

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背景：吻合口瘺及狹窄的形成仍然是食管癌患者行食管切除術及胃管重建術後嚴重併發症。由於在胃管近端的吻合口灌注完全取決於微循環，使之容易出現灌注不足。我們推測，增加灌注壓可以改善胃管吻合口處的血流量。
方法：9 頭豬行胃管重建術。通過鐳射散斑成像和溫度記錄成像分別測量胃管基部、中間部、近吻合口處以及頂部的血流量和溫度。取平均動脈壓（MAP）50-110 毫米汞柱作為分段測量點。
結果：除了 MAP，實驗中血流動力學總體沒有改變。在每一個相同水準 MAP，胃管頂部血流量明顯低於基部和中間部。升高 MAP 並未對胃管任何位置的血流量造成顯著影響。溫度分佈與不同部位流量分佈相似。升高 MAP 沒有改變胃管任何位置的溫度。
結論：胃管上部的血流量相比更近端的部位有所下降。胃組織血流量不隨灌注壓增加而上升。因此，不建議通過升高 MAP 到超常水準來增加吻合口組織血流量以及減少術後併發症。
(宋村笛 譯 馬皓琳 李士通校)
BACKGROUND: Anastomotic leakage and stricture formation remain an important surgical challenge after esophagectomy with gastric tube reconstruction for cancer of the esophagus. The perfusion of the anastomotic site at the proximal site of the gastric tube depends exclusively on

the microcirculation, making it susceptible to hypoperfusion. We hypothesized that increasing the perfusion pressure would improve blood flow at the anastomotic site of the gastric tube.
METHODS: A gastric tube was reconstructed in 9 pigs. Laser speckle imaging and thermographic imaging were used to measure blood flow and temperature, respectively, at the base, medial part, future anastomotic site, and top of the gastric tube. Measurements were repeated at every stepwise increase of mean arterial blood pressure (MAP) from 50 to 110 mm Hg.
RESULTS: Besides MAP, global hemodynamics did not change throughout the experiment. The blood flow in the top of the gastric tube was significantly lower than the flow in the base and medial part of the gastric tube at all levels of MAP. Increasing MAP did not have a significant effect on blood flow at any location in the gastric tube. Distribution of temperature was similar to distribution of flow for the different locations. Increases in MAP did not change temperature values at any location of the gastric tube.
CONCLUSION: Blood flow in the upper part of the gastric tube is decreased compared with more proximal sites. Gastric tissue blood flow does not increase with increased perfusion pressure. Therefore, it is not recommended to increase MAP to supranormal levels to increase anastomotic tissue blood flow and reduce postoperative complications.

產科麻醉的新熱點：2009 Gerard W.Ostheimer 講座

What's New in Obstetric Anesthesia: The 2009 Gerard W. Ostheimer Lecture
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Anesth Analg 2010; 110:564-569

本文總結了 2008 年以來與產科麻醉密切相關的出版物。在 70 多本被認為對產科麻醉實踐最有影響的英文刊物中的數千篇文章中選出 42 篇。
(龔寅 譯 馬皓琳 李士通 校)
This article summarizes the most relevant publications in obstetric anesthesiology from 2008. Forty-two articles were selected from a pool of several thousand in >70 English-language journals that were deemed as having the most impact on the practice of obstetric anesthesia.

頸動脈內膜剝脫術後發生頸部血腫病人的氣道管理

Airway Management in Patients Who Develop Neck Hematomas After Carotid Endarterectomy
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Anesth Analg 2010; 110:588-593

背景：頸動脈內膜剝脫術後病人由於頸部血腫或水腫而發生的進行性氣道受壓是令人擔心的併發症。儘管如此，關於此人群中氣道管理技術和病人轉歸之間的關係還沒有得到系統性的研究。我們報導了對頸動脈內膜剝脫術後患者運用各項技術進行氣道管理的成功率。

方法：我們回顧分析了明尼蘇達州羅切斯特市梅奧診所十年來在頸動脈內膜剝脫術後 72 小時內需要進行氣道管理用於頸部探查的病人。
結果：在十年裡我們的醫療機構中共有 3225 名患者進行了頸動脈內膜剝脫手術。44 名患者（1.4%）由於頸部血腫需要進行頸部探查術，其中有 42 名需要在頸部探查術前立即進行氣道管理（另外 2 名病人在頸動脈內膜剝脫術後氣道導管尚未拔除）。從 CEA 完成到重回手術室進行血腫清除的平均時間是 6.0 ± 6.0 小時(平均值± 標準差; 範圍, <1-32 小時)。在麻醉誘導前進行纖維支氣管鏡插管的 20 名患者中有 15 名（75%）成功，剩下的 5 名患者纖維支氣管鏡插管失敗後改用直接喉鏡法插管也成功了（3 名患者在麻醉誘導前完成了插管，2 名在誘導後完成了插管）。其餘 22 名患者首先用直接喉鏡作為氣道管理方法，而未嘗試用纖維支氣管鏡。在未進行麻醉誘導的情況下直接喉鏡法插管的成功率是 5/7（71%），而進行全麻誘導後直接喉鏡法插管的成功率是 13/15（87%）。血腫減壓使 4 名直接喉鏡下氣管插管失敗的患者中的 3 名直接喉鏡下插管成功，剩下的 1 名患者則做了氣官切開。用於血腫清除的喉鏡插管過程中未遇到困難的患者中有 36%發現有動脈出血點，而發生困難插管的患者概率為 6%(*P* = 0.03)。44 名患者中有 36 名(82%)在頸部探查術後 24 內就拔除了氣管導管。沒有氣道管理相關併發症發生。血腫清除術後 2 周沒有患者死亡。
結論：在全麻誘導前後我們可以採用多種方法成功地進行氣道控制。既可以選用可視纖維支氣管鏡插管也可以選用直接喉鏡。在聲門暴露困難的情況下，通過外科切開使氣道減壓可方便插管。
(薑旭暉 譯 馬皓琳 李士通 校)
BACKGROUND: Progressive airway compromise from neck hematoma and edema is a feared complication of carotid endarterectomy (CEA). Despite this, the relationship of airway management technique to patient outcome has not been systematically studied in this population. We report the rate of successful airway management using various techniques in post-CEA patients.
METHODS: A 10-year retrospective analysis was conducted to identify patients requiring airway management for neck exploration within 72 hours after CEA at Mayo Clinic, Rochester, MN.
RESULTS: Three thousand two hundred twenty-five patients underwent CEA over a 10-year period at our institution. Forty-four (1.4%) required neck exploration for hematoma, and 42 of these required airway management immediately before neck exploration surgery. (The tracheal tube had not been removed after CEA in the remaining 2 patients.) The average interval between the completion of CEA and return to the operating room for hematoma evacuation was 6.0 ± 6.0 hours (mean ± sd; range, <1-32 hours). Fiberoptic airway management, performed before the induction of anesthesia, was successful in 15 of 20 patients (75%) and, in patients in whom fiberoptic tracheal intubation failed, direct laryngoscopy (DL) was successful in all 5 (3 before and 2 after the induction of general anesthesia). In the remaining 22 patients, DL was used as the initial management technique without a trial of fiberoptic intubation. DL was successful in 5 of 7 patients (71%) when performed before induction of general anesthesia and was successful in 13 of 15 patients (87%) when performed after induction of general anesthesia. Hematoma decompression facilitated DL in 3 of 4 failures of DL; tracheostomy was performed in the remaining patient. An arterial site of bleeding was subsequently identified in 36% of patients in whom no difficulty was encountered during laryngoscopy for hematoma evacuation versus 6% in whom difficulty was noted (*P* = 0.03). In 36 of 44 patients (82%), the tracheal tube was removed

within 24 hours of surgery for neck exploration. No adverse events related to airway management were noted. There were no deaths at 2 weeks after hematoma evacuation.
CONCLUSIONS: Multiple techniques resulted in successful airway control both before and after the induction of general anesthesia. Tracheal intubation was accomplished with both fiberoptic visualization and DL. In instances of poor direct visualization of the glottis, decompression of the airway by opening of the surgical incision may facilitate intubation of the trachea.

啟動大鼠 脊髓α-2 腎上腺受體而非 μ-阿片受體，降低鞘內注射 N-甲基-D-天冬氨酸（NMDA）所致脊髓 NR1 亞單位磷酸化增加和感受傷害行為學改變

Activation of Spinal α-2 Adrenoceptors, but Not μ-Opioid Receptors, Reduces the Intrathecal *N*-Methyl-d-Aspartate-Induced Increase in Spinal NR1 Subunit Phosphorylation and Nociceptive Behaviors in the Rat

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背景：我們實驗室先前的研究顯示,神經病理模型大鼠鞘內(IT)注射α-2 腎上腺受體激動劑可樂定產生的抗痛覺過敏作用與脊髓 NMDA 受體 NR1 亞單位磷酸化(pNR1)的明顯降低有關。我們在本研究中驗證鞘內注射可樂定或者 μ 阿片受體的激動劑[d-Ala2, NMe-Phe4, Gly-ol5]-腦啡肽(DAMGO)是否能減輕注射 NMDA 所致的自發痛和 pNR1 表達增加。
方法：我們檢驗了注射可樂定(20 μg/大鼠)和 DAMGO (1 μg/大鼠)對鞘內注射 NMDA 所致的自發感受傷害行為學和脊髓背側角 pNR1 表達的影響。同時，驗證可樂定的作用是否通過α-2A 或 α-2C 腎上腺素能受體介導。最後，對大鼠脊髓 pNR1、α-2A 或 α-2C 腎上腺受體或 μ-阿片受體進行雙重染色免疫組化處理。
結果：鞘內給予可樂定而不是 DAMGO ，明顯降低了 NMDA 所致 pNR1 表達和感受傷害性行為的增加。可樂定的止痛作用可以被α-2A (BRL44408, 30 μg/大鼠) 或者 α-2C (JP-1302, 50 μg/大鼠)腎上腺受體拮抗劑所阻斷。此外，免疫細胞化學顯示脊髓 pNR1 免疫反應細胞同時含有α-2A 和 α-2C 腎上腺素受體。
結論：結果顯示，啟動脊髓背側角α-2 腎上腺受體而非 μ-阿片受體明顯降低鞘內注射 NMDA 所致 pNR1 表達和感受傷害性行為的增加。此外，這些發現顯示脊髓 NR1 磷酸化的調節與鞘內注射可樂定對突觸後神經元活性的作用相關。
(江繼宏 譯 馬皓琳 李士通 校)

BACKGROUND: A previous study from our laboratories showed that a significant reduction in spinal *N*-methyl-d-aspartate (NMDA) receptor NR1 subunit phosphorylation (pNR1) is associated with the antiallodynic effect produced by intrathecal (IT) injection of the α-2

adrenoceptor agonist, clonidine, in neuropathic rats. In this study, we determined whether the spontaneous pain and increased pNR1 expression induced by NMDA injection are reduced by IT injection of either clonidine or the μ-opioid receptor agonist, [d-Ala2, NMe-Phe4, Gly-ol5]-enkephalin (DAMGO).
METHODS: We examined the effect of clonidine (20 μg/rat) or DAMGO (1 μg/rat) injection on IT NMDA-induced spontaneous nociceptive behavior and pNR1 expression in the spinal dorsal horn. We also determined whether the effect of clonidine is mediated by α-2A or α-2C adrenoceptors. Finally, rat spinal cords were immunohistochemically processed for double staining of pNR1 and α-2A or α-2C adrenoceptors or μ-opioid receptors.
RESULTS: The NMDA-induced increase in both pNR1 expression and nociceptive behavior was significantly reduced by IT clonidine but not DAMGO. This analgesic effect of clonidine was blocked by administration of either an α-2A (BRL44408, 30 μg/rat) or an α-2C (JP-1302, 50 μg/rat) adrenoceptor antagonist. In addition, immunocytochemistry revealed that spinal pNR1 immunoreactive cells co-contain α-2A and α-2C adrenoceptors.
CONCLUSIONS: These results demonstrate that the IT NMDA-induced increase in pNR1 expression and nociceptive behavior is significantly reduced by activation of α-2 adrenoceptors, but not μ-opioid receptors, in the spinal cord dorsal horn. Furthermore, these findings suggest that the modulation of spinal NR1 phosphorylation is linked to the effect of IT clonidine on postsynaptic neuronal activity.

心肺動脈分流手術對小鼠全身白細胞介素-6 釋放，腦核因數-κB 表達和神經認知功能的影響

The Impact of Cardiopulmonary Bypass on Systemic Interleukin-6 Release, Cerebral Nuclear Factor-kappa B Expression, and Neurocognitive Outcome in Rats

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背景：行心肺動脈分流手術（CPB）的心臟手術後出現神經認知功能障礙一直以來都影響著患者的生存品質，炎症反應可能是造成這一後果的原因之一。我們設計這個實驗就是為了研究小鼠在心肺動脈分流手術後圍手術期全身白細胞介素-6（IL-6）的濃度，腦核因數-κB（NF-κB）的表達以及神經認知功能的狀況。氧合器大小對這些結果的影響也一併被監測研究。
方法：小鼠被隨機分為四組：控制對照組（7 例，未行麻醉）；偽手術組（10 例，行麻醉，插套管，但未行心肺動脈分流手術）；兩組手術組，一組（10 例）行麻醉，插套管，並用一台小容量的小鼠氧合器進行 90 分鐘的心肺動脈分流手術；另一組（10 例）行麻醉，插套管，並用一台新生兒氧合器進行 90 分鐘的心肺動脈分流手術。分別於心肺動脈分流手術前、終止時以及兩小時後（或相同時間點）監測全身白細胞介素-6。用免疫組化法於術後 21 天測海馬腦核因數-κB 的表達值。以術前模擬洞板實驗測試結果作為神經認知功能的基礎值，於術後 21 天複測。

結果：兩組手術組的全身白細胞介素-6 的水平均比偽手術組高；相較於小鼠氧合器組，新生兒氧合器組的全身白細胞介素-6 水準在術後 2 小時大大增高（手術組/小鼠氧合器：220pg/mL[16-415]；手術組/新生兒氧合器：1400pg/mL[592-5812]）（P< 0.05）。實驗組海馬腦核因數-κB 的值比控制對照組要高（10+/- 4）。相較於偽手術組（173+/- 24），實驗組更多地表達為腦核因數-κB 陽性神經元。（手術組/新生兒氧合器 271+/- 57，手術組/小鼠氧合器 269+/- 72）。各個組的神經認知及行為功能均未改變，無法比較。
結論：實驗表明，心肺動脈分流手術所引起的全身炎症反應以及海馬腦核因數-κB 的表達與術後神經認知功能的損傷並不相關。這也就提示，除外心肺動脈分流手術以及炎症反應，可能是其他原因導致了心臟手術後神經認知功能的損傷。
(單嘉琪譯 薛張綱校)

BACKGROUND: Neurocognitive deficits after cardiac surgery with cardiopulmonary bypass (CPB) continue to affect patients' quality of life, and an inflammatory reaction may be one of the contributors. We designed this experiment to study perioperative systemic interleukin-6 (IL-6) concentrations, cerebral expression of nuclear factor-kappa B (NF-[kappa]B), and neurocognitive outcome after CPB in young rats. The impact of oxygenator size on these outcomes was also assessed.
METHODS: Rats were randomly assigned to 1 of 4 groups: control (n = 7, nonanesthetized), sham-operated rats (n = 10, anesthetized, cannulated, and not connected to CPB), and 2 CPB groups, anesthetized, cannulated, and subjected to 90 min of CPB, using either a small-volume rat oxygenator (CPB/rat oxygenator, n = 10) or a neonate oxygenator (CPB/neonate oxygenator, n = 10). Systemic IL-6 was determined before, at the end of, and 2 h after CPB or at equivalent times. Hippocampal NF-[kappa]B expression was assessed on postoperative day 21 using immunohistochemistry. Neurocognitive performance was assessed with the modified hole-board test at baseline and for 21 postoperative days.
RESULTS: Both CPB groups had increased systemic IL-6 levels compared with sham, with the neonate oxygenator causing a substantially larger increase at 2 h after CPB compared with the rat oxygenator group (CPB/rat oxygenator: 220 pg/mL [16-415]; CPB/neonate oxygenator: 1400 pg/mL [592-5812]) (P < 0.05). Hippocampal NF-[kappa]B was increased in experimental groups compared with controls (10 +/- 4). CPB resulted in more NF-[kappa]B-positive neurons (271 +/- 57 CPB/neonate oxygenator and 269 +/- 72 CPB/rat oxygenator) compared with sham operation (173 +/- 24). Neurocognitive and behavioral performances were unaltered and comparable among all groups.
CONCLUSIONS: Pronounced systemic inflammatory responses to experimental CPB associated with increased hippocampal expression of NF-[kappa]B were not accompanied by neurocognitive impairment. This suggests that other factors beyond CPB and inflammatory responses might contribute to adverse neurocognitive outcomes after cardiac surgery.

脈壓差與冠脈搭橋術後的長期生存狀況

Pulse Pressure and Long-Term Survival After Coronary Artery Bypass Graft Surgery.

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背景：縱向研究資料顯示脈壓差增寬往往是罹患冠心病及死亡的重要預知因素，但目前仍不清楚其是否會降低冠心病患者冠脈搭橋術後的長期生存率。因此，此項研究旨在評價脈壓差增寬患者在行冠狀動脈搭橋術後的長期生存狀況。
方法：選取 1993 年 1 月至 2004 年 7 月間行冠狀動脈搭橋手術的患者為該項回顧性觀察研究的研究物件，對其中 973 名患者行長期生存狀況評估。將麻醉誘導前自動記錄保存系統中前 3 次血壓測量結果的中間值定為患者的基礎動脈血壓。運用 Cox 比例風險回歸模型評估基礎脈壓差對患者術後生存狀況的影響，同時將所測得的基礎平均動脈壓、收縮壓、舒張壓及是否合併糖尿病、Hannan 風險指數、抑肽酶使用情況、體外迴圈持續時間等作為協同變數一併引入進行分析。
結果：隨訪期間共有 220 例（22.9%）患者死亡（中位數：7.3 年[第一四分位數：5 年，第三四分位數：10 年]），其中 94 例為心腦血管原因。基礎脈壓差增寬是預測患者術後長期生存率下降的重要因素，具有顯著的統計學差異（P<0.001）；此外，Hannan 風險指數（P<0.001）、體外迴圈持續時間（P<0.001）、是否合併糖尿病（P<0.001）等也是具有顯著統計學意義的重要預知因數。基礎動脈收縮壓(P=0.40)、舒張壓(P=0.38)及平均動脈壓(P=0.78)與患者術後長期生存狀況無關。脈壓差的危險比（已對模型中的其它變數進行校正）為每升高 10mmHg1.11(1.05-1.18)。
結論：冠狀動脈搭橋術後患者長期生存狀況不佳與圍手術期脈壓差增寬密切相關。回顧先前所報導的脈壓差與住院患者致命或非致命血管併發症間的聯繫，應考慮將脈壓差修訂並納入已制定的手術危險評估、患者諮詢及治療指南中。
(范羽譯 薛張綱校)

Background: Data from longitudinal studies reveal that widened pulse pressure (PP) is a major predictor of coronary heart disease and mortality, but it is unknown whether PP similarly decreases survival after coronary artery bypass graft (CABG) surgery for coronary heart disease. We therefore assessed long-term survival in patients with increased PP at the time of presentation for CABG surgery.
Methods: In this retrospective observational study of patients undergoing CABG surgery between January 1993 and July 2004, 973 subjects were included for assessment of long-term survival. Baseline arterial blood pressure (BP) measurements were defined as the median of the first 3 measurements recorded by the automated record keeping system before induction of anesthesia. The effect of baseline PP on survival after surgery was evaluated using a Cox proportional hazards regression model and bootstrap resampling with baseline mean arterial BP, systolic BP, diastolic BP, diabetes, Hannan risk index, aprotinin use, and cardiopulmonary bypass time as covariates.
Results: There were 220 deaths (22.9%) during the follow-up period (median, 7.3 yr [Q1: 5, Q3: 10 yr]) including 94 deaths from cardiovascular causes. Increased baseline PP was a significant predictor of reduced long-term survival (P < 0.001) along with Hannan risk index (P < 0.001), duration of cardiopulmonary bypass (P < 0.001), and diabetes (P < 0.001). Baseline systolic (P = 0.40), diastolic (P = 0.38), and mean arterial BPs (P = 0.78) were not associated with long-term survival. The hazard ratio for PP (adjusted for other covariates in the model) was 1.11 (1.05-1.18) per 10-mm Hg increase.
Conclusions: An increase in perioperative PP is associated with poor long-term survival after CABG surgery. Together with our previous report linking PP to in-hospital fatal and nonfatal vascular complications, the established models for surgical risk assessment, patient counseling, and treatment should be revised to include PP.

多科協作共迎止血難題

Multidisciplinary approach to the challenge of hemostasis.

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由麻醉學，血液學，血庫，重症監護和多個外科（包括創傷外科，心臟外科，兒外科，神經外科，婦科及血管科）的專家組成的多學科聯合專家組于2008年1月召開了一次會議專門討論了關於不同臨床情況下的止血方法及出血病人的管理。會議著重關注於術中的相應評估和措施。止血有多種定義，臨床上將之定義為控制活動性出血而不伴有病理性血栓栓塞事件(在促凝和抗凝，纖溶和抗纖溶之間達到平衡點)。止血目前仍存在很多問題，比如止血藥物用於治療時缺乏科學的證據和規範化的指南，需要一個可信而快速的實驗室檢查指標以及病人的個體差異性。臨床上需要有意義的又快速準確的實驗室檢查結果來反映病人當前的出凝血功能從而指導臨床治療決策。當前使用中的常規出凝血功能實驗室檢查(如血小板計數，凝血酶原時間/國際標準化比值和活化部分凝血酶時間)並不能反應人體複雜的出凝血功能狀態，從而有時會誤導臨床工作者。儘管凝血彈性描記法和彈性測定法等這些點狀局部凝血功能監測方法可以提供全面地凝血功能狀態，但是這些監測方法有耗時，難以解讀，需要專門受過培訓的人員操作等缺點。臨床上迫切需要發展出一些實驗室檢查項目來反映抗凝抗血小板藥物的作用效果，預測出血併發症，指導是否需要以及何時需要使用血製品或者藥物治療。予會專家們組成了一個治療出血病人的組織，該組織將會進行多學科間合作勾通促進關於止血治療現狀的討論以及止血的未來研究進展。止血法需要大量研究支援，包括有意義的臨床終點指標合適的研究人群的臨床對照試驗，還有觀察性研究，佇列研究和大樣本量的資料庫。由於保持出凝血功能的平衡非常複雜，仍需要進一步的研究和多學科間的合作以改進對病人的處理改善病人的預後。
(黃劍譯 薛張綱校)

A multidisciplinary panel consisting of experts chosen by the 2 chairs of the group representing experts in anesthesiology, blood banking, hematology, critical care medicine, and various surgical disciplines (trauma, cardiac, pediatric, neurologic, obstetrics, and vascular) convened in January 2008 to discuss hemostasis and management of the bleeding patient across different clinical settings, with a focus on perioperative considerations. Although there are many ways to define hemostasis, one clinical definition would be control of bleeding without the occurrence of pathologic thrombotic events (i.e., when balance among procoagulant, anticoagulant, fibrinolytic, and antifibrinolytic activities is achieved). There are common hemostatic challenges that include lack of scientific evidence and standardized guidelines for the use of therapeutic drugs, need for reliable and rapid laboratory tools for measuring hemostasis, and individual variability. Clinically meaningful and accurate real-time laboratory data reflecting a patient's hemostatic status are needed to guide treatment decisions. Current available routine laboratory tests of hemostasis (e.g., platelet count, prothrombin time/international normalized ratio, and activated partial thromboplastin time) do not reflect the complexity of in vivo hemostasis and can mislead the clinician. Although point-of-care coagulation monitoring tests including measures of

thromboelastography/elastometry provide insight into overall hemostatic status, they are time-consuming to perform, complex to interpret, and require trained personnel. There is a particular need to develop laboratory tests that can measure the effects of anticoagulant and antiplatelet agents for individual patients, predict bleeding complications, and guide therapy when and if treatment with blood products or pharmacologic drugs is required. Formation of an organization comprised of specialists who treat bleeding patients will foster multidisciplinary collaborations and promote discussions of the current state of hemostasis treatment and future priorities for hemostasis research. Controlled trials with clinically meaningful end points and suitable study populations, as well as observational studies, investigator-initiated studies, and large registry and database studies are essential to answer questions in hemostasis. Because of the complexities of maintaining hemostatic balance, advances in hemostasis research and continuing communication across specialties are required to improve patient care and outcomes.

手術解剖學分級對於術後蘇醒室內應用止吐藥物的影響

The Effect of an Anatomically Classified Procedure on Antiemetic Administration in the Postanesthesia Care Unit.

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背景：關於不同手術類型對術後噁心嘔吐（PONV）的影響一直存在著爭議。我們希望通過這個回顧性的資料分析研究來分析不同手術類型（根據解剖學定義來劃分和比較）在術後蘇醒室觀察的 2 小時內對應用止吐治療情況的影響。
方法：我們所使用的腫瘤手術（樣本量 n=18,109）回顧性分析的資料來自於我們的自動化麻醉資訊系統資料庫。我們將手術類型按解剖學定義分為七大類，並將體表肌肉骨骼及淺表手術作為對照組。我們也就另九大噁心嘔吐危險因素對每位患者進行了分析，這九大危險因素為：性別，吸煙狀況，噁心嘔吐史或運動障礙，麻醉時間，預防性止吐藥物的應用，術中阿片類藥物、非甾體類抗炎藥物、硬膜外的應用，以及術後阿片類藥物的應用。在調整平衡其他危險因素的同時，就不同手術類型在術後蘇醒室觀察的 2 小時內對應用止吐治療情況的影響應用多項變異邏輯性回歸的方法進行評估分析。
結果：相較於體表肌肉骨骼及淺表手術，接受神經外科手術（P<0.0001），頭頸部手術（P<0.0001），和腹部手術（P<0.0001）的患者在蘇醒室明顯需要應用更多的止吐藥物，而接受胸外科手術（P=0.02）的患者在蘇醒室所需接受的止吐藥物則明顯較少。乳房或腋窩手術（P=0.74）以及內鏡手術（P=0.28）使用止吐藥物的情況與對照組並無明顯差異。以下幾大因素與術後蘇醒室早期應用止吐藥物明顯相關：女性，不吸煙者，有噁心嘔吐史或運動障礙史，麻醉時間，以及術中或術後應用過阿片類藥物。
結論：通過使用我們的自動化麻醉資訊系統資料庫研究分析，我們發現，就人群而言，根據解剖學定義所劃分的不同類型的手術與術後蘇醒室早期應用止吐藥物的頻率增多有關。
(李瑩譯 薛張綱校)

BACKGROUND: The effect of the type of surgical procedure on postoperative nausea and vomiting (PONV) rate has been debated in the literature. Our goal in this retrospective database study was to investigate the effect the type of surgical procedure (categorized and compared anatomically) has on antiemetic therapy within 2 h of admission to the postanesthesia care unit (PACU).
METHODS: We retrospectively analyzed data for oncology surgeries (n = 18,109), from our automated anesthesia information system database. We classified the types of surgical procedures anatomically into seven categories, with the integumentary musculoskeletal and the superficial surgeries chosen as the referent group. Our analysis included nine other risk factors for each patient, such as gender, smoking status, history of PONV or motion sickness, duration of anesthesia, number of prophylactic antiemetics administered, intraoperative opioids, ketorolac, epidural use, and postoperative opioids. Multivariate logistic regression was used to assess the effect of the type of surgery on antiemetic administration within the first 2 h of PACU admission, while adjusting for the other risk factors.
RESULTS: Compared with integumentary musculoskeletal and superficial surgeries, patients undergoing neurological ($P < 0.0001$), head or neck ($P < 0.0001$), and abdominal ($P < 0.0001$) surgeries were administered PACU antiemetic significantly more often, whereas patients undergoing thoracic surgeries were administered PACU antiemetic significantly less often ($P = 0.02$). Breast or axilla ($P = 0.74$) and endoscopic ($P = 0.28$) procedures did not differ from the referent category. Female, nonsmoker, history of PONV or motion sickness, anesthesia duration, and intraoperative and postoperative opioid administration were significantly associated with antiemetic administration during early PACU admission.
CONCLUSIONS: Using our automated anesthesia information system database, we found that the type of surgery, when categorized anatomically, was associated with an increased frequency of early PACU antiemetic administration in our population.

吸入麻醉藥在老年阿茨海默症小鼠中的效能

Inhaled Anesthetic Potency in Aged Alzheimer

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背景：隨著人口老齡化，需要手術治療的明顯或初期阿茨海默症老年人數量在增加。全麻可能加重阿茨海默症的症狀和病理改變，所以減少麻醉暴露可能很重要。我們需要知道阿茨海默症持續性的病理改變是否會改變麻醉藥效能。
方法：在 12 至 14 月齡的三重轉基因阿茨海默症(3xTgAD)小鼠為實驗組和以野生型 C57BL6 小鼠為對照組的研究中，用 MAC 來觀察以翻正反射消失為終點時異氟醚、氟

烷、和七氟醚的誘導效能和起效時間。三重轉基因阿茨海默症小鼠的模型通過 APP_{Swe}, Tau, 和 PS1 人類轉基因這三個與人類阿茨海默症家族型相關的基因而實現。
結果：三重轉基因阿茨海默症小鼠對吸入麻醉藥稍有抵抗（從 8%至 30%不等），但在起效時間方面三種吸入麻醉藥在兩組間沒有差別。
結論：該結果顯示阿茨海默症的基因易感性和病理改變使之對三種吸入麻醉藥的催眠作用敏感性下降。起效時間沒有改變。
(姚敏敏譯 薛張綱校)
BACKGROUND: The number of elderly patients with frank or incipient Alzheimer's disease (AD) requiring surgery is growing as the population ages. General anesthesia may exacerbate symptoms of and the pathology underlying AD, so minimizing anesthetic exposure may be important. This requires knowledge of whether the continuing AD pathogenesis alters anesthetic potency.
METHODS: We determined the induction potency and emergence time for isoflurane, halothane, and sevoflurane using the minimum alveolar anesthetic concentration for loss of righting reflex as an end point in 12- to 14-mo-old triple transgenic Alzheimer (3xTgAD) mice and wild type C57BL6 controls. 3xTgAD mice model AD by harboring three distinct mutations: the APP_{Swe}, Tau, and PS1 human transgenes, each of which has been associated with familial forms of human AD.
RESULTS: The 3xTgAD mice exhibited mild resistance (from 8% to 30%) to volatile anesthetics but displayed indistinguishable emergence patterns from all three inhaled anesthetics.
CONCLUSIONS: These results show that the genetic vulnerabilities and neuropathology associated with AD produce a small but significant decrease in sensitivity to the hypnotic actions of three inhaled anesthetics. Emergence times were not altered.

麻醉藥導致的神經元凋亡以及應對方法

The Young: Neuroapoptosis Induced by Anesthetics and What to Do About It
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Anesth Analg 2010 110: 442-448

在美國及全世界每年有數以百萬計的胎兒、嬰兒、和兒童暴露于麻醉藥。在神經發育的關鍵階段應用麻醉藥物被認為是安全的，且沒有長期不良結果。然而，近期的報導提供的很多證據顯示在快速突觸形成期，即大腦生長突增期，未成熟動物大腦暴露於麻醉藥會引起廣泛神經元退化凋亡，抑制神經發生，導致嚴重的長期神經認知功能損害。在這裡，我們總結了現有麻醉藥導致的大腦病理改變及相關的長期神經認知缺陷方面的證據，討論如何保證麻醉藥物有益作用同時制定保護大腦避免潛在損害的有用方法。
(姚敏敏譯 薛張綱校)
Millions of human fetuses, infants, and children are exposed to anesthetic drugs every year in the United States and throughout the world. Anesthesia administered during critical stages of neurodevelopment has been considered safe and without adverse long-term consequences. However, recent reports provide mounting evidence that exposure of the immature animal brain to anesthetics during the period of rapid synaptogenesis, also known as the brain growth spurt

period, triggers widespread apoptotic neurodegeneration, inhibits neurogenesis, and causes significant long-term neurocognitive impairment. Herein, we summarize currently available evidence for anesthesia-induced pathological changes in the brain and associated long-term neurocognitive deficits and discuss promising strategies for protecting the developing brain from the potentially injurious effects of anesthetic drugs while allowing the beneficial actions of these drugs to be realized.

缺少 N-甲基-d-天冬氨酸受體亞單位 GluR1 的突變小鼠中異氟醚和一氧化氮固定性減小由基因敲除的激發效應引起

Reduced Immobilizing Properties of Isoflurane and Nitrous Oxide in Mutant Mice Lacking the N-Methyl-d-Aspartate Receptor GluR1 Subunit Are Caused by the Secondary Effects of Gene Knockout

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Anesth Analg 2010 110: 461-465

背景：直到最近，N -甲基- D -天冬氨酸（NMDA）受體被認為可能能夠介導吸入麻醉藥如異氟醚和一氧化氮所產生的固定性。然而，新的證據顯示這種受體在取消此運動反應中並沒有以前想像的重要。為了提供對於這一觀點進一步的支援或者質疑的證據，我們研究了在敲除亞單位 GluR 1 造成 NMDA 受體功能障礙的轉基因動物身上異氟醚和一氧化氮的固定性能。
方法：小鼠中吸入麻醉藥的固定性能通過最低有效肺泡濃度（MAC）來定量，由經典的夾尾試驗評估。
結果：和野生型對照組相比，NMDA 受體亞單位 GluR1 敲除的小鼠中異氟醚的 MAC 值更大，顯示了其對異氟醚固定性的反作用。在以前的研究中顯示敲除小鼠具有由遺傳操縱引起的擴大的單胺類活性，而由此引起的 MAC 值增加可以在我們的實驗中通過 5 羥色胺 2A 受體拮抗劑酮舍林或者多巴胺 D2 受體拮抗劑氟呱啶的預處理來廢除，而這種劑量在野生型動物中不會影響 MAC 值。突變小鼠同時表現出對異氟醚對於一氧化氮 MAC 值減小效應的反作用，但是這種反作用可能同樣通過酮舍林或氟呱啶被廢除。因此，基因敲除小鼠中吸入麻醉藥的固定作用減小可能繼發于增加的單胺活性，而非直接由 NMDA 受體功能受損引起。
結論：我們的結果證實，NMDA 受體對於異氟醚和一氧化氮的固定作用並沒有重大貢獻。此外，他們表明這種改變的能力繼發于基因操縱，從而影響全球基因敲除研究獲得的結果。
(俞佳譯 薛張綱校)
BACKGROUND: Until recently, the N-methyl-d-aspartate (NMDA) receptor was considered to possibly mediate the immobility produced by inhaled anesthetics such as isoflurane and nitrous oxide. However, new evidence suggests that the role of this receptor in abolition of the movement response may be less important than previously thought. To provide further evidence supporting or challenging this view, we examined the anesthetic potencies of isoflurane and

nitrous oxide in genetically modified animals with established NMDA receptor dysfunction caused by GluR 1 subunit knockout.
METHODS: The immobilizing properties of inhaled anesthetics in mice quantitated by the minimum alveolar anesthetic concentration (MAC) were evaluated using the classic tail clamp method.
RESULTS: Compared with wild-type controls, NMDA receptor GluR 1 subunit knockout mice displayed larger isoflurane MAC values indicating a resistance to the immobilizing action of isoflurane. Knockout mice were previously shown to have enhanced monoaminergic tone as a result of genetic manipulation, and this increase in MAC could be abolished in our experiments by pretreatment with the serotonin 5-hydroxytryptamine type 2A receptor antagonist ketanserin or with the dopamine D2 receptor antagonist droperidol at doses that did not affect MAC values in wild-type animals. Mutant mice also displayed resistance to the isoflurane MAC-sparing effect of nitrous oxide, but this resistance was similarly abolished by ketanserin and droperidol. Thus, resistance to the immobilizing action of inhaled anesthetics in knockout mice seems to be secondary to increased monoaminergic activation after knockout rather than a direct result of impaired NMDA receptor function.
CONCLUSIONS: Our results confirm recent findings indicating no critical contribution of NMDA receptors to the immobility induced by isoflurane and nitrous oxide. In addition, they demonstrate the ability of changes secondary to genetic manipulation to affect the results obtained in global knockout studies.

1987 至 2006 年北美的惡性高熱病人的臨床表現、治療和併發症

Clinical Presentation, Treatment, and Complications of Malignant Hyperthermia in North America from 1987 to 2006

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背景：我們分析了北美惡性高熱管理處接到的惡性高熱病例的臨床表現、治療和併發症報告。
方法：我們的入選標準：1987 年 1 月 1 日之 2006 年 12 月 31 日遞交的 AMRA（對麻醉的異常代謝或骨骼肌反應）報告；臨床分級量表列為“很像”或“幾乎肯定”的惡性高熱；美國或加拿大居民；超過一種麻醉用藥的。排除標準：病理學除外惡性高熱；為了明確該併發症，病人使用丹曲林後療效不確切或很小的被排除。應用 Wilcoxon 秩和和 Pearson 精確χ^2 檢驗。一個根據 Hosmer-Lemeshow 標準逐步確診的惡性高熱多樣化模型建立。
結果：一個以青年男性（74.8%）為主的樣本。總共有 6.5%的人有惡性高熱家族史；152 名惡性高熱患者中有 77 名報導了超過 2 例的不確切的既往全麻經歷。10 例皮膚溫度未升高。常見的惡性高熱前兆體征為高碳酸血症、竇性心動過速或咬肌痙攣。63.5% 的病人體溫異常（中位最大值為 39.1°C）較上述三個體征早出現。78.6%的病人同時出現肌溶解和呼吸性酸中毒，只有 26.0%的病人有代謝性酸中毒。丹曲林的總劑量的中位數是 5.9 mg/kg（q1/4 為 3.0 mg/kg，q3/4 為 10.0 mg/kg），22 名患者未接受丹曲林且痊癒了。53.9%的病人接受了碳酸氫鹽治療。併發症不包括復發、心跳驟停或死亡，這些出現在

181 名惡性高熱患者中的 63 名患者身上。21 名患者體溫<41.6°C（人類極限高溫）出現了血液或神經系統併發症。最高體溫每上升 2°C 併發症出現的可能性增加 2.9 倍，使用丹曲林治療 30 分鐘後為 1.6 倍。
結論：體溫上升可能是惡性高熱的早期體征。體溫上升發生頻率最高，1/3 發生代謝性酸中毒，全麻過程中精確的體溫監測和早期的丹曲林治療可降低 35%的惡性高熱發病率。(張玥琪譯，薛張綱校)
BACKGROUND: We analyzed cases of malignant hyperthermia (MH) reported to the North American MH Registry for clinical characteristics, treatment, and complications.
METHODS: Our inclusion criteria were as follows: AMRA (adverse metabolic/musculoskeletal reaction to anesthesia) reports between January 1, 1987 and December 31, 2006; "very likely" or "almost certain" MH as ranked by the clinical grading scale; United States or Canadian location; and more than one anesthetic drug given. An exclusion criterion was pathology other than MH; for complication analysis, patients with unknown status or minor complications attributable to dantrolene were excluded. Wilcoxon rank sum and Pearson exact $\chi 2$ tests were applied. A multivariable model of the risk of complications from MH was created through stepwise selection with fit judged by the Hosmer-Lemeshow statistic.
RESULTS: Young males (74.8%) dominated in 286 episodes. A total of 6.5% had an MH family history; 77 of 152 patients with MH reported ≥2 prior unremarkable general anesthetics. In 10 cases, skin liquid crystal temperature did not trend. Frequent initial MH signs were hypercarbia, sinus tachycardia, or masseter spasm. In 63.5%, temperature abnormality (median maximum, 39.1°C) was the first to third sign. Whereas 78.6% presented with both muscular abnormalities and respiratory acidosis, only 26.0% had metabolic acidosis. The median total dantrolene dose was 5.9 mg/kg (first quartile, 3.0 mg/kg; third quartile, 10.0 mg/kg), although 22 patients received no dantrolene and survived. A total of 53.9% received bicarbonate therapy. Complications not including recrudescence, cardiac arrest, or death occurred in 63 of 181 patients (34.8%) with MH. Twenty-one experienced hematologic and/or neurologic complications with a temperature <41.6°C (human critical thermal maximum). The likelihood of any complication increased 2.9 times per 2°C increase in maximum temperature and 1.6 times per 30-minute delay in dantrolene use.
CONCLUSION: Elevated temperature may be an early MH sign. Although increased temperature occurs frequently, metabolic acidosis occurs one-third as often. Accurate temperature monitoring during general anesthetics and early dantrolene administration may decrease the 35% MH morbidity rate.

羥乙基澱粉(HES 130/0.42 and HES 200/0.5)活化腎小管上皮細胞的效應

The effect of hydroxyethyl starches (HES 130/0.42 and HES 200/0.5) on activated renal tubular epithelial cells.

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Anesth Analg. 2010 Feb;110(2):531-40. Epub 2009 Nov 12.
背景：急性腎功能衰竭是敗血症的一個常見併發症。羥乙基澱粉(HES)廣泛應用于此類患者。然而，敗血症時應用 HES 對患者腎功能的影響仍有爭論。我們建立了腫瘤壞死因數

α（TNF-a）引導的人類近端小管上皮細胞(HK-2）的離體模型，來確定 HES 130/0.42 和 HES 200/0.5 對這些活化細胞的效果。
方法：在有 HES 130/0.42 或 200/0.5 的環境下應用 TNF-a 刺激 HK-2 細胞，同時設立空白對照。經過 4，10 和 18 小時的培養後，測定單核細胞趨化蛋白－1(MCP-1)，該蛋白是中性粒細胞和巨噬細胞的重要化學趨化因數。同時進行細胞活性和毒性測定。
結果：在 TNF-a 的刺激下 MCP-1 雙倍表達。在持續 10 小時和 18 小時的刺激期間應用含 2% 和 4% HES 200/0.5 液體，MCP-1 的濃度可下降 26% 和 56% (P < 0.05)。TNF-a 的刺激導致細胞活性顯著下降了 53%-63%，而應用 HES 130/0.42 聯合培養，細胞活性僅下降 32%-40% (P < 0.005)， 應用 HES 200/0.5 聯合培養後細胞活性受 TNF－a 的影響更少 (P < 0.001)。TNF-α 引導的細胞凋亡率因 HES 200/0.5 的應用而減少(P < 0.05)。
結論：這項離體研究顯示兩種羥乙基澱粉產品均可通過炎性刺激調節細胞損傷。HES 200/0.5 較 HES 130/0.42 的效果更顯著，意味著不同類型的羥乙基澱粉之間可能存在不同的生物學效應。
(張釗譯 薛張綱校)
BACKGROUND: Acute renal failure is a frequent complication of sepsis. Hydroxyethyl starch (HES) is widely used in the treatment of such patients. However, the effect of HES on renal function during sepsis remains controversial. We established an in vitro model of tumor necrosis factor-alpha (TNF-alpha)-stimulated human proximal tubular epithelial (HK-2) cells to assess the possible effects of HES 130/0.42 and HES 200/0.5 on these activated cells.
METHODS: HK-2 cells were stimulated with TNF-alpha in the presence or absence of HES 130/0.42 or 200/0.5. After 4, 10, and 18 h of incubation, monocyte chemoattractant protein-1 (MCP-1), a key chemoattractant for neutrophils and macrophages, was measured. In addition, viability and cytotoxicity assays were performed.
RESULTS: MCP-1 expression was doubled upon TNF-alpha exposure. In the presence of 2% and 4% HES 200/0.5 in 98% (96%) medium over a stimulation time period of 10 h and 18 h, the MCP-1 concentration was decreased between 26% and 56% (P < 0.05). TNF-alpha stimulation resulted in a significant decrease of viability by 53%-63%, whereas viability decreased by only 32%-40% in coincubation with HES 130/0.42 (P < 0.005) and remained even less affected by TNF-alpha in the presence of HES 200/0.5 (P < 0.001). The TNF-alpha-induced cell death rate was attenuated in the presence of HES 200/0.5 (P < 0.05).
CONCLUSIONS: This in vitro study shows that both HES products modulate cell injury upon inflammatory stimulation. The effect was more pronounced in the HES 200/0.5 group than for HES 130/0.42, suggesting a possible biological difference between the HES types.

一項使用空氣或鹽水阻力消失試驗鑒定硬膜外間隙有效性的回顧性研究

A Retrospective Effectiveness Study of Loss of Resistance to Air or Saline for Identification of the Epidural Space

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Anesth Analg 2010 110: 558-563.

背景：比價用空氣和鹽水阻力消失試驗來鑒定硬膜外間隙的隨機試驗證明了鹽水的優越性。我們假設在實際臨床工作中麻醉醫師使用他們更擅長的技術包括使用空氣或鹽水都將得到相似的鎮痛結果。

方法：對 929 名產婦要求行椎管內分娩鎮痛的記錄進行回顧性分析，比較鎮痛技術（硬膜外或硬膜外聯合蛛網膜下腔，空氣或鹽水行阻力消失試驗）、鎮痛結果（開始的舒適度，不對稱阻滯，追加病人自控硬膜外鎮痛劑量的需求，導管的更換）及併發症（感覺異常，靜脈內或鞘內置管，意外蛛網膜刺傷）。

結果：929 例分娩鎮痛中，52.6%用空氣進行阻力消失試驗，47.4%使用鹽水。在至少實施過 10 次椎管內阻滯的麻醉醫生中有 82%至少 70%的次數只使用一種物質。空氣組和鹽水組的病人特徵、鎮痛技術和阻滯成功率無差異。對於使用一種物質的操作者，使用一種更擅長的技術意味著更少的穿刺次數(1.3 ± 0.7 比 1.6 ± 0.8, *P* = 0.001)、更少的感覺異常(8.7% 比 18.5%, R= 0.42, *P* = 0.007)、更少的意外蛛網膜刺傷(1.0% 比 4.4%, R= 0.23, *P* = 0.03)。

結論：在麻醉醫生謹慎使用的前提下，用空氣和鹽水行阻力消失試驗定位硬膜外間隙的阻滯成功率無明顯差別。

(朱蘭芳譯，薛張綱校)

BACKGROUND: Randomized trials comparing air to saline for loss of resistance (LOR) for identification of the epidural space have suggested the superiority of saline. We hypothesized that, in actual clinical practice, anesthesiologists using their preferred technique would produce similar analgesic outcomes with either air or saline.

METHODS: The labor analgesia records for 929 parturients requesting neuraxial analgesia were reviewed with respect to technique (epidural or combined spinal-epidural; air or saline for LOR), analgesic outcomes (initial comfort, asymmetry of the block, need for physician top-up during patient-controlled epidural analgesia, and catheter replacement), and complications (paresthesia, IV or intrathecal catheter placement, and unintentional dural puncture).

RESULTS: Of 929 labor analgesics analyzed, 52.6% were performed with LOR to air and 47.4% to saline. Among anesthesiologists who performed at least 10 blocks, 82% used 1 medium at least 70% of the time. There were no differences between the air and saline groups in patient characteristics, analgesic technique, or block success. Among operators with a preference for 1 medium, use of the preferred technique was associated with fewer attempts (1.3 ± 0.7 vs 1.6 ± 0.8, *P* = 0.001), fewer paresthesias (8.7% vs 18.5%, odds ratio = 0.42, *P* = 0.007), and fewer unintentional dural punctures (1.0% vs 4.4%, odds ratio = 0.23, *P* = 0.03).

CONCLUSIONS: When used at the anesthesiologist's discretion, there is no significant difference in block success between air and saline for localization of the epidural space by LOR.

全麻下行頸動脈內膜剝脫術患者在頸動脈鉗夾後，吸入氧濃度和呼末二氧化碳對腦氧合的影響

The Influence of Inspired Oxygen Fraction and End-Tidal Carbon Dioxide on Post–Cross-Clamp Cerebral Oxygenation During Carotid Endarterectomy Under General Anesthesia

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背景：百分之十到十五的行頸動脈鉗閉的患者出現了繼發於大腦低灌注引起的神經功能損傷。上述的損傷可以通過增加吸入氧濃度(Fio_2)來逆轉其損傷，在清醒患者行頸動脈鉗夾術時增加 Fio_2 可以通過改善大腦局部的氧合(rSO_2)。在頸動脈內膜剝脫術中，頸動脈鉗夾期間出現腦血流異常的改善，而在頸動脈鉗夾後正常；通過腦動電流描記法發現，這種異常與低碳酸血症有關。我們設計了這項研究，研究了對於全麻下行頸動脈內膜剝脫術患者在頸動脈鉗夾期間 Fio_2 和呼末二氧化碳($Petco_2$)與 rSO_2 的關係。
方法：20 名患者入選這項研究。10 名行選擇性分流。病人接受全身麻醉。使用 INVOS 5100B 監測儀器(Somanetics Corporation, Troy, MI)測量 rSO_2 頸動脈鉗夾後，根據以下要求連續調節 Fio_2 和分鐘通氣量：Fio_2 30%, $Petco_2$ 30–35 mm Hg; 2) Fio_2 100%, $Petco_2$ 30–35 mm Hg；和 3) Fio_2 100%, $Petco_2$ 40–45 mm Hg。在每個時間點分別記錄手術和非手術處的 rSO_2，並行血氣分析。
結果：分流及沒有分流的患者的結果分開分析。增加 Fio_2: 相比於 Fio_2 30%，非分流組患者給予 100% 的氧氣並維持 $Petco_2$ 在 30–35 mm Hg 範圍可以增加手術處 rSO_2 8% (P = 0.008)，非手術處增加 rSO_2 6% (P = 0.011)。 在分流組患者,相比于 Fio_2 30%，給予 100% 的氧氣並維持 $Petco_2$ 在 30–35 mm Hg 範圍，在手術和非手術處均增加 rSO_2 4% (手術處 P = 0.008，非手術處 P = 0.011)。增加 $Petco_2$: Fio_2 維持在 100%時，相比於 $Petco_2$ 維持在 30–35 mm Hg，對於非分流患者, $Petco_2$ 維持在 40–45 mm Hg，手術處 rSO_2 增加了 6% (P = 0.008)，二非手術處增加了 5% (P = 0.024) 。對於分流患者 Fio_2 維持在 100%，相比於 $Petco_2$ 維持在 30–35 mm Hg，$Petco_2$ 維持在 40–45 mm Hg 時，手術處 rSO_2 增加了 3% (P = 0.018)，非手術處增加了 4% (P = 0.007)。
結論：對於全麻行頸動脈內膜剝脫術患者，頸動脈鉗夾期間增加 Fio_2 可以有效改善 rSO_2。增加 $Petco_2$ 可能也會改善患者的 rSO_2。
(陳珺珺譯 薛張綱校)
BACKGROUND: Ten to fifteen percent of awake patients develop neurological deficits secondary to cerebral hypoperfusion after carotid artery cross-clamping. The reversal of such deficits by increasing the inspired oxygen fraction (Fio_2) has been demonstrated, and regional cerebral oxygenation (rSO_2) has been shown to improve during carotid cross-clamping in awake patients by increasing Fio_2. Paradoxical improvements in cerebral blood flow during carotid endarterectomy (CEA) at the time of cross-clamping and normalization of post–cross-clamp electroencephalographic abnormalities have been induced by hypocapnia. We performed this study to determine the influence of Fio_2 and end-tidal carbon dioxide ($Petco_2$) on rSO_2 in patients undergoing CEA with general anesthesia during carotid cross-clamping.
METHODS: Twenty patients were recruited. Ten underwent elective shunting. Patients received standardized general anesthesia. rSO_2 was measured using the INVOS 5100B monitor (Somanetics Corporation, Troy, MI). After carotid cross-clamping, Fio_2 and minute ventilation were sequentially adjusted: 1) Fio_2 30%, $Petco_2$ 30–35 mm Hg; 2) Fio_2 100%, $Petco_2$ 30–35 mm Hg; and 3) Fio_2 100%, $Petco_2$ 40–45 mm Hg. At each point, rSO_2 was recorded from both operative and nonoperative sides, and arterial blood gas analysis was performed.
RESULTS: Results from shunted and unshunted patients were analyzed separately. Increasing Fio_2: Administration of 100% oxygen while maintaining $Petco_2$ in the range 30–35 mm Hg in unshunted patients resulted in an 8% increase (P = 0.008) in rSO_2 on the operative side and a 6%

increase (P = 0.011) on the nonoperative side compared with an Fio_2 of 30%. In shunted patients, administration of 100% oxygen while maintaining the $Petco_2$ in the range 30–35 mm Hg resulted in a 4% increase in rSO_2 on both the operative side (P = 0.008) and the nonoperative side (P = 0.011) compared with an Fio_2 of 30%. Increasing $Petco_2$: In unshunted patients, there was a 6% (P = 0.008) increase in rSO_2 on the operative side and a 5% increase (P = 0.024) on the nonoperative side at $Petco_2$ 40–45 mm Hg compared with $Petco_2$ 30–35 mm Hg maintaining Fio_2 at 100%. In shunted patients, there was a 3% increase (P = 0.018) in rSO_2 on the operative side and a 4% increase (P = 0.007) on the nonoperative side at $Petco_2$ 40–45 mm Hg compared with $Petco_2$ 30–35 mm Hg maintaining Fio_2 at 100%.
CONCLUSION: rSO_2 is reliably improved during carotid cross-clamping by increasing Fio_2 in patients undergoing CEA with general anesthesia. Additional improvement in rSO_2 may be gained by increasing $Petco_2$.

甘氨酸轉運體－2 抑制劑 ALX1393 在大鼠急性疼痛模型中的抗傷害的作用

The Antinociceptive Effect of Intrathecal Administration of Glycine Transporter-2 Inhibitor ALX1393 in a Rat Acute Pain Model

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背景：脊髓背側角甘氨酸能神經元與脊髓水準抑制外周炎症及慢性疼痛有關。煙胺比林對碘氧基甲醚甘氨酸轉運體－2 (GlyT2)可以重攝取突觸前釋放的甘氨酸，並調節甘氨酸的神經傳遞。在這項研究中，我們研究選擇性 GlyT2 抑制劑 ALX1393 是否可以在大鼠急性疼痛模型中誘導抗傷害性刺激的效應。
方法：在雄性 Sprague-Dawley 大鼠的鞘內植入導管。分別在鞘內注射 ALX1393(劑量分別為 4, 20, or 40 μg)，通過輕彈尾巴、熱平板、壓迫爪子及福馬林試驗評估大鼠對於溫度、機械刺激及化學性傷害性刺激的反應。此外，我們還進一步研究 ALX1393 是否對運動功能有影響，並進行了迴圈試驗。
結果：對於溫度及機械刺激，ALX1393 表現出劑量依賴性抗傷害的作用。在注射 ALX1393 後 15 分鐘表現出最大的效果，顯著的作用時間持續 60 分鐘。這種抗傷害性刺激的作用可以立刻被士的甯完全逆轉。在福馬林試驗中，ALX1393 的抗疼痛作用表現為劑量依賴性，在早期及晚期均有效，並且早期的作用更顯著。相比於抗傷害性作用，40μg 的 ALX1393 對於運動功能沒有影響。
結論：這項試驗證明了 ALX1393 具有對抗急性疼痛引起的傷害性刺激作用。這些發現提示神經遞質轉運體抑制劑可以用來治療急性疼痛，選擇性 GlyT2 抑制劑是可以用來治療急性疼痛的藥物。
(陳珺珺譯 薛張綱校)

BACKGROUND: Glycinergic neurons in the spinal dorsal horn have been implicated in the inhibition of spinal pain processing in peripheral inflammation and chronic pain states. Neuronal isoform glycine transporter-2 (GlyT2) reuptakes presynaptically released glycine and regulates

the glycinergic neurotransmission. In this study, we examined whether a selective GlyT2 inhibitor, ALX1393, elicits an antinociceptive effect in a rat acute pain model.
METHODS: Male Sprague-Dawley rats were implanted with a catheter intrathecally. The effects of intrathecal administration of ALX1393 (4, 20, or 40 μg) on thermal, mechanical, and chemical nociception were evaluated by tail flick, hot plate, paw pressure, and formalin tests. Furthermore, to explore whether ALX1393 affects motor function, a rotarod test was performed.
RESULTS: ALX1393 exhibited antinociceptive effects on the thermal and mechanical stimulations in a dose-dependent manner. The maximal effect of ALX1393 was observed at 15 min after administration, and a significant effect lasted for about 60 min. These antinociceptive effects were reversed completely by strychnine injected immediately after the administration of ALX1393. In the formalin test, ALX1393 inhibited pain behaviors in a dose-dependent manner, both in the early and late phases, although the influence was greater in the late phase. In contrast to antinociceptive action, ALX1393 did not affect motor function up to 40 μg.
CONCLUSIONS: This study demonstrates the antinociceptive action of ALX1393 on acute pain. These findings suggest that the inhibitory neurotransmitter transporters are promising targets for the treatment of acute pain and that the selective inhibitor of GlyT2 could be a novel therapeutic drug.

在超聲引導下在膕窩經側路行坐骨神經阻滯：比較分別行脛神經及腓總神經阻滯及坐骨神經分叉近端阻滯起效時間

Ultrasound-Guided Sciatic Nerve Block in the Popliteal Fossa Using a Lateral Approach: Onset Time Comparing Separate Tibial and Common Peroneal Nerve Injections Versus Injecting Proximal to the Bifurcation

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背景：我們假設，相比於阻滯坐骨神經分叉近端，通過超聲引導下阻滯坐骨神經分叉遠端可以縮短完全阻斷脛神經和腓總神經的時間。
方法：76 名行足部或腳踝手術的患者進行坐骨神經阻滯，阻滯的位點在坐骨神經分叉近端或遠端。局麻藥的最大量為 28 mL 1.5%的甲派卡因加 100 μg 可樂定加 1 mL 8.4%的碳酸氫鈉，總共為 30ml。通過超聲引導下調整進針的位置。阻滯成功定義為兩神經分佈區域針刺感覺完全消失 46 分鐘。
結果：脛神經－腓總神經阻滯組起效的時間顯著快於坐骨神經組。(19.2 vs 26.1 分鐘，P = 0.006).
結論：相比於坐骨神經分叉近端阻滯，在膕窩分叉後分別行脛神經和腓總神經阻滯的起效時間較快。
(陳珺珺譯 薛張綱校)
BACKGROUND: We hypothesized that blocking the tibial and common peroneal nerves individually using ultrasound distal to sciatic bifurcation would decrease time to complete block compared with a block proximal to the bifurcation.

METHODS: Seventy-six patients undergoing foot or ankle surgery received a sciatic nerve block either proximal or distal to the point of bifurcation. A mixture of 28 mL 1.5% mepivacaine with 100 µg clonidine and 1 mL 8.4% sodium bicarbonate for a total of 30 mL was used. Ultrasound was used to guide needle adjustments to achieve circumferential spread. Block success was defined as a loss of sensation to pinprick in both nerve distributions within 46 minutes.
RESULTS: Patients in the tibial-peroneal group had significantly faster time to complete block than the sciatic group (19.2 vs 26.1 minutes; $P = 0.006$).
CONCLUSIONS: Blocking the tibial and common peroneal nerves in the popliteal fossa separately provides for a faster onset than a prebifurcation sciatic block.