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腎素血管緊張素在心血管穩態中作用更新

An Update of the Role of Renin Angiotensin in Cardiovascular Homeostasis

Farag, Ehab MD, FRCA*†,§; Maheshwari, Kamal MD*†; Morgan, Joseph MD‡; Sakr Esa, Wael Ali MD, PhD*; Doyle, D. John MD, PhD§

Anesthesia & Analgesia 2015 120 275–292

腎素血管緊張素系統（RAS）是人體內主要的血管收縮相關的系統，通過血管緊張素Ⅱ與血管緊張素 I 受體之間的相互作用來發揮生理作用（即經典的 RAS 模型）。儘管如此，隨著七肽血管緊張素 1-7 的發現和 RAS 系統的概念的變化，我們所理解的可以降低動脈血壓的 RAS 生理系統也發生了巨大的改變。在這篇綜述中，我們聚焦在最新發現的 RAS 系統的功能上，特別是這些最新發現的功能的潛在臨床意義，尤其是在治療心血管疾病最新的藥理學領域。

（呂越昌譯 薛張綱校）

The renin angiotensin system (RAS) is thought to be the body's main vasoconstrictor system, with physiological effects mediated via the interaction of angiotensin II with angiotensin I receptors (the "classic" RAS model). However, since the discovery of the heptapeptide angiotensin 1-7 and the development of the concept of the "alternate" RAS system, with its ability to reduce arterial blood pressure, our understanding of this physiologic system has changed dramatically. In this review, we focus on the newly discovered functions of the RAS, particularly the potential clinical significance of these developments, especially in the realm of new pharmacologic interventions for treating cardiovascular disease.

氯胺酮急性損傷大鼠腦內線粒體功能並增強超氧化物歧化酶活性

Acute Ketamine Impairs Mitochondrial Function and Promotes Superoxide Dismutase Activity in the Rat Brain

Venâncio, Carlos DVM, PhD*†; Félix, Luís MSc†; Almeida, Vanessa MSc*; Coutinho, João PhD‡; Antunes, Luís DVM, PhD†; Peixoto, Francisco PhD*; Summavielle, Teresa PhD§

Anesthesia & Analgesia 2015 120 320–328

背景：氯胺酮常常與改變線粒體功能和氧化應激有關。然而，氯胺酮在體內對線粒體生物能量和氧化還原狀態的作用研究甚少。越來越多的證據支持一氧化氮（NO）可能爲氯胺酮副作用的調節介質。本文我們研究了 NO 在氯胺酮麻醉中對腦線粒體功能和氧化還原狀態的調節機制。

方法：成年雄性大鼠分別給予腹腔注射單劑量氯胺酮（50 mg/kg、100 mg/kg、150mg/kg）或氯胺酮聯合 N-硝基-L-精氨酸（3mg/kg），6 小時後將動物處死。採集腦組織和血標本進行 NO 測定和線粒體分離，並運用多個變數來評價腦線粒體的功能。

結果：氯胺酮可干擾複合物 I 功能，增加耗氧量，使谷氨酸-蘋果酸基質氧化磷酸化的效率受損，降低 NADH 泛醌氧化還原酶活性。此外，在給予 50mg/kg 和 100mg/kg 的劑量後，線粒體一氧化氮合酶（mtNOS）活性和血漿 NO 水準有所增加。氯胺酮增加過氧化氫的產生並觸發超氧化物歧化酶作用。mtNOS 抑制劑可以通過 N-硝基-L-精氨酸來部分或完全阻止這些效果產生。

結論：氯胺酮急性給藥會損害線粒體複合物 I 的作用，增強 mtNOS 活性，增加過氧化氫和 NO 的產生，從而觸發超氧化物歧化酶的作用並增強抗氧化活性。本研究結果闡明在氯胺酮麻醉中 NO 的調製作用，爲臨床作用機制提供依據。

（江淩慧譯 薛張綱校）

BACKGROUND: Ketamine is often associated with altered mitochondrial function and oxidative stress. Nevertheless, limited data are still available regarding the in vivo action of ketamine in mitochondrial bioenergetics and redox state. Accumulating evidence supports a role for nitric oxide (NO) as a possible modulator of ketamine's side effects. In the present study, we investigated the role of NO modulation on ketamine anesthesia at the level of brain mitochondrial function and redox status.

METHODS: Adult male rats received a single dose of ketamine (50, 100, or 150 mg/kg IP) or a combination of ketamine and N-nitro-L-arginine (3 mg/kg IP). Animals were killed 6 hours after treatment. Brain and blood samples were collected for plasma NO determination and mitochondria isolation. Several variables of brain mitochondrial function were evaluated.

RESULTS: Ketamine interfered with complex I function, revealing increased oxygen consumption in state 4, impaired oxidative phosphorylation efficiency of glutamate-malate substrate, and decreased NADH-ubiquinone oxidoreductase activity. In addition, mitochondrial NO synthase (mtNOS) activity and NO plasma levels were increased for the 50 and 100 mg/kg doses. Ketamine administration increased hydrogen peroxide generation and triggered superoxide dismutase activity. All these effects could totally or partially be prevented by mtNOS inhibition through N-nitro-L-arginine.

CONCLUSIONS: Acute ketamine administration impaired the function of mitochondrial complex I leading to increased mtNOS activity, increased generation of hydrogen peroxide and NO, resulting in superoxide dismutase triggering, and improved antioxidant activity. The present findings clarify the role of NO modulation in ketamine anesthesia, providing new data on a relevant clinical mechanism.

使用拉曼光譜學原理的方法檢測表明在兒童腹腔鏡手術的氣腹中有空氣的存在

Gas Analysis Using Raman Spectroscopy Demonstrates the Presence of Intraperitoneal Air (Nitrogen and Oxygen) in a Cohort of Children Undergoing Pediatric Laparoscopic Surgery

Taylor, Susan P. MD, MPH*; Sato, Thomas T. MD†; Balcom, Anthony H. MD‡; Groth, Travis MD‡; Hoffman, George M. MD§

Anesthesia & Analgesia 2015 120 349–354

臨床上，在腹腔鏡手術中發生的嚴重的氣體栓塞事件雖然罕見但卻是災難性的。病例報告顯示，腹腔中除了充入腹腔的氣體外，空氣也存在。我們研究了在實驗及常規兒童手術中，應用不同的設備及充氣方法後氣腹中空氣的成分。在一種模擬的腹腔鏡手術中，我們應用拉曼光譜的方法檢測了充入模擬氣腹和從氣腹中回收的氣體中氮氣、氧氣及二氧化碳

的含量。我們隨後分析了在常規的腹腔鏡手術中應用二氧化碳充入及抽出管道中進入和回收氣體中的組成，發現其中有 10%的空氣含量存在。體外實驗中，在充入 0.2L 以下氣體的情況下，在充入氣體管道的末端是檢測不到二氧化碳的。但在 0.4L 以下時，氮氣是持續存在的，從模擬氣腹回收的氣體中大約有 40% ± 8%的氮氣含量。在臨床試驗中，預充氣體能將氮氣的含量從原來的 78% ± 0.5%降到 23% ± 15%，但不管應用什麼充氣技術，在隨後的檢測樣本中均有超過 10%的空氣存在。腹腔鏡實踐中常規允許一定量的空氣充入腹腔中。在設備中預充二氧化碳雖然能減少氣腹中空氣的含量但卻不能完全排除空氣，因此，當血管破損發生後，栓塞的氣體中含有不同量的氮氣、氧氣和二氧化碳。在兒童患者中，當氣腹的充氣空間充滿後，氮氣的含量接近未充氣系統中室內空氣中的含量。小的充氣量中含有高濃度的氮氣可造成新生兒及小兒災難性的空氣栓塞。

（王飛譯 薛張綱校）

Clinically significant gas embolism during laparoscopy is a rare but potentially catastrophic event. Case reports suggest that air, in addition to the insufflation gas, may be present. We studied the effects of equipment design and flushing techniques on the composition of gas present under experimental and routine pediatric surgical conditions. Concentrations of nitrogen (N2), oxygen (O2), and carbon dioxide (CO2) were measured by Raman spectroscopy in gas delivered to and retrieved from a mock peritoneum during simulated laparoscopy. We then analyzed the composition of insufflated and recovered gases during elective laparoscopic procedures conducted with CO2-preflushed and unflushed tubing to determine the presence of significant (10%) quantities of air. In vitro, CO2 was not detected at the distal end of insufflator tubing until after delivery of approximately 0.2 L of gas, and N2 persisted until >0.4 L was delivered, with 40% ± 8% (mean ± SD, range 33%–49%) recovered from the mock peritoneum at the termination of initial insufflation. In clinical studies, preflushing reduced the initial concentration of N2 from 78% ± 0.5% to 23% ± 15%, but >10% air was detected in all subsequent samples, regardless of insufflation technique. Laparoscopic equipment and practice routinely permit delivery of air to the insufflated cavity. Purging the equipment with CO2 reduces but does not eliminate air (N2, O2) within the peritoneal cavity during laparoscopy. Thus, when vascular injury occurs, embolized gases will contain variable quantities of N2, O2, and CO2. As the initial insufflation volume diminishes and approaches the volume of the insufflation tubing, which occurs in infants and young pediatric patients, the concentration of N2 will approximate that of room air in an unflushed system. Small insufflation volumes containing high N2 concentrations can contribute to catastrophic air emboli in neonates and small pediatric patients.

通過測定一氧化碳彌散率和肺泡表面活性物質 B 型蛋白來評估麻醉、肌松和機械通氣對肺功能的影響

The Effects of Anesthesia, Muscle Paralysis, and Ventilation on the Lung Evaluated by Lung Diffusion for Carbon Monoxide and Pulmonary Surfactant Protein B

Di Marco, Fabiano MD, PhD*; Bonacina, Daniele MD†; Vassena, Emanuele MD†; Arisi, Erik MD†; Apostolo, Anna MD‡; Banfi, Cristina PhD‡; Centanni, Stefano MD, PhD*; Agostoni, Piergiuseppe MD, PhD§ ‖ ; Fumagalli, Roberto MD, PhD†¶

Anesthesia & Analgesia 2015 120 373–380

背景：麻醉患者的肺泡-動脈氧分壓差通常會增加。本研究旨在評估麻醉，肌松以及短期機械通氣對肺功能的影響。

方法：我們選取 45 名接受非胸部手術並且無肺部疾病的病人，測定他們的一氧化碳彌散率（DLCO），包括肺毛細血管血容量（Vc),肺泡-毛細血管屏障的導電性以及肺泡表面活性物質 B 型蛋白（肺泡損傷的標誌）

結果：麻醉、肌松以及機械通氣都會導致肺泡氣體交換受損，伴隨著 DM 和 Vc 的下降，麻醉誘導後 DLCO 值也立即下降。然而，DM 的下降是由於肺容量的下降，Vc 的變化並非如此，而是由於 Vc/肺泡容積比值的顯著下降。儘管 DLCO 和它的各組成成分在麻醉誘導後立即下降，但在接下來的 1 到 3 小時內它們的數值並沒有進一步下降。而表面活性物質 B 型蛋白在麻醉誘導後並沒有立即改變，但在誘導後 1 小時後有所增加，而誘導 3 小時後則進一步增加。肺泡損傷的程度與肺灌注和肺順應性的下降有關（即潮氣量與呼末肺容量之比）。

結論：短時間麻醉及控制通氣將會帶來：（1）與肺順應性和肺灌注下降相關的肺泡損傷（2）主要與肺容量下降相關的氣體交換障礙，同時也與肺灌注下降有關。

（潘豔譯 薛張綱校）

BACKGROUND: An increased alveolar-arterial oxygen tension difference is frequent in anesthetized patients. In this study, we evaluated the effect on the lung of anesthesia, muscle paralysis, and a brief course of mechanical ventilation.

METHODS: Lung diffusion for carbon monoxide (DLCO), including pulmonary capillary blood volume (Vc) and conductance of the alveolar-capillary membrane (DM), and pulmonary surfactant protein type B (a marker of alveolar damage) were measured in 45 patients without pulmonary disease undergoing extrathoracic surgery.

RESULTS: Anesthesia, muscle paralysis, and mechanical ventilation led to impairment of gas exchange, with a reduction of DLCO values immediately after anesthetic induction due to a concomitant reduction of both DM and Vc. While changes in DM were due to the reduction of lung volume, changes in Vc were not limited to volume loss, since the Vc/alveolar volume ratio decreased significantly. Although DLCO and its components decreased immediately after induction, none of the values decreased further at 1 and 3 hours. Surfactant protein type B, however, was unchanged immediately after anesthesia but increased at 1 hour after induction and further increased after 3 hours of anesthesia. The level of alveolar damage correlated with the reduction of lung perfusion and lung dynamic strain (i.e., ratio between tidal volume and end-expiratory lung volume).

CONCLUSIONS: A brief course of anesthesia and controlled ventilation leads to: (1) alveolar damage, which is correlated with lung strain and perfusion, and (2) impaired gas exchange mainly due to volume loss but also to reduced aerated lung perfusion.

新生兒心肺轉流術後過量出血以及預後

Excessive Postoperative Bleeding and Outcomes in Neonates Undergoing Cardiopulmonary Bypass

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背景：進行心臟手術的新生兒特別易於發生與心肺轉流（CPB）相關的凝血功能異常，CPB 後出血的風險增高。不成熟的凝血系統，CPB 預充時嚴重的血液稀釋，CPB 時長時間的低體溫，過多的縫合增加了新生兒 CPB 後出血的風險以及術後嚴重併發症的發生。

方法：我們回顧性分析了在 2010 年 1 月至 2011 年 12 月 31 日之間在 CPB 下行複雜先天性心臟病手術的 169 名新生兒的病史資料。收集並分析了圍術期患者的資料，通過測定術後 24h 的胸腔引流量（CTO），CPB 術後輸血的需要量以及嚴重的術後併發症，包括腎功能不全、血液透析、血栓、體外膜肺以及住院死亡率，從而重點分析了 CPB 後的出血量。我們使用 Spearman 相關性分析來確定多個圍術期的變數和 24hCTO 及術後血製品需

要量之間的關係。此外，我們使用 logistic 回歸分析來確定過量出血（定義爲 24h CTO 大於第 75 百分位數）及術後重大併發症之間的關係。

結果：24h CTO 和術後輸血需要量與先天性心臟病手術風險評分（RACHS-1），CPB 時間及低體溫之間顯著相關。Logistic 回歸分析發現 CPB 後過量出血使術後血透（相對危險度[RR] 12.0；可信區間，1.50–54.69；P=0.02）以及 ECMO（RR 9.95；可信區間，3.07–28.47；P=0.0008）的獨立預測因素。RACHS-1 評分是住院死亡率的有意義的預測因素（P=0.03）。

結論：新生兒 CPB 術後過量出血與術後不良事件的增加獨立相關，尤其是術後血透以及 ECMO 支持。我們在新生兒中研究的結果與近期一致：在兒童 CPB 後增加的輸血需要量與術後重大併發症的發生獨立相關。我們的研究結果可以幫助臨床醫生預測新生兒心肺轉流術後潛在的併發症的發生以及分配資源以處理這些不良事件。

（杜芳譯 薛張綱校）

BACKGROUND: Neonates undergoing cardiac surgery are especially prone to the hemostatic alterations of cardiopulmonary bypass (CPB) and are at high risk for post-CPB bleeding. An immature coagulation system, significant hemodilution from the CPB prime, long CPB times at low temperatures, and extensive suture lines increase neonates' susceptibility to bleeding after CPB. In this study, we examined the relationship between excessive bleeding in neonates after CPB and major postoperative adverse events.

METHODS: We retrospectively reviewed the medical records of 169 neonates who underwent complex congenital heart surgery with CPB between January 1, 2010, and December 31, 2011. Perioperative data were collected and analyzed with specific focus on post-CPB bleeding as measured by 24-hour postoperative chest tube output (CTO), post-CPB transfusion requirements, and major postoperative adverse events, including renal dysfunction, dialysis, thrombosis, extracorporeal membrane oxygenation (ECMO), and in-hospital mortality. We used Spearman correlation to determine correlations between multiple perioperative variables and 24-hour CTO and postoperative blood product requirements. Also, we used logistic regression analysis to determine the association between excessive bleeding (defined as 24-hour CTO >75th percentile) and major postoperative adverse events.

RESULTS: Significant correlations were found between 24-hour CTO and postoperative blood product transfusion with weight, Risk Adjustment for Congenital Heart Surgery (RACHS-1) score, CPB time, and lowest temperature. Logistic regression found that excessive bleeding after CPB was an independent predictor of postoperative dialysis (relative risk [RR] 12.0; confidence interval, 1.50–54.69; P = 0.02) and ECMO (RR 9.95; confidence interval, 3.07–28.47; P = 0.0008). RACHS-1 score was a significant predictor of in-hospital mortality (P = 0.03).

CONCLUSIONS: Excessive postoperative bleeding in neonates after CPB is independently associated with increased adverse events, specifically the need for postoperative dialysis and ECMO support. Our findings in neonates are congruent with other recent research that also has found increasing transfusion requirements after pediatric CPB to be independently associated with an increase in major postoperative adverse events. Our results may aid clinicians in anticipating potential adverse events after neonatal bypass and in allocating the resources necessary to manage these events.

超聲用於評估兒科患者喉罩放置位置的一項觀察性研究

An Ultrasound Evaluation of Laryngeal Mask Airway Position in Pediatric Patients: An Observational Study

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背景：對於兒科患者，喉罩（LMA）經常在喉咽處移位元而需要重新定位。當 LMA 的頂端放置在食管入口時，杓狀軟骨向前側移位。當 LMA 時旋轉或傾斜時，杓狀軟骨的腹側運動可能導致杓狀軟骨的不對稱，這一現象可被超聲檢測到。在本研究中，我們試圖用超評估兒科患者 LMA 移位的發生率。主要研究終點是使用超聲和支氣管鏡檢查（FOB）評估喉罩移位的發生率。次要終點是測定使用超聲和支氣管鏡用於發現喉罩移位間的關係，並且歸納出超聲診斷 LMA 移位元的影像表現。

方法：在這項觀察研究包括有 100 名兒童。麻醉誘導後，我們分別在喉罩置入前後將超聲探頭置於患者頸前得到聲門處圖像用以評估。纖維支氣管鏡用於評估喉罩位置(纖支鏡喉罩分級及喉罩旋轉分級)。放置好喉罩後，可以通過超聲就杓狀軟骨的對稱性進行評估。基於聲門中線來評估杓狀軟骨的不對稱性，並且相對的杓狀軟骨被分爲 0 到 3 級（超聲杓狀軟骨分級)。我們對超聲杓狀軟骨分級、纖支鏡喉罩分級及喉罩旋轉分級之間相互關係進行分析。

結果：不對稱的杓狀軟骨的發生率爲 50%（95%可信區間(CI)，40% - 60%）。對於纖支鏡，喉罩移位的發生率爲 78%（95%可信區間，69% - 86%），喉罩旋轉分級爲 43%（95%可信區間，33% - 53%）。纖支鏡檢查喉罩移位的發生率較高（P < 0.0001），但旋轉的發生率是相似的（P = 0.395）。超聲杓狀軟骨分級與纖支鏡喉罩分級無相關性 （P = 0.611），但與喉罩旋轉分級呈顯著相關（P < 0.0001，95%可信區間，60% - 83%）。用於檢測喉罩旋轉,超聲靈敏度爲 93%（95%可信區間，81% - 98%），特異度爲 82%（95%可信區間，70% - 91%）。陽性和陰性預測值分別爲 80%（95%可信區間，66% - -90%）和 94%（95%可信區間，83% - 99%），準確度爲 87%（95%可信區間，79% - 93%）。

結論：雖然超聲無法檢測喉罩放置的最適宜深度，但我們可以認爲超聲是一種精確檢測喉罩旋轉移位的工具。

（黃文惠譯 薛張綱校）

BACKGROUND: In children, the laryngeal mask airway (LMA) is frequently displaced within the hypopharynx, resulting in repositioning of the device. When the tip of the LMA is placed in the esophageal inlet, the arytenoids are moved ventrally. When the LMA is rotated or deviated, the ventral movement of the arytenoids may result in asymmetric elevation of an arytenoid cartilage, which can be detected with ultrasound (US). In this study, we sought to estimate the incidence of LMA malposition detected with US in pediatric patients. The primary end point was to compare the incidence of LMA malposition between US and fiber optic bronchoscopy (FOB). The secondary end points were to find the interrelationship between US-detected and FOB-detected malposition of the LMA and to locate the diagnostic performance of US in detecting LMA malposition.

METHODS: In this observational study, 100 consecutive children were included. After anesthetic induction, US evaluation was performed before and after LMA insertion to obtain the glottic image on the anterior neck. FOB was performed to assess LMA position (FOB LMA grade and LMA rotation grade). With a post-LMA US image, the symmetry of the arytenoid cartilages was evaluated. Asymmetrical elevation of an arytenoid cartilage in reference to the glottic midline and the opposite arytenoid cartilage was graded as 0 to 3 (US arytenoid grade). The interrelationships between US arytenoid grade and FOB LMA grade or LMA rotation grade were assessed.

RESULTS: The incidence of asymmetrical elevation of an arytenoid was 50% (95% confidence interval [CI], 40%–60%). On FOB, the incidence of LMA malposition was 78% (95% CI, 69%–86%), and that of LMA rotation was 43% (95% CI, 33%–53%). The incidence of LMA malposition was higher with FOB (P< 0.0001), but the incidence of rotation was similar (P = 0.395). US arytenoid grade did not correlate with FOB LMA grade (P = 0.611) but showed a significant correlation with LMA rotation grade (P< 0.0001; 95% CI, 60%–83%). To detect a rotated LMA, US had a sensitivity of 93% (95% CI, 81%–98%) and a specificity of 82% (95%

CI, 70%–91%). The positive and negative predictive values were 80% (95% CI, 66%–90%) and 94% (95% CI, 83%–99%), respectively. The accuracy was 87% (95% CI, 79%–93%).

CONCLUSIONS: Although US could not detect the suboptimal depth of an LMA, US has promise of being an accurate tool in detecting a rotated LMA.

刺激性揮發麻醉劑通過 TRPA1 和 TRPV1 通道導致離體小鼠氣管神經性炎症

Irritant Volatile Anesthetics Induce Neurogenic Inflammation Through TRPA1 and TRPV1 Channels in the Isolated Mouse Trachea

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背景：揮發性全身麻醉劑對氣管神經末梢的刺激作用導致氣道痙攣的分子機制還不甚清楚。神經肽的釋放和神經性炎症有明確作用。

方法：我們分析液體澆注的離體小鼠氣管釋放降鈣素基因相關肽（CGRP）的基礎釋放量及受刺激後的釋放量，用於指示感覺神經元的啓動，其中使用刺激性揮發麻醉劑（地氟烷和異氟烷）及非刺激性揮發麻醉劑（七氟烷）作爲刺激物。在 38 攝氏度條件下，在不同的氣體濃度(0.5 倍, 1 倍, 或 2 倍最小肺泡濃度 [MAC])和不同的氧濃度中給予氣管刺激。用辣椒素受體 TRPV1 和化學受體 TRPA1 的無變異的以及兩基因均被敲除的小鼠作爲組織供體。

結果：地氟烷以及異氟烷導致濃度相關的氣管 CGRP 的釋放，兩者均在 1MAC（人）時達到飽和，也就是分別爲 6% 和 1.25%的濃度。 地氟烷組，氧氣濃度（25%或者 94%）的差異對結果沒有影響。七氟烷 1MAC 濃度不會導致氣管 CGRP 的釋放。TRPV1 小鼠的地氟烷反應降低 75%，而 TRPA1 和雙敲除變異體完全沒有反應。.

結論：我們的結果證實了臨床經驗所顯示的：在相同的麻醉氣體濃度下，地氟烷較異氟烷更具刺激性，而七氟烷不會刺激氣管支氣管感覺神經釋放神經肽或者導致神經性炎症。就兩個刺激受體通道而言，TRPA1 較 TRPV1 更多地與介導不良反應有關，甚至可以延伸導致全身促炎反應後遺症。

（蓋曉冬譯 薛張綱校）

BACKGROUND: Irritating effects of volatile general anesthetics on tracheal nerve endings and resulting spastic reflexes in the airways are not completely understood with respect to molecular mechanisms. Neuropeptide release and neurogenic inflammation play an established role.

METHODS: The basal and stimulated calcitonin gene-related peptide (CGRP) release from the isolated superfused mouse trachea was analyzed as an index of sensory neuron activation, applying irritant (desflurane and isoflurane) and nonirritant (sevoflurane) volatile anesthetics as stimuli. Various gas concentrations (0.5-, 1-, or 2-fold minimum alveolar concentration [MAC]) and different O2 atmospheres were used for tracheal stimulation at 38°C. Null mutants of the capsaicin receptor TRPV1 and of the chemoreceptor TRPA1, as well as double knockout mice, were used as tissue donors.

RESULTS: Desflurane and, less so, isoflurane caused a concentration-dependent tracheal CGRP release, both saturating at 1 MAC (human), that is, 6% and 1.25%, respectively. With desflurane, the O2 concentration (25% or 94%) did not make a difference. Sevoflurane 1 MAC did not activate tracheal CGRP release. TRPV1 mice showed 75% reduced desflurane responses, and TRPA1 and double-null mutants showed no responses at all.

CONCLUSIONS: Our results confirm the clinical experience that desflurane is more irritating than isoflurane at equal anesthetic gas concentration, whereas sevoflurane does not activate

tracheobronchial sensory nerves to release neuropeptides and induce neurogenic inflammation. Both irritant receptor channels, TRPA1 more than TRPV1, are involved in mediating the adverse effects that may even extend to systemic proinflammatory sequelae

異氟醚通過保護線粒體呼吸與超分子構造而避免心肌缺血性損傷

Isoflurane Protects the Myocardium Against Ischemic Injury via the Preservation of Mitochondrial Respiration and Its Supramolecular Organization

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背景：異氟醚已被證明可限制心肌缺血性損傷。此效應推測部分通過影響線粒體而介導。本研究考察這一假設，即異氟醚維持線粒體呼吸鏈功能，從而限制心肌缺血損傷期間的線粒體損傷和線粒體膜解體。

方法：小鼠（9-12 周齡）暴露於（1.0MAC）異氟醚 36 h 後給予 30 min 的冠狀動脈血管鉗夾，隨後進行 24 h 再灌注。再灌注每 4h 分離心肌線粒體。2、3、5-氯化三苯基四氮唑染色用於確定心肌梗塞大小。應用藍綠溫和聚丙烯醯胺凝膠電泳及特定的生化分析，研究線粒體呼吸鏈功能。通過丙二醛形成量化線粒體脂質過氧化反應；通過鈣離子誘導腫脹評估線粒體膜的完整性。通過液相色譜質譜法和質譜法確認蛋白。

結果：共 31 只小鼠納入研究。暴露於異氟醚的小鼠心肌梗死面積更少（P=0.0011，與缺血/再灌注[I/ R]組相比），且線粒體呼吸複合物 III 損傷更小（P =0.0008，與 I / R 組相比）。異氟醚穩定了由複雜 III / IV 低聚物組成的線粒體超複合體（P =0.0086，與 I / R 組相比）。丙二醛形成減少（P =0.0019，與 I / R 組相比）以及對鈣離子誘導腫脹敏感性降低（P=0.0010，與 I / R 組相比）進一步證實了經異氟醚處理後線粒體損傷可減輕。

結論：本研究結果支持異氟醚維持線粒體呼吸鏈在體功能使心臟免受缺血性損傷的假說。這些效應可能部分由於對線粒體超分子組織和最小化氧化損傷的保護，避免了線粒體膜完整性缺失。

（柳韶華 譯 陳傑 校）

BACKGROUND: Isoflurane has been demonstrated to limit myocardial ischemic injury. This effect is hypothesized to be mediated in part via effects on mitochondria. We investigated the hypothesis that isoflurane maintains mitochondrial respiratory chain functionality, in turn limiting mitochondrial damage and mitochondrial membrane disintegration during myocardial ischemic injury.

METHODS: Mice (9–12 weeks of age) received isoflurane (1.0 minimum alveolar concentration) 36 hours before a 30-minute coronary artery occlusion that was followed by 24 hours of reperfusion. Cardiac mitochondria were isolated at a time point corresponding to 4 hours of reperfusion. 2,3,5-Triphenyltetrazoliumchloride staining was used to determine myocardial infarct size. Mitochondrial respiratory chain functionality was investigated using blue native polyacrylamide gel electrophoresis, as well as specific biochemical assays. Mitochondrial lipid peroxidation was quantified via the formation of malondialdehyde; mitochondrial membrane integrity was assessed by Ca2+-induced swelling. Protein identification was achieved via liquid chromatography mass spectrometry/mass spectrometry.

RESULTS: Thirty-one mice were studied. Mice receiving isoflurane displayed a reduced myocardial infarct size (P = 0.0011 versus ischemia/reperfusion [I/R]), accompanied by a preserved activity of respiratory complex III (P = 0.0008 versus I/R). Isoflurane stabilized mitochondrial supercomplexes consisting of oligomers from complex III/IV (P = 0.0086 versus I/R). Alleviation of mitochondrial damage after isoflurane treatment was further demonstrated as

decreased malondialdehyde formation (P = 0.0019 versus I/R) as well as a diminished susceptibility to Ca2+-induced swelling (P = 0.0010 versus I/R).

CONCLUSIONS: Our findings support the hypothesis that isoflurane protects the heart from ischemic injury by maintaining the in vivo functionality of the mitochondrial respiratory chain. These effects may result in part from the preservation of mitochondrial supramolecular organization and minimized oxidative damage, circumventing the loss of mitochondrial membrane integrity.

2-去氧-D-葡萄糖增強小鼠麻醉作用

2-Deoxy-D-Glucose Enhances Anesthetic Effects in Mice

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背景：揮發性藥物的全身麻醉機制仍知之甚少。有研究認爲能量水準上線粒體功能障礙和降低與全身麻醉效應相關。 2-去氧-D-葡萄糖（2-DG），一種葡萄糖的類似物，可以抑制己糖激酶和減少細胞三磷酸腺苷（ATP）水準。 3-硝基丙酸是另外一種可消耗 ATP 水準的化合物。與此相反，艾地苯醌和左旋肉堿可挽救能量不足。因此本研究試圖確定 2-DG 和/或 3-硝基丙酸是否可以增強異氟醚的麻醉作用，而艾地苯醌和左旋肉堿是否能夠逆轉 2-DG 的作用。

方法： C57BL / 6J（8 月齡）小鼠暴露於不同濃度的異氟醚，同時給予或不給予 2-DG、3-硝基丙酸、艾地苯醌和左旋肉堿處理。觀察實驗小鼠異氟烷誘導的翻正反射（LORR）消失。處理後評估 H4 人類神經膠質瘤細胞的 ATP 水準。最後，通過 31P-磁共振光譜來確定異氟醚對小鼠大腦 ATP 水準的影響。

結果：2-DG 增強異氟醚引起的 LORR（P =0.002，N =15）。3-硝基丙酸可以增強異氟烷的麻醉作用（P = 0.005，N =15）。艾地苯醌（艾地苯醌+生理鹽水與艾地苯醌+2-DG 比較：P =0.165，N= 15），而非左旋肉堿（左旋肉毒堿+生理鹽水與左旋肉毒堿+2-DG：P <0.0001，N= 15）可以抑制 2-DG 對小鼠異氟烷誘導的 LORR 的增強作用，同時證實 2-DG 無法增強艾地苯醌預處理小鼠異氟烷誘導的 LORR 作用。艾地苯醌（艾地苯醌+生理鹽水與艾地苯醌+2-DG 相比：P =0.177，N = 6），而非左旋肉堿（左旋肉毒堿+生理鹽水與左旋肉毒堿+2-DG 相比：P = 0.029，N= 6）可以緩和 2-DG 減少細胞內 ATP 水準的作用，同時證實 2-DG 無法減少艾地苯醌預處理細胞的 ATP 水準。最後，異氟醚可以減少培養細胞和小鼠大腦細胞中 ATP 水準（β-ATP：P =0.003，N =10; β-ATP/磷酸：P =0.006，N =10; β-ATP/無機磷酸鹽：P=0.001 ，N= 10）。

結論：初步研究中得出的這些結果建立了一個體系並產生假設，即 2-DG 通過降低能耗水準增強麻醉作用。這些結果促進對麻醉機制的進一步探討。

（池曉穎 譯 陳傑 校）

BACKGROUND: The mechanisms of general anesthesia by volatile drugs remain largely unknown. Mitochondrial dysfunction and reduction in energy levels have been suggested to be associated with general anesthesia status. 2-Deoxy-D-glucose (2-DG), an analog of glucose, inhibits hexokinase and reduces cellular levels of adenosine triphosphate (ATP). 3-Nitropropionic acid is another compound which can deplete ATP levels. In contrast, idebenone and L-carnitine could rescue deficits of energy. We therefore sought to determine whether 2-DG and/or 3-nitropropionic acid can enhance the anesthetic effects of isoflurane, and whether idebenone and L-carnitine can reverse the actions of 2-DG.

METHODS: C57BL/6J mice (8 months old) received different concentrations of isoflurane with and without the treatments of 2-DG, 3-nitropropionic acid, idebenone, and L-carnitine.

Isoflurane-induced loss of righting reflex (LORR) was determined in the mice. ATP levels in H4 human neuroglioma cells were assessed after these treatments. Finally, 31P-magnetic resonance spectroscopy was used to determine the effects of isoflurane on brain ATP levels in the mice.

RESULTS:2-DG enhanced isoflurane-induced LORR (P = 0.002, N = 15). 3-Nitropropionic acid also enhanced the anesthetic effects of isoflurane (P = 0.005, N = 15). Idebenone (idebenone + saline versus idebenone + 2-DG: P = 0.165, N = 15), but not L-carnitine (L-carnitine + saline versus L-carnitine + 2-DG: P < 0.0001, N = 15), inhibited the effects of 2-DG on enhancing isoflurane-induced LORR in the mice, as evidenced by 2-DG not enhancing isoflurane-induced LORR in the mice pretreated with idebenone. Idebenone (idebenone + saline versus idebenone + 2-DG: P = 0.177, N = 6), but not L-carnitine (L-carnitine + saline versus L-carnitine + 2-DG: P = 0.029, N = 6), also mitigated the effects of 2-DG on reducing ATP levels in cells, as evidenced by 2-DG not decreasing ATP levels in the cells pretreated with idebenone. Finally, isoflurane decreased ATP levels in both cultured cells and mouse brains (β-ATP: P = 0.003, N = 10; β-ATP/phosphocreatine: P = 0.006, N = 10; β-ATP/inorganic phosphate: P = 0.001, N = 10).

CONCLUSIONS: These results from our pilot studies have established a system and generated a hypothesis that 2-DG enhances anesthetic effects via reducing energy levels. These findings should promote further studies to investigate anesthesia mechanisms.

氧供的有效性和二氧化碳波形的可靠性：4 種鼻導管的交叉比較

The Effectiveness of Oxygen Delivery and Reliability of Carbon Dioxide Waveforms: A Crossover Comparison of 4 Nasal Cannulae

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背景：有效氧輸送和準確的呼氣末 CO2（ETCO2）採樣是對呼吸功能受損患者鼻導管（NCs）吸氧基本要求。本實驗研究了 4 種 NC 設計：分叉鼻塞（NPs）供氧同時在兩側均有 CO2 感測器（Hudson）,獨立的 O2/CO2 NPs(Salter),CO2 感測器位於 NPs 上，在 NPs 外通過多噴口（Oridion）和雙噴口（Medline）進行氧供。本研究假設 NCs 之間設計的差異會影響氧供和 ETCO2 檢測。

方法：45 名 18 至 35 歲的健康志願者參與了這項無限制、隨機、區組設計試驗，每個受試者在一個試驗階段按照他們的意願控制 4 種 NCs 的 4 個交叉研究週期。監測包括心電圖,由 Hauge 氣道的後咽 O2 抽樣（Sharn 麻醉產品，Tampa，弗羅裡達州）和 NC ETCO2。在 11 名志願者中，橈動脈血液採樣用於檢測 O2 和 CO2 分壓(PaO2 和 PaCO2)。在吸入空氣和 2、4、6Lpm 的新鮮氧氣（FGFs）期間，依照隨機化原則，提供每種 NC，收集每 2 分鐘資料（ETCO2，咽後 O2，PaO2 和 PaCO2）。統計分析採用 SAS 分析軟體，9.3 版本及 JMP 統計軟體，11 版本(SAS Institute Inc., Cary, NC)，P<0.05 爲統計學差異。

結果：血氣分析表明每次實驗期間 PaCO2 穩態值與基線值無差異。不同 NC 間，在基線和 2 Lpm O2 時 PaO2 沒有差異。吸氧 4 Lpm 時，獨立 NPs 和分叉 NCs 的 PaO2 顯著高於多噴口 NC。吸氧 2、4、6 Lpm 時，獨立 NPs 的咽後 O2 顯著高於應用多噴口和雙噴口 NCs。吸氧 2Lpm 時，分叉 NPs 的咽後 O2 顯著高於應用多噴口 NC；並在吸氧 4、6 Lpm 時顯著高於噴口 NC。分叉 NPs 的 ETCO2 顯著低於其他 3 種 NCs，這與在較高 FGF 時難以追蹤 CO2 相一致。

結論：NCs 爲肺功能受損患者提供了額外的 O2 吸入。ETCO2 準確測定有利於評估呼吸頻率並確定在肺換氣不足時是否發生 CO2 瀦留。這些發現表明應用分叉鼻塞（NPs）的鼻導管（NC）在氧供時是最有效的，並且在較高 FGFs 時提供最可靠且穩定的 CO2 波形。

（王筱婧 譯 陳傑 校）

BACKGROUND: Effective O2 delivery and accurate end-tidal CO2 (ETCO2) sampling are essential features of nasal cannulae (NCs) in patients with compromised respiratory status. We studied 4 NC designs: bifurcated nasal prongs (NPs) with O2 delivery and CO2 sensing in both NPs (Hudson), separate O2/CO2 NPs (Salter), and CO2 sensing in NPs with cloud O2 delivery outside the NPs via multi vents (Oridion) and dual vents (Medline). We hypothesized that design differences between NCs would influence O2 delivery and ETCO2 detection.

METHODS: Forty-five healthy volunteers, 18 to 35 years, participated in an unrestricted, randomized block design, each subject serving as their own control in a 4-period crossover study design of 4 NCs during one session. Monitoring included electrocardiogram, posterior pharynx O2 sampling from a Hauge Airway (Sharn Anesthesia Products, Tampa, FL), and NC ETCO2. In 11 volunteers, radial artery blood was sampled from a catheter for partial pressures of O2 and carbon dioxide (PaO2 and PaCO2) determination. Per randomization, each NC was positioned, and data were collected over 2 minutes (ETCO2, pharyngeal O2, PaO2, and PaCO2) during room air and during O2 fresh gas flows (FGFs) of 2, 4, and 6 Lpm. Statistical analyses were performed with SAS Analytics Pro, Version 9.3, and JMP Statistical Software, Version 11 (SAS Institute Inc., Cary, NC), significance at $P < 0.05$.

RESULTS: Blood gas analyses indicated PaCO2 during steady state at each experimental time period remained unchanged from physiologic baseline. PaO2 did not differ between NC devices at baseline or 2 Lpm O2. The PaO2 at 4 Lpm from the separate NPs and bifurcated NCs was significantly higher than the multi-vented NC. Pharyngeal O2 with the NC with separate NPs was significantly higher than multivented and dual-vented cloud delivery NCs at 2, 4, and 6 Lpm FGF. Pharyngeal O2 with the NC with bifurcated NPs was significantly higher than the multi-vented NC at 2 Lpm, and higher than cloud delivery NCs at 4 and 6 Lpm FGF. ETCO2 was significantly lower with the NC with bifurcated NPs compared to the other 3 NCs, consistent with errant CO2 tracings at higher FGF.

CONCLUSIONS: NCs provide supplemental inspired O2 concentrations for patients with impaired pulmonary function. Accurate measures of ETCO2 are helpful in assessing respiratory rate and determining whether CO2 retention is occurring from hypoventilation. These findings suggest the NC with separate NPs was the most effective in delivering O2 and the most consistent at providing reliable CO2 waveforms at higher FGFs.

在國家麻醉臨床預後登記中觀察到的圍術期心臟驟停發生率和危險因素

The Incidence and Risk Factors for Perioperative Cardiac Arrest Observed in the National Anesthesia Clinical Outcomes Registry

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背景：在手術室和復蘇室裡，對於經嚴密監測的手術患者，心臟驟停是一個罕見但重要的事件。近期發表文獻報導了後續住院期間而非圍術期手術患者心臟驟停的發生率。本文假設在此期間（圍術期和復蘇期）心臟驟停發生率較報導的住院期間發生率更低。

方法：抽取 2010 年至 2013 年國家麻醉臨床預後登記的所有心臟驟停和急性圍手術期死亡的資料並且分析麻醉相關危險因素。比較其與發表的術後住院期間心臟驟停發生率差異性。

結果：總體而言，心臟驟停的風險爲 5.6／10000 例，低於先前報導的手術患者住院期間總體死亡率，且心臟停跳相關死亡率爲 58.4%。心臟驟停的發生率隨著年齡和 ASA 分級增加而增加。男性心臟驟停率發生率和死亡率更高。

結論：國家麻醉臨床預後登記是檢驗圍手術期和麻醉相關預後的一種新興資源。心臟驟停在手術期間比在住院期間發生率更低，大部分發生於 ASA III–V 級的患者。難以解釋男性死亡風險較高的現象，故需要進一步研究。

（徐歡 譯 陳傑 校）

BACKGROUND: Cardiac arrest is a rare but important event in the operating room and postanesthesia care unit, when surgical patients are most intensively monitored. Several recent publications have reported the rate of cardiac arrest in surgical patients during the subsequent hospital stay but have not uniquely identified the immediate perioperative period. We hypothesized that cardiac arrest during this time (intraprocedure and postanesthesia care) would occur at a lower frequency than that described for inpatient hospital care in the available literature.

METHODS: We extracted data from all cardiac arrests and immediate perioperative deaths reported to the National Anesthesia Clinical Outcomes Registry for the period from 2010 to 2013 and analyzed for anesthesia-related risk factors. We compared these data to published rates of in-hospital cardiac arrest after surgery.

RESULTS: Overall, the risk of cardiac arrest was 5.6 per 10,000 cases, which is less than in previous reports of in-hospital arrests in surgical patients overall, with an associated mortality from the arrest of 58.4%. The rate of cardiac arrest increased with age and ASA physical status. The rate of cardiac arrest was significantly higher for males, as was the mortality.

CONCLUSIONS: The National Anesthesia Clinical Outcomes Registry is an emerging resource for examination of perioperative and anesthesia-related outcomes. Cardiac arrest is less frequent in the periprocedural setting than later in the hospital course, with most arrests predictably occurring in patients with ASA physical status III–V. The finding of increased risk of mortality in male patients cannot be readily explained and should prompt future research attention.

晶體液和膠體液：通過系統回顧和 meta 回歸分析探索兩者在液體需求中的差異

Crystalloids Versus Colloids: Exploring Differences in Fluid Requirements by Systematic Review and Meta-Regression

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背景：液體正平衡與較差的預後相關。關於不同類型不同量的液體如何達到相同終點的臨床意義重大。大分子比小分子在血管內持續更長時間,與晶體相比，較少的膠體可能達到相同的血流動力學終點。然而，最近的臨床資料挑戰了這個生理概念，研究者報告了在不同人群低於預期的晶體/膠體比率。

方法: 截止至 2013 年 12 月 18 日,在 MEDLINE、EMBASE 和 CENTRAL 上檢索所有關於在各種患者中比較膠體和晶體的研究來進行此項系統檢索。計算每個研究的晶體/膠體比值。對所有研究進行了描述性分析,並對那些有輸注液體量完整資料的研究（均值和標準差）進行 meta 分析。根據研究和人口學特徵進行分組。然後通過 meta 回歸分析評估晶體/膠體比值差異的一些可能原因。

結果:在 976 項研究中,48 項在最後分析中被保留;24 項研究有足夠資料進行 meta 分析。在所有這些包括 meta 分析的研究中，晶體/膠體比例爲 1.5(95%置信區間,1.36 - -1.65)，存在顯著的非均質性(I2 = 94%)。從 meta 回歸分析,近十年的出版文獻(P = 0.001)和白蛋白亞組研究(P = 0.001)的濃度（張力）與晶體/膠體比例管理相關。meta 分析所有出版物的異質性下降是最小的,且納入的近十年出版物顯示下降幅度最大(R2 = 12%)。

結論:與膠體相比，要達到同樣的目標需要更大的晶體容量,比值約爲 1.5(1.36 - 1.65),但研究中有明顯異質性。晶體/膠體比率近年來似乎有所下降,比值的差異與白蛋白的濃度相關。然而研究的高異質性的主要原因尚不清楚。

（林雨軒 譯 陳傑 校）

BACKGROUND: Positive fluid balance has been associated with worse outcomes, and knowledge of differences in the amounts of different types of fluid needed to achieve the same end points may have important clinical implications. Large molecules persist longer in the blood vessels than smaller molecules, such that less IV colloid may be needed to achieve similar hemodynamic end points compared with crystalloid. Recent clinical data have, however, challenged this physiological concept, with investigators reporting lower-than-expected crystalloid/colloid ratios in various populations.

METHODS: We performed a systematic search in MEDLINE, EMBASE, and CENTRAL up to December 18, 2013, to retrieve all studies comparing (any) crystalloid with (any) colloid in all types of patients. The crystalloid/colloid ratio was calculated for each study. Descriptive analysis was performed for all studies, and a meta-analysis was performed in those studies reporting full data (in terms of means and standard deviations) of infused fluid volumes. Studies were grouped according to study and population characteristics. A meta-regression analysis was then performed to evaluate some of the possible reasons for differences in crystalloid/colloid ratios across studies.

RESULTS: From 976 studies, 48 were retained for the final analysis; 24 of the studies had sufficient data for meta-analysis. The crystalloid/colloid ratio across all the studies included in the meta-analysis was 1.5 (95% confidence interval, 1.36–1.65) with marked heterogeneity among studies (I2 = 94%). From the meta-regression analysis, decade of publication across all publications (P = 0.001) and concentration (tonicity) in the subgroup of albumin studies (P = 0.001) were associated with the administered crystalloid/colloid ratio. The reduction in heterogeneity among studies for all publications in the meta-regression was minimal, with the maximal decrease obtained when decade of publication was considered (R2 = 12%).

CONCLUSIONS: Greater fluid volumes are required to meet the same targets with crystalloids than with colloids, with an estimated ratio of 1.5 (1.36–1.65), but there is marked heterogeneity among studies. The crystalloid/colloid ratio seems to have decreased over the years, and differences in ratios are correlated with the concentration of albumin solutions; however, the main reasons behind the high heterogeneity among studies remain unclear.

靶向治療對兒科肺動脈高壓患者圍術期發病率和死亡率影響

The Impact of Targeted Therapies for Pulmonary Hypertension on Pediatric Intraoperative Morbidity or Mortality

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背景：肺動脈高壓（PHT）是麻醉期間主要不良事件的重要風險因素，據報導其發生率爲 5%至 7%，並可繼發急性肺動脈高壓危象或右心室缺血。PHT 新型治療方法已可減少死亡率。在此次單中心研究中，研究者統計了在當前治療 PHT 的策略下麻醉期間主要和次要事件發生率。

方法：研究者回顧了 2008 年至 2012 年間 PHT 患兒進行非心肺轉流手術的記錄。研究者記錄了麻醉前的臨床主要症狀，體征和研究資料，並收集了圍術期併發症和死亡（至術後 7 天）的發生率及類型。

結果：資料來自于 122 位元患者進行的 284 例手術。次要（3.9%）和主要（3.2%）併發症的發生率和先前的報導沒有發生變化。PHT 的病因和併發症無顯著相關性（P＝0.14）。疾病導向的治療和併發症減少無相關性：治療組 4.1%，未治療組 8.6%（組間比 P 值均大於 0.14）。接受家庭氧療的患兒有更多的併發症（P＝0.02）。多因素 Logistic 回歸分析發現年齡和 PHT 分級是併發症的有效預測因素（ P 值均小於等於 0.03）。

結論：儘管使用了最新的以疾病爲導向的治療措施，麻醉期間 PHT 患兒的不良事件風險依然很高。併發症的危險因素包括年齡和 PHT 的嚴重程度。

（張帆 譯 陳傑 校）

BACKGROUND: Pulmonary hypertension (PHT) is a significant risk factor for major adverse events during anesthesia, with a reported incidence of 5% to 7%, secondary to acute pulmonary hypertensive crises or right ventricular ischemia. Newer therapies for treating PHT have reduced mortality. In this single-center study, we investigated the frequency of major and minor events during anesthesia under the current strategies to manage PHT.

METHODS: We reviewed the records of children with PHT who underwent noncardiopulmonary bypass procedures from 2008 to 2012. Clinically important symptoms, physical signs, and results of investigations present before anesthesia were recorded. The incidence and type of intraoperative complications and death (up to 7 days) were collected.

RESULTS: Data were collected for 122 patients undergoing 284 procedures. Minor (3.9%) and major (3.2%) complication rates were unchanged from previous publications. The etiology of PHT was not significant for complications (P = 0.14). Disease-modifying agents were not associated with reduced complications: 4.1% in treated versus 8.6% untreated (all P > 0.14). Patients receiving home oxygen had more complications (P = 0.02). Multiple logistic regression identified age and degree of PHT as significant predictors of complications (all P ≤ 0.03).

CONCLUSIONS: The risk for adverse events during anesthesia in patients with PHT remains high, despite newer disease-modifying treatments. Risk factors for complications include age and severity of PHT.

在神經病理性疼痛的大鼠模型中鞘內給予安非他酮，一種多巴胺和去甲腎上腺素再攝取抑制劑的抗痛覺過敏效應

The Antihyperalgesic Effects of Intrathecal Bupropion, a Dopamine and Noradrenaline Reuptake Inhibitor, in a Rat Model of Neuropathic Pain

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背景:抗抑鬱藥物通常用於治療神經病理性疼痛，通過增加脊髓的去甲腎上腺素和 5 -羥色胺水準達到鎮痛效果。臨床研究也表明：作爲一種多巴胺和去甲腎上腺素再攝取抑制劑，安非他酮的對治療神經病理性疼痛具有很強療效，然而，脊髓多巴胺在神經病理性疼痛方面的作用機制尚不清楚。本文假設安非他酮通過增加脊髓的去甲腎上腺素和多巴胺水準抑制神經病理性疼痛。此項研究考察了對神經病理性疼痛的大鼠模型鞘內注射安非他酮，其有效性及潛在機制。

方法：雄性 SD 大鼠麻醉後，右側 L5 脊神經進行結紮(SNL)以生產後足機械性痛覺過敏。在有或沒有鞘內注射 α2 腎上腺素受體和多巴胺 D2 受體拮抗劑條件下，在鞘內注射安非他酮前後測量退縮閾值以完成爪壓測試。鞘內注射安非他酮後對在體腰髓灰質後角的微量透析可以測量去甲腎上腺素和多巴胺的濃度。同時也測量了正常大鼠和 SNL 後 2、3、4 周大鼠同側腰髓後角中去甲腎上腺素和多巴胺含量。

結果:鞘內注射安非他酮產生劑量依賴性抗痛覺過敏效應 (3、10、30 和 100 ug,P < 0.001)。使用(安非他酮注射前 15 分鐘)α2-腎上腺素受體拮抗劑咪唑克生(3、10 和 30 ug,P < 0.001)和 D2 受體拮抗劑舒必利(3、10 和 30 ug,P < 0.001)進行鞘內預處理可劑量依賴性地逆轉此效應（30ug 組）。微量透析表明在鞘內注射安非他酮(30 ug)後脊髓背角去甲腎上腺素和多巴胺濃度增加（分別 P < 0.001,P = 0.001）。此外脊髓背角的去甲腎上腺素和多巴胺含量在 SNL 2 周後增加(分別 P < 0.001,P = 0.044),然後逐漸下降。

結論:這些研究結果表明,下行抑制通路，如去甲腎上腺素和多巴胺系統，其適應性與神經病理性疼痛的維持相關，脊髓去甲腎上腺素和多巴胺在神經病理性疼痛中起到抑制作用。

（ 秦懿 譯 陳傑 校）

BACKGROUND: Antidepressants are often used for the treatment of neuropathic pain, and their analgesic effects rely on increased noradrenaline and serotonin levels in the spinal cord. Clinical studies have also shown that bupropion, a dopamine and noradrenaline reuptake inhibitor, has strong efficacy in neuropathic pain; however, the role of spinal cord dopamine in neuropathic pain is unknown. We hypothesized that bupropion inhibits neuropathic pain by increasing noradrenaline and dopamine in the spinal cord. In the present study, we determined the efficacy and underlying mechanisms of intrathecal administration of bupropion in a rat model of neuropathic pain.

METHODS: Male Sprague-Dawley rats were anesthetized, and right L5 spinal nerve ligation (SNL) was performed to produce mechanical hyperalgesia of the hindpaw. Withdrawal threshold to a paw pressure test was measured before and after intrathecal administration of bupropion, without or with intrathecal antagonists for α2-adrenoceptors and dopamine D2 receptors. In vivo microdialysis was performed in the dorsal horn of the lumbar spinal cord to measure noradrenaline and dopamine concentrations after intrathecal injection of bupropion. We also measured the noradrenaline and dopamine contents in the ipsilateral dorsal lumbar spinal cord in normal rats and in rats 2, 3, and 4 weeks after SNL.

RESULTS: Intrathecal injection of bupropion produced a dose-dependent antihyperalgesic effect (3, 10, 30, and 100 ug, P < 0.001). The effect (30 ug) was dose-dependently reversed by intrathecal pretreatment (15 minutes before bupropion injection) with the α2-adrenoceptor antagonist idazoxan (3, 10, and 30 ug, P < 0.001) and D2 receptor antagonist sulpiride (3, 10, and 30 ug, P < 0.001). Microdialysis revealed that noradrenaline and dopamine concentrations in the spinal dorsal horn were increased after intrathecal injection of bupropion (30 ug, P < 0.001 and P = 0.001, respectively). Furthermore, the noradrenaline and dopamine contents in the spinal dorsal horn were increased 2 weeks after SNL (P < 0.001 and P = 0.044, respectively) and then decreased gradually.

CONCLUSIONS: These findings suggest that plasticity of descending inhibitory pathways such as the noradrenaline and dopamine systems contributes to the maintenance of neuropathic pain and that spinal cord noradrenaline and dopamine both play an inhibitory role in neuropathic pain.

一項關於脊麻複合靜脈鎮靜下行全膝置換術時鼻持續正壓通氣對動脈二氧化碳分壓影響的初步研究

A Pilot Study on the Effect of Nasal Continuous Positive Airway Pressure on Arterial Partial Pressure of Carbon Dioxide During Spinal Anesthesia with Intravenous Sedation for Total Knee Arthroplasty

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背景：儘管高碳酸血症程度很少被量化，手術患者的深度鎮靜可能與低通氣、氣道塌陷和高碳酸血症相關。 這項前瞻、隨機、對照臨床初步研究評估了對脊麻（SAB）複合深度鎮靜下行全膝置換術（TKA）保持自主呼吸的患者進行鼻持續性正壓通氣(nCPAP)與標準氣道管理的差異。

方法：納入 40 名 ASA 分級 I-III、SAB 下行 TKA 患者，術中給予異丙酚使患者深鎮靜程度達到改良觀察者評估警覺性/鎮靜評分（MOAA/SS ）2 級。 由麻醉團隊自行決定鼻腔或口腔通氣裝置的放置，但不能與 nCPAP 同時使用。 當 MOAA/SS 達到 2 級時，進行動脈血氣分析(ABG-1)。之後患者被隨機分配接受 nCPAP(nCPAP 組,N= 20)或標準氣道管理（對照組,N = 20）處理。 30 分鐘後進行第二次 ABG(ABG-2)以評估 nCPAP 對 PaCO2 的影響。 主要療效終點是從基線至第 30 分鐘時 PaCO2 的改變。

結果: nCPAP 與對照組基礎 PaCO2 值相似，分別爲 54.5 和 56.1mmHg。而與對照組（中位數 0.95 mm Hg [10%至 90%四分位間距爲−4.75 至 9.85]）相比， nCPAP 組的 PaCO2 明顯下降（ 中位數−4.6 mm Hg[四分位間距爲−14.75 至 9.85]) (P = 0.015；95%可信區間[CI]爲−9.5−9.5)。 對照組 PaCO2 從 ABG-1 至 ABG-2 是相似的(中位數[10%至 90% 四分位間距]爲 56.1 mm Hg [47.2–67.0] 與 56.6 mm Hg [46–68.8]相比; P = 0.52; 中位數的 95%可信區間爲 −3.4 to 3.4)。 40%患者在 ABG1 之前都接受了一種通氣裝置。這些患者的基線 PaCO2 值與非氣道裝置患者相似。

結論: 對在 SAB 下行 TKA 的患者進行深度鎮靜可導致中度高碳酸血症(平均值和中位數 PaCO2=55mmHg)。 實驗表明與接受標準氣道管理的患者相比，接受 nCPAP 處理的患者有減少 PaCO2 趨勢。然而估計處理間差異的範圍較大， 從 1.4 至 12.6mmHg 不等。接受深度鎮靜的對照組患者，基線 PaCO2 類似于持續深鎮靜後 30min 的 PaCO2。 最後事先給予或不給予氣道裝置的深鎮靜患者，其基線 PaCO2 並無差異。

（李慧 譯 陳傑 校）

BACKGROUND: Deep sedation of surgical patients may be associated with hypoventilation, airway collapse, and hypercarbia, although the extent of hypercarbia is rarely quantified. In this prospective, randomized, controlled clinical pilot study, we assessed the efficacy of nasal continuous positive airway pressure (nCPAP) for reducing arterial partial pressure of carbon dioxide (PaCO2) among deeply sedated, spontaneously ventilated patients undergoing total knee arthroplasty (TKA) under subarachnoid block (SAB), versus standard airway management in a control group.

METHDS: Forty ASA status I–III patients underwent deep sedation with propofol to level 2 on the Modified Observers Assessment of Alertness/Sedation Scale during TKA performed under SAB. Nasal or oral airways were placed at the discretion of the anesthesia team, but they were not used in conjunction with nCPAP. Baseline arterial blood gas analysis (ABG-1) was performed after Modified Observers Assessment of Alertness/Sedation Scale level 2 was reached. Patients were then randomized to receive nCPAP (nCPAP group, N = 20) or standard oxygen mask management (control group, N = 20). A second ABG (ABG-2) was performed 30 minutes later to assess the effect of nCPAP on PaCO2. The primary efficacy end point was change in PaCO2 from baseline to the 30-minute time point.

RESULTS: Baseline (ABG-1) PaCO2 values were similar between nCPAP and control groups with median values of 54.5 and 56.1 mm Hg, respectively. There was a significant decline in PaCO2 in the nCPAP group (median of −4.6 mm Hg [10th–90th quantile, −14.55 to 3.85]) as compared with the control group (median of 0.95 mm Hg [−4.75 to 9.85]; P = 0.015; 95% confidence interval [CI] for location shift = −9.5 to −1.3). Within the control group, PaCO2 was similar from ABG-1 to ABG-2 (median [10th–90th quantile] = 56.1 mm Hg [47.2–67.0] vs 56.6 mm Hg [46–68.8]; P = 0.52; 95% CI for the median = −3.4 to 3.4). Forty percent of all patients received an airway before ABG-1. The baseline PaCO2 value of patients receiving an airway was not different from that of patients without an airway (median [10th–90th quantile] = 56.0 mm Hg [46.0–68.4] vs 54.1 mm Hg [45.6–65.6], respectively; P = 0.33; 95% CI for location shift = −2.30 to 7.20).

CONCLUSIONS: Deep sedation of TKA patients during SAB resulted in moderate hypercarbia (mean and median PaCO2 = 55). There was a trend showing that nCPAP treatment reduced PaCO2 versus treatment for control group patients receiving standard airway management; however, estimated treatment difference varied widely, from 1.4 to 12.6 mm Hg. Among control group patients, the initial PaCO2 during deep sedation was similar to the PaCO2 when measured after a 30-minute period of continued deep sedation. Finally, baseline PaCO2 among deeply sedated patients who received an airway was not different from that of patients who did not receive an airway.

呼氣末正壓通氣在外科領域條件下對功能性內窺鏡鼻竇手術的影響

The influence of positive end-expiratory pressure on surgical field conditions during functional endoscopic sinus surgery.

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背景：功能性內窺鏡鼻竇手術（FESS）是鼻腔鼻竇疾病外科治療中的中流砥柱。這個手術也有一定的風險。大部分風險與手術的品質有關。因此，研究這種能提高手術品質的機制很重要。我們試圖確定呼氣末正壓通氣（PEEP）是否對急診手術患者的術野品質產生有害的影響。

方法：407 例患者隨機採用 5 cm H2O PEEP 或 零 PEEP 通氣策略。手術野的品質每 15 分鐘用一個有效的手術評分方法進行測量。

結果：PEEP 的加入沒有對術後品質發生任何可衡量的效果（比值比[OR]（95%置信區間[CI]）= 1.06，P = 0.895（0.44-2.58）側面 1；或（95% CI）= 0.56，P = 0.356（0.16-1.93）側面 2）。吸氣峰壓確實影響手術成績。每增加 1 cm H2O 的壓力，超過 15 cm H2O 的總壓力將貢獻于增加更高的手術得分率。每增加 1 cm H2O 的吸氣壓，將有超過 15 cm H2O 用於增加手術得分。

結論：在鼻內鏡手術中通過增加平均吸氣壓力低於 15cm H2O 的 PEEP，可以避免手術野的模糊程度。

（王曉莉譯 李士通審校）

BACKGROUND: Functional endoscopic sinus surgery (FESS) is the mainstay of surgical treatment for sinonasal disease. This surgery carries certain risks. Most of these risks relate to the quality of the surgical field. Thus, mechanisms by which the surgical field can be improved are important to study. We sought to determine whether positive end-expiratory pressure (PEEP) had a deleterious effect on the quality of the surgical field in patients undergoing primary FESS.

METHODS: Forty-seven patients were randomized to a ventilation strategy using either 5 cm H2O of PEEP or zero added PEEP. The quality of the surgical field was measured every 15 minutes using a validated surgical scoring method.

RESULTS: The addition of PEEP did not have any measurable effect on the surgical field scores after onset of surgery (odds ratio [OR] (95% confidence interval [CI]) = 1.06 (0.44-2.58), P = 0.895 for side 1; OR (95% CI) = 0.56 (0.16-1.93), P = 0.356 for side 2). The peak inspiratory pressure did have an effect on surgical grades. Every cm H2O of added pressure over 15 cm H2O total pressure contributing to increased odds of higher surgical field score. For each cm H2O increase in inspiratory pressure above 15cm H2O increased the surgical field score (OR [95% CI] 1.13 [1.04-1.22], P = 0.002).

CONCLUSIONS: During FESS surgery if PEEP is added, it is important to keep the mean inspiratory pressure below 15cm H2O to avoid worsening surgical field conditions.

丙泊酚（得普利麻）和脂肪乳通過削弱 GLUT4 的轉運增強 2 型糖尿病病人心臟的胰島抵抗

Propofol (Diprivan®) and Intralipid® Exacerbate Insulin Resistance in Type-2 Diabetic Hearts by Impairing GLUT4 Trafficking.

Lou PH[1], Lucchinetti E, Zhang L, Affolter A, Gandhi M, Zhakupova A, Hersberger M, Hornemann T, Clanachan AS, Zaugg M.

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背景：當靜脈麻醉藥——丙泊酚作爲脂肪乳劑基製劑給藥（得普利麻）時，能促進胰島素抵抗，但丙泊酚和它的溶劑，脂肪乳，對心臟胰島素抵抗的直接影響是未知的。

方法：健康和 2 型糖尿病大鼠的心臟（果糖餵養產生）的進行 10μM 異丙酚或相同濃度的溶劑脂肪乳劑（25μM）±胰島素（100U•L） 有氧灌注 60 分鐘。葡萄糖攝取，糖酵解和糖原代謝用葡萄糖測定。 Akt 和 GSK3β，AMPK，ERK1 / 2，p38MAPK 和 S6K1，JNK，蛋白激酶 Cθ（PKCθ），以及蛋白激酶 CCβII（PKCβII）的活化，用免疫印跡測定。 GLUT4 和胰島素受體底物-1（IRS-1）的 Ser307（H312），Ser1100（H1101）的磷酸化物，和 Tyr608（hTyr612）進行了測定。質譜用於測定醯基肉堿，磷脂和鞘脂。

結果：得普利麻和脂肪乳降低胰島素誘導葡萄糖的攝取和重定向葡萄糖糖原儲備糖尿病。降低葡萄糖的攝取，伴隨著較低的 GLUT4 轉運肌膜。得普利麻和脂肪乳滅活 GSK3β 但糖尿病心活化 AMPK 和 ERK1 / 2。得普利麻只增加 Akt 磷酸化（的 Ser473 / Thr308）和易位 PKCθ 和 PKCβII 的在健康的心臟肌膜，而它在糖尿病的心啓動 S6K1 以及 p38 和易位 PKCβII。此外，只有得普利麻在健康和糖尿病心臟磷酸 IRS-1 在 Ser1100（H1101）。 JNK 表達，磷酸化的 IRS-1 Ser307（H312），並 PKCθ 表達和轉位的增加，而 GLUT4 表達減少胰島素治療的糖尿病心。磷脂醯甘油，磷脂，和 C18-鞘脂積累得普利麻灌注和脂肪乳灌注糖尿病心。

結論：丙泊酚和脂肪乳主要是促進 2 型糖尿病患者的心臟胰島素抵抗。

（王曉莉譯 李士通審校）

BACKGROUND: The IV anesthetic, propofol, when administered as fat emulsion-based formulation (Diprivan) promotes insulin resistance, but the direct effects of propofol and its solvent, Intralipid, on cardiac insulin resistance are unknown.

METHODS: Hearts of healthy and type-2 diabetic rats (generated by fructose feeding) were aerobically perfused for 60 minutes with 10 μM propofol in the formulation of Diprivan or an equivalent concentration of its solvent Intralipid (25 μM) ± insulin (100 mU•L). Glucose uptake, glycolysis, and glycogen metabolism were measured using [H]glucose. Activation of Akt, GSK3β, AMPK, ERK1/2, p38MAPK, S6K1, JNK, protein kinase Cθ (PKCθ), and protein kinase CCβII (PKCβII) was determined using immunoblotting. GLUT4 trafficking and phosphorylations of insulin receptor substrate-1 (IRS-1) at Ser307(h312), Ser1100(h1101), and Tyr608(hTyr612) were measured. Mass spectrometry was used to determine acylcarnitines, phospholipids, and sphingolipids.

RESULTS: Diprivan and Intralipid reduced insulin-induced glucose uptake and redirected glucose to glycogen stores in diabetic hearts. Reduced glucose uptake was accompanied by lower GLUT4 trafficking to the sarcolemma. Diprivan and Intralipid inactivated GSK3β but activated AMPK and ERK1/2 in diabetic hearts. Only Diprivan increased phosphorylation of Akt(Ser473/Thr308) and translocated PKCθ and PKCβII to the sarcolemma in healthy hearts, whereas it activated S6K1 and p38MAPK and translocated PKCβII in diabetic hearts.

Furthermore, only Diprivan phosphorylated IRS-1 at Ser1100(h1101) in healthy and diabetic hearts. JNK expression, phosphorylation of Ser307(h312) of IRS-1, and PKCθ expression and translocation were increased, whereas GLUT4 expression was reduced in insulin-treated diabetic hearts. Phosphatidylglycerol, phosphatidylethanolamine, and C18-sphingolipids accumulated in Diprivan-perfused and Intralipid-perfused diabetic hearts.

CONCLUSIONS: Propofol and Intralipid promote insulin resistance predominantly in type-2 diabetic hearts

在一系列體外的實驗中發現硬質和軟質氣管交換導管所造成的明顯的肺損傷

Macroscopic Barotrauma Caused by Stiff and Soft-Tipped Airway Exchange Catheters: An In Vitro Case Series

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背景：許多呼吸道管理指南包括氣道交換導管都有所報導，但是在使用過程中，不論是導管位置異常還是通過氣道交換導管給氧都會導致氣道壓傷。

方法：我們運用離體豬肺模型來觀察 2 種不同的氣道交換導管，一種爲標準導管，而另一種是軟導管，分別通過他們給予高壓源下的 4 種不同流速的氧氣。在實驗過程中，導管位置可以在在隆突上方也可以在其下方，最重要的就是對抗其阻力，保持位置固定。這項實驗選擇了 32 例樣本。

結果：我們發現，置於隆突上方的氣道交換導管在給氧後沒有造成明顯的肺損傷。但是導管位置在隆突下方的案例中，給氧後我們發現，不論氧氣的流速是多少，都會造成明顯的肺損傷，而氧流速越大，造成的肺損傷越快而且越廣泛。通過使用 2.5 或 4 條的“注射器”將會立即導致肺組織損傷，而且氣道交換導管會導致損傷的進一步加劇。同時我們發現兩種不同的氣道交換導管的結果造成的損傷都是一樣的。

結論：我們的實驗結果和以往關於放置于隆突下方的氣道交換導管造成的肺損傷的報導是相同的，並且證明了放置于隆突下方的氣道交換導管給氧的風險。一種新的氣道交換導管，在製造時設計爲放置于牙齒水準，將會減少原來置於隆突水準的氣道交換導管所造成的損傷，並且增加使用這種導管的安全性。

（李蔚文譯 李士通審校）

BACKGROUND: Many airway management guidelines include the use of airway exchange catheters (AECs). There are reports, however, of harm from their use, from both malpositioning and in particular from the administration of oxygen via an AEC leading to barotrauma.

METHODS: We used an in vitro pig lung model to investigate the safety of administering oxygen at 4 different flow rates from a high-pressure source via 2 different AECs: a standard catheter and a soft-tipped catheter. Experiments were performed with the catheters positioned either above the carina or below it at the first point of resistance to advancement (hold-up). The experiments were then repeated to produce a series of 32 cases.

RESULTS: With an AEC positioned above the carina, we did not observe macroscopic lung damage after the administration of oxygen. The administration of oxygen through an AEC positioned below the carina resulted in macroscopic barotrauma regardless of the rate of oxygen delivery. Increasing speed of oxygen flow led to faster and more extensive damage. Use of an “injector” at 2.5 or 4 bar led to instantaneous macroscopic lung damage and advancement of the AEC through the lung tissue. Our observations were the same when both types of AECs were used.

CONCLUSIONS: Our results are consistent with reports of harm during the use of AECs and demonstrate the risk of administering oxygen through these devices when they are positioned below the carina. An indicator, ideally made on an AEC at the time of manufacture and designed to lie at the same level as the teeth, may be useful in preventing the insertion of that AEC beyond the level of the carina and improve the safety of using such devices.

外源性表面活性物質的治療對於二次損傷的小鼠肺模型的影響

The Effects of Exogenous Surfactant Treatment in a Murine Model of Two-Hit Lung Injury

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背景：在急性呼吸窘迫綜合征的患者中，我們發現肺內源性表面活性物質生成發生了改變，而肺表面活性物質的替代劑的使用將會改變臨床結果。然而，一些試驗中的肺表面活性物質已經造成了混亂的結果。我們設計了動物右肺損傷的模型來研究外源性表面活性物質對於已經損傷以及未損傷的肺所造成的炎症方面的影響。

方法：我們將鹽酸（1.5mL/kg）灌入小鼠的右側支氣管，並且延長機械通氣（25mL/kg）時間（7h）。在 3 小時後，實驗組的小鼠右肺給予 1mL/kg 的外源性表面活性物質，而鹽水組小鼠右肺給予 0.9%的氯化鈉，另外對照組小鼠沒有給予任何處理（包括鹽酸灌注和呼吸機損傷）。我們從氣體交換、肺順應性和支氣管肺泡炎症（包括細胞、蛋白和細胞因數）這幾方面來評價實驗結果。我們運用顯著的方差分析和 Tukey 事後檢驗進行系統分析。

結果：每組的小鼠中至少十分之八存在評估的變數可以分析。表面活性物質處理後明顯增加了吸入的氧氣比分數後的動脈血氧張力，並且明顯提高了呼吸系統靜態順應性（分別爲實驗組 P=0.027，鹽水組 P=0.007）。表面活性物質明顯增強了支氣管肺泡炎症反應，炎性細胞：實驗組爲 685（602-733），鹽水組爲 216（125-305）*1000/mL(P<0.001)；在肺泡灌洗液中的蛋白，實驗組爲 1442±588，鹽水組爲 743±647μg/mL（P=0.027）。然而這些差異在對側健肺中並沒有被發現（P=0.96，P=0.54，炎症細胞 131（78-195）和 119（87-149）*1000/mL，蛋白 135±100 和 173±115μg/mL）。

結論：外源性表面活性物質用於鹽酸灌注損傷的右肺，將會增加肺的氣體交換和整個呼吸系統的順應性。但是，損傷的右肺中炎症反應標誌物也會增加，儘管這些在左側健肺中沒有被發現。這些資料表明肺表面活性物質對於損傷和未損傷的肺泡都有增強其功能的作用。

（李蔚文譯 李士通審校）

BACKGROUND: Because pulmonary endogenous surfactant is altered during acute respiratory distress syndrome, surfactant replacement may improve clinical outcomes. However, trials of surfactant use have had mixed results. We designed this animal model of unilateral (right) lung injury to explore the effect of exogenous surfactant administered to the injured lung on inflammation in the injured and noninjured lung.

METHODS: Mice underwent hydrochloric acid instillation (1.5 mL/kg) into the right bronchus and prolonged (7 hours) mechanical ventilation (25 mL/kg). After 3 hours, mice were treated with 1 mL/kg exogenous surfactant (Curosurf®) (surf group) or sterile saline (NaCl 0.9%) (vehicle group) in the injured (right) lung or did not receive any treatment (hydrochloric acid, ventilator-induced lung injury). Gas exchange, lung compliance, and bronchoalveolar

inflammation (cells, albumin, and cytokines) were evaluated. After a significant analysis of variance (ANOVA) test, Tukey post hoc test was used for statistical analysis.

RESULTS: At least 8 to 10 mice in each group were analyzed for each evaluated variable. Surfactant treatment significantly increased both the arterial oxygen tension to fraction of inspired oxygen ratio and respiratory system static compliance ($P = 0.027$ and $P = 0.007$, respectively, for surf group versus vehicle). Surfactant therapy increased indices of inflammation in the acid-injured lung compared with vehicle: inflammatory cells (685 [602–773] and 216 [125–305] × 1000/mL, respectively; $P < 0.001$) and albumin in bronchoalveolar lavage (BAL) (1442 ± 588 and 743 ± 647 μg/mL, respectively; $P = 0.027$). These differences were not found ($P = 0.96$ and $P = 0.54$) in the contralateral (uninjured) lung (inflammatory cells 131 [78–195] and 119 [87–149] × 1000/mL and albumin 135 ± 100 and 173 ± 115 μg/mL).

CONCLUSIONS: Exogenous surfactant administration to an acid-injured right lung improved gas exchange and whole respiratory system compliance. However, markers of inflammation increased in the right (injured) lung, although this result was not found in the left (uninjured) lung. These data suggest that the mechanism by which surfactant improves lung function may involve both uninjured and injured alveoli.

由於小兒圍手術期呼吸事件產生的醫院額外費用和住院時間

Excess Costs and Length of Hospital Stay Attributable to Perioperative Respiratory Events in Children

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背景：由於圍手術期呼吸事件導致多餘的住院費用和住院時間的認知在醫院規劃是有用的。在這項研究中，我們在泰國南部的一家三級醫院，比較小兒有圍手術期呼吸事件和沒有該事件成本（多餘的住院費用和間接費用）和住院長度。

方法：對 2012 十一月到 2013 十二月在 songklanagarind 兒童醫院年齡＜15 歲患者，全麻兒童進行前瞻性佇列研究。孩子們沒有孩子前預匹配（1:1）使用一個隨機選擇的程式對門診/住院，手術類型，手術費（泰銖），ASA 分級，年齡＜9 歲，和不同的手術＜6 個月的時間進行分組。主要終點是術後多餘的住院費用和住院天數。手術後住院天數，多餘的住院費用和間接費用，組與組之間的父母的收入損失進行比較採用 Wilcoxon 符號秩檢驗。術後住院天數比較採用 McNemarχ 檢測。障礙模型被用來預測術後住院天數和住院天數。多個混合效應線性回歸被用來確定預測的調整多餘的住院費用和間接費用

結論：研究共包括 430 名兒童（215 配對）。更多的呼吸事件的孩子需要術後住院（81% vs 72%，P = 0.004），並且有較長的住院天數手術後（中位數[四分位數]：1 [1] 1 [0 VS 3.5—2]；P＜0.001）和產生較高的超額成本（P＜0.001）而不是間接成本（P = 0.23）。在多變數分析中，圍手術期呼吸事件是一個重要的預測因數，預測手術後住院時長（優勢比，2.56；95%置信區間，）成本（成本比 1.30 [1.12，1.53]）和間接成本（成本比 1.58 [1.20，2.08]），可以調整病人的麻醉特點。普遍覆蓋（74%）與 35%和 64%更高的超額成本與審計總署相比較（17%）和自付（7%），分別爲（P = 0.003）。

（田園譯 李士通審校）

BACKGROUND: Knowledge of the excess hospital costs and prolonged length of stay attributable to perioperative respiratory event (PRE) in pediatric anesthesia is useful for hospital planning. In this study, we compared costs (excess hospital costs and indirect costs) and length

of hospital stay between children who had PRE and did not have PRE for noncardiac surgery at a tertiary care hospital in southern Thailand.

METHODS: A prospective matched cohort study was conducted in children aged <15 years who underwent general anesthesia between November 2012 and December 2013 at Songklanagarind Hospital. PRE children were matched with no PRE children (1:1) using a random selection procedure on outpatients/inpatients, type of surgery, surgical charge (baht), ASA physical status, age difference <9 years, and difference in time of surgery <6 months. Primary end points were excess hospital costs and number of days hospitalized after surgery. Number of days hospitalized after surgery, excess hospital costs and indirect costs regarding transportation, and income loss of parents between groups were compared using Wilcoxon signed rank test. Any hospital stay after surgery between groups was compared using McNemar χ^2 test. A hurdle model was used to predict any hospital stay and number of days hospitalized after surgery. Multiple mixed-effects linear regression was used to identify predictors of adjusted excess hospital costs and indirect costs.

RESULTS: A total 430 children were included (215 matched pairs). More PRE children required hospital stay after surgery (81% vs 72%, $P = 0.004$), and PRE children had a longer number of days hospitalized after surgery (median [interquartile ranges]: 1 [1–3.5] vs 1 [0–2];$P < 0.001$) and incurred higher excess costs ($P < 0.001$) but not indirect costs ($P = 0.23$). In multivariate analysis, PRE was a significant predictor for hospital stay after surgery (odds ratio, 2.56; 95% confidence interval, 1.23–5.31), longer hospitalization (count ratio, 2.10 [1.31–3.35]), higher excess costs (cost ratio, 1.30 [1.12–1.53]), and indirect cost (cost ratio, 1.58 [1.20–2.08]) after adjusting for patient and anesthesia characteristics. Universal coverage (74%) was associated with 35% and 64% higher excess cost compared with the Comptroller General's Department (17%) and self-pay (7%), respectively ($P = 0.003$).

CONCLUSIONS: The effects of PRE in pediatric anesthesia were hospital stay after surgery, 2 times longer hospitalization, 30% higher excess hospital costs, and 58% higher indirect cost among outpatients. Hospital policy to efficiently manage hospital beds and compensatory budget should be developed.

異丙酚誘導新生大鼠腦電圖發作：糖皮質激素和 γγ-氨基丁酸 A 型受體介導的激勵作用

Propofol-Induced Electroencephalographic Seizures in Neonatal Rats: The Role of Corticosteroids and γ-Aminobutyric Acid Type A Receptor-Mediated Excitation

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背景：中樞神經系統興奮和抑制之間的不平衡可能會導致病理結果。我們研究麻醉劑異丙酚通過內分泌活性物質和 γ-氨基丁酸 A 型受體（GABAAR）介導的選擇性激發 GABAAR 對新生大鼠腦電圖癲癇發作作用機理

方法：出生 4 天的 SD 大鼠 6，接受小手術植入電極，腹腔注射異丙酚（40 毫克公斤−•1）前 1 小時和 1 小時後觀察腦電活動（40 毫克公斤−•1）。各種治療前給予丙泊酚 15 分鐘。

結論：異丙酚麻醉時發生的腦電圖癲癇發作樣持續低幅度尖峰。血清多種皮質酮增加（T（10）=−5.062；P = 0.0005）醛固酮（T（10）=−5.069；P = 0.0005）增加，在動物身上進行的實驗操作相同，見於研究腦電活動丙泊酚給藥後 1 小時。預處理與布美他尼，Na+ K+—2Cl–共轉運體抑制劑，從而減少 GABAAR 介導的激勵，消除異丙酚引起發作尖峰腦電活動。鹽皮質激素和糖皮質激素受體拮抗劑 RU486，RU 28318 和抑鬱症腦電圖癲癇發

作，但不影響丙泊酚的尖峰腦電圖的影響。依託咪酯，在劑量足以引起翻正反射，弱增加血清皮質醇水準和誘發腦電圖癲癇發作。

（田園譯 李士通審校）

BACKGROUND: An imbalance between excitation and inhibition in the developing central nervous system may result in a pathophysiological outcome. We investigated the mechanistic roles of endocrine activity and γ-aminobutyric acid type A receptor (GABAAR)-mediated excitation in electroencephalographic seizures caused by the GABAAR-selective anesthetic propofol in neonatal rats.

METHODS: Postnatal day 4–6 Sprague Dawley rats underwent a minor surgical procedure to implant electrodes to measure electroencephalographic activity for 1 hour before and 1 hour after intraperitoneal administration of propofol (40 mg·kg−1). Various treatments were administered 15 minutes before administration of propofol.

RESULTS: Episodes of electroencephalographic seizures and persistent low-amplitude spikes occurred during propofol anesthesia. Multifold increases in serum levels of corticosterone ($t(10) = -5.062$; $P = 0.0005$) and aldosterone ($t(10) = -5.069$; $P = 0.0005$) were detected 1 hour after propofol administration in animals that underwent experimental manipulations identical to those used to study electroencephalographic activity. Pretreatment with bumetanide, the Na+–K+–2Cl– cotransporter inhibitor, which diminishes GABAAR-mediated excitation, eliminated both seizure and spike electroencephalographic activities caused by propofol. Mineralocorticoid and glucocorticoid receptor antagonists, RU 28318 and RU486, depressed electroencephalographic seizures but did not affect the spike electroencephalographic effects of propofol. Etomidate, at a dose sufficient to induce loss of righting reflex, was weak at increasing serum corticosteroid levels and eliciting electroencephalographic seizures. Etomidate given to corticosterone-pretreated rat pups further increased the total duration of electroencephalographic seizures caused by administration of exogenous corticosterone ($t(21) = -2.512$, $P = 0.0203$).

CONCLUSIONS: Propofol increases systemic corticosteroid levels in neonatal rats, which along with GABAAR-mediated excitation appear to be required for propofol-induced neonatal electroencephalographic seizures. Enhancement of GABAAR activity alone may not be sufficient to elicit neonatal electroencephalographic seizures.

大鼠急性術後疼痛模型中自發痛行爲比機械誘發疼痛對嗎啡或丁丙諾啡更敏感

Spontaneous Pain-Like Behaviors Are More Sensitive to Morphine and Buprenorphine Than Mechanically Evoked Behaviors in a Rat Model of Acute Postoperative Pain

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背景：nonevoked 自發性疼痛是術後最棘手的問題。醫師通常利用人類視覺類比評分或口頭的數位評定量表評估這種形式的疼痛。最近的研究提出，自發抬足（SFL）的行爲是一種動物脊髓神經損傷後表達自發性疼痛的行爲。在目前的研究中，我們系統的描述了大鼠急性術後疼痛行爲，其中包括比較鎮痛治療誘發的行爲

方法：對四系手動 5 分鐘的時間用 10 分鐘的時間間隔記錄每個測試結果。隨後用電子 Von Frey 計測定縮爪閾值。年齡的影響進行評估，大鼠在不同年齡組的測試：2，7，和大於 26 個月。丁丙諾啡和嗎啡的作用在單獨的一組動物進行了測試，測試前腹腔注射生理鹽水，嗎啡（0.01，0.1，1，或 2 毫克/公斤），或丁丙諾啡（0.001，0.01，或 0.1 毫克/公斤）

結果：SFL 行爲在切開後的第三或第四天顯著恢復 3 小時內達到高峰（P＜0.0001）。這些行爲的表現在不同的動物年齡（2，7，和 26 個月；P = 0.30 並沒有顯著不同。機械性痛覺過敏逆轉藥物的半數有效劑量（0.0452 毫克/公斤；95% CI，0.0259–0.0787）顯著大於反轉快速（0.0027 毫克/公斤；95% CI，0.0009–0.0083；P＜0.0001）和長期（0.0004 毫克/公斤，95% CI，0，0.0035；P = 0.001）切開後。同樣，在術後 3 小時，對於機械超敏反應行爲逆轉嗎啡的半數有效劑量（2.901 毫克/公斤；95% CI，1.132–7.436）大於 SFL 計數（0.4044 毫克/公斤；95% CI，0.1048–1.561；P = 0.0103）和功能的持續時間（0.0309 毫克/公斤；95% CI，0.0095–0.0998；P＜0.0001）。

結論：本研究表明，與機械誘發的行爲相比，誘導大鼠後肢足底切口 SFL 的行爲對鎮痛嗎啡與丁丙諾啡有較高的檢測靈敏度

（田園譯 李士通審校）

BACKGROUND: Nonevoked spontaneous pain is most problematic for postoperative patients. Physicians assess this form of pain using the human visual analog scale or verbal numeric rating scale. Recent studies have proposed that spontaneous foot-lifting (SFL) behaviors are an expression of spontaneous pain in animals after spinal nerve injury or adjuvant-induced inflammation. In the current study, we characterize SFL behaviors in a rat model of acute postoperative pain, which includes comparisons with evoked behaviors to analgesic treatments.

METHODS: SFL was manually recorded over four 5-minute periods with 10-minute intervals between each testing session. Paw-withdrawal thresholds were subsequently measured with an electronic von Frey esthesiometer. To evaluate the effects of age, rats were tested in different age groups: 2, 7, and >26 months. The effects of buprenorphine and morphine were tested in a separate group of animals, which received intraperitoneal injections of saline, morphine (0.01, 0.1, 1, or 2 mg/kg), or buprenorphine (0.001, 0.01, or 0.1 mg/kg) before testing.

RESULTS: SFL behaviors peaked at 3 hours after incision and significantly recovered by the 3rd or 4th postoperative day (P < 0.0001). The presentation of these behaviors did not significantly vary with animal age (2, 7, and >26 months old; P = 0.30). SFL behaviors, with the exception of rapid SFL at 3 hours after incision, did not show significant correlation with paw-withdrawal threshold behaviors. The median effective dose of buprenorphine for reversal of mechanical hyperalgesia (0.0452 mg/kg; 95% CI, 0.0259–0.0787) was significantly larger than for reversing rapid (0.0027 mg/kg; 95% CI, 0.0009–0.0083; P < 0.0001) and prolonged (0.0004 mg/kg, 95% CI, 0.0000, 0.0035; P = 0.001) SFL at 3 hours after incision. Similarly, the median effective dose of morphine for reversal of mechanical hypersensitivity behaviors (2.901 mg/kg; 95% CI, 1.132–7.436) was larger than for SFL count (0.4044 mg/kg; 95% CI, 0.1048–1.561; P = 0.0103) and SFL duration (0.0309 mg/kg; 95% CI, 0.0095–0.0998; P < 0.0001) at 3 hours after incision.

CONCLUSIONS: The present study demonstrates that a hindpaw plantar incision induces SFL behaviors in rats and that these behaviors have higher bioassay sensitivity to analgesic interventions with morphine and buprenorphine compared with mechanically evoked behaviors.