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心肺轉流術卒中實驗模型中抑肽酶可以改善腦功能但是並不能縮小腦梗塞的面積

Aprotinin Improves Functional Outcome but Not Cerebral Infarct Size in an Experimental Model of Stroke During Cardiopulmonary Bypass

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Anesth Analg 2010;111(1):38-45

背景：抑肽酶是一種非特異性的絲氨酸蛋白酶抑制劑，一直被用於減少心肺轉流術後的出血和減輕心肺轉流術後的全身性炎症反應。以往各種研究結果既有表明抑肽酶可以改善心臟術後腦功能的，也有結果是恰恰相反的。我們設計了本研究來測定應用抗炎劑量的抑肽酶是否能夠改善心肺轉流術中局部腦缺血模型大鼠的神經系統組織結構和功能。
方法：在外科手術準備後，小鼠被隨機分成兩組：抑肽酶組（60,000 kIU/kg 靜脈注射）和對照組（0.9% NaCl 靜脈注射）。溫度正常的心肺轉流術持續 60 分鐘，在此期間右大腦中動脈閉塞手術與心肺轉流術時間部分重疊，持續 60 分鐘。我們測定了操作前、心肺轉流術結束時、心肺轉流術結束後 2 和 24 小時時的細胞因數值（腫瘤壞死因數-α、白介素[IL]-1β、IL-6 和 IL-10）。在手術後第三天我們對小鼠進行了神經病學功能測試和腦梗死面積組織學測定。
結果：抑肽酶組和對照組相比，IL-1β 值 (*P* = 0.035) 和 IL-6 值(*P* = 0.047)都有所下降，表明了全身性炎症反應有所減輕。抑肽酶組神經病學功能測試結果也更好，抑肽酶組 27 [8]比對照組 32 [6]（中位數[四分位距]，*P* = 0.042)。但是兩組的腦梗塞面積沒有區別（抑肽酶組 306 [27] mm^3比對照組 297 [52] mm^3，*P* = 0.599）。
結論：在這個心肺轉流術期間發生的腦缺血實驗模型中，抑肽酶減輕了心肺轉流術引起的全身性炎症反應。雖然兩組的腦梗塞面積沒有區別，但是抑肽酶對短期的神經病學功能測試結果有些需改善。
（姜旭暉譯，馬皓琳，李士通校）

BACKGROUND: Aprotinin, a nonspecific serine protease inhibitor, has been used to decrease bleeding and reduce the systemic inflammatory response after cardiopulmonary bypass (CPB). Studies have variably linked aprotinin administration with both improved as well as adverse cerebral consequences after cardiac surgery. We designed this study to determine whether an antiinflammatory dose of aprotinin could improve the histologic and functional neurologic outcome in a rat model of focal cerebral ischemia during CPB.
METHODS: After surgical preparation, the animals were randomized into 2 groups: an aprotinin group (60,000 kIU/kg IV) and a control group (0.9% NaCl IV). Normothermic CPB was performed for 60 minutes during which time a partial overlapping 60 minutes of right middle cerebral artery occlusion was induced. Cytokines (tumor necrosis factor-α, interleukin [IL]-1β, IL-6, and IL-10) were measured at baseline, the end of CPB, then 2 and 24 hours after CPB. On postoperative day 3, the animals underwent functional neurologic testing and histologic assessment of cerebral infarct volume.
RESULTS: There was a reduction in systemic inflammation in the aprotinin group compared with the control group, demonstrated by lower levels of IL-1β (*P* = 0.035) and IL-6 (*P* = 0.047). The aprotinin group also had a better functional neurologic performance (median [interquartile range]: aprotinin 27 [8] vs control 32 [6]; *P* = 0.042). However, there was no difference in cerebral infarct volume (aprotinin 306 [27] mm^3 vs control 297 [52] mm^3; *P* = 0.599).

CONCLUSIONS: In this experimental model of stroke occurring during CPB, aprotinin decreased the systemic inflammatory response to CPB. Although there was no difference in the cerebral infarct volume, there was a small improvement in the short-term functional neurologic outcome in the aprotinin group.

使用異丙酚麻醉與七氟醚麻醉相比減輕日間手術病人的術後疼痛

Day-Surgery Patients Anesthetized with Propofol Have Less Postoperative Pain than Those Anesthetized with Sevoflurane

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Anesth Analg 2010;111(1):83-85

背景：近期有研究發現使用異丙酚麻醉與吸入麻醉藥麻醉相比可以減輕病人的術後疼痛。
方法：在本次隨機雙盲研究中，80 位接受日間婦科診斷性腹腔鏡手術的病人靜脈給予異丙酚或吸入七氟醚實施麻醉。主要的測量內容是用視覺類比評分（VAS）測量疼痛程度。
結果：接受異丙酚麻醉的病人與吸入七氟醚麻醉的病人相比疼痛較輕（P=0.01），其他測量的臨床指標之間無顯著差異。
結論：使用異丙酚麻醉與吸入麻醉藥麻醉相比病人的術後疼痛較輕。
（劉伍 譯 馬皓琳 李士通 校）

BACKGROUND: There have been recent studies suggesting that patients anesthetized with propofol have less postoperative pain compared with patients anesthetized with volatile anesthetics.
METHODS: In this randomized, double-blind study, 80 patients undergoing day-case diagnostic laparoscopic gynecological surgery were either anesthetized with IV propofol or sevoflurane. The primary outcome measured was pain on a visual analog scale.
RESULTS: Patients anesthetized with propofol had less pain compared with patients anesthetized with sevoflurane (P = 0.01). There was no difference in any of the other measured clinical outcomes.
CONCLUSIONS: The patients anesthetized with propofol appeared to have less pain than patients anesthetized with sevoflurane.

美國和歐洲神經肌肉阻滯管理的現狀調查

A Survey of Current Management of Neuromuscular Block in the United States and Europe

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Anesth Analg 2010;111(1):110-119

背景：術後殘餘神經肌肉阻滯經常發生。歐洲最近的臨床調查表明神經肌肉阻滯藥的使用常常沒有適當的監測。美國還沒有這方面的對照研究。通過本調查研究，我們比較了當前美國和歐洲麻醉醫生對神經肌肉阻滯藥的使用和態度。
方法：我們對美國和歐洲的麻醉醫生進行了一個以互聯網為基礎的調查。麻醉病人安全基金會和歐洲麻醉協會給它們的所有活躍會員發了電子郵件，邀請他們在一個專用的網際協議位址敏感的網站上不具名地回答一系列問題。這個調查在網上進行了 60 天。用 χ^2 核對總和 Fisher's 精確檢驗來比較 2 組的臨床調查專案。
結果：接受調查的總人數為 2636 人。美國(64.1%)和歐洲(52.2%)的大多數應答者評估臨床上顯著的術後殘餘神經肌肉無力的發生率<1% ($P < 0.0001$)。常規藥理學逆轉歐洲比美國少（分別為 18% 和 34.2%，$P < 0.0001$），而臨床醫生可用的定量監護儀美國(22.7%)比歐洲(70.2%)少($P < 0.0001$)。然而，19.3%的歐洲醫生和 9.4%的美國醫生從不使用神經肌肉監護儀。大多數應答者表示不管是常規的神經肌肉刺激器還是定量四個成串監護儀都應列入最低監測標準。
結論：我們的結果顯示麻醉醫生對監測神經肌肉功能的最佳方法尚沒有統一的結論。我們應當通過發展正式訓練計畫和/或出版關於最佳實踐的官方指南來努力改善蘇醒以減少術後神經肌肉無力的發生率和病人的發病率。
（周潔 譯 馬皓琳 李士通 校）

BACKGROUND: Postoperative residual neuromuscular block is a frequent occurrence. Recent surveys of clinical practice in Europe suggest that neuromuscular blocking drugs are often administered without appropriate monitoring. No comparable survey has been undertaken in the United States (US). From this survey, we compared current clinical neuromuscular practice and attitudes between anesthesia practitioners in the US and Europe.
METHODS: We conducted an Internet-based survey among anesthesia practitioners in the US and Europe. The Anesthesia Patient Safety Foundation and the European Society of Anaesthesiology e-mailed all of their active members, inviting them to anonymously answer a series of questions on a dedicated Internet Protocol address–sensitive website. The survey was available online for 60 days. The χ^2 test and Fisher's exact test were used to compare clinical survey items between the 2 cohorts.
RESULTS: A total of 2636 completed surveys were received. Most respondents from the US (64.1%) and Europe (52.2%) estimated the incidence of clinically significant postoperative residual neuromuscular weakness to be <1% ($P < 0.0001$). Routine pharmacologic reversal was less common in Europe than in the US (18% vs 34.2%, respectively; $P < 0.0001$), and quantitative monitors were available to fewer clinicians in the US (22.7%) than in Europe (70.2%) ($P < 0.0001$). However, 19.3% of Europeans and 9.4% of Americans never use neuromuscular monitors. Most respondents reported that neither conventional nerve stimulators nor quantitative train-of-four monitors should be part of minimum monitoring standards.

CONCLUSIONS: Our results suggest a lack of agreement among anesthesia providers about the best way to monitor neuromuscular function. Efforts to improve awareness by developing formal training programs and/or publishing official guidelines on best practices to reduce the incidence of postoperative neuromuscular weakness and patient morbidity are warranted.

在低氧血症中通過脈搏心輸出量－血氧測定法檢測高鐵血紅蛋白的準確性

Accuracy of Methemoglobin Detection by Pulse CO-Oximetry During Hypoxia

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背景：雖然高鐵血紅蛋白會造成血氧計值（SpO_2）評估動脈功能氧飽和度（SaO_2）的偏移，但是血中的高鐵血紅蛋白很難通過常規的脈搏血氧測定法來檢測。一種新引進的脈搏心輸出量血氧計(Masimo Rainbow SET® Radical-7 Pulse CO-Oximeter, Masimo Corp., Irvine, CA)附加了非創傷性的監測儀，監測血中微量的碳氧血紅蛋白和高鐵血紅蛋白的成分。我們的研究目的是監測低氧是否會影響該設備監測高鐵血紅蛋白讀取的準確性，以及是否高鐵血紅蛋白的存在會有損 Radical-7 和常規的脈搏血氧計(Nonin 9700, Nonin Medical Inc., Plymouth, MN)發現 SaO_2 下降的能力。
方法：兩個研究組分別包括 8 名和 6 名健康的成年人，每個人安置多個感測器和橈動脈導管供血液採樣。第一組，靜脈給予將近 300mg 的亞硝酸鈉來增加高鐵血紅蛋白的水準，目標為 7%－8%，通過吸入氧濃度的不同造成 SaO_2(70%–100%)水準不同的低氧。第二組，目標為室內空氣中高鐵血紅蛋白 15%以及 $SaO_2$80%水準。脈搏心輸出量血氧計讀數與多波長輻射血氧計測定的動脈血數值進行比較。通過觀察在不同缺氧水準上有意義的讀取錯誤發生率來分析脈搏心輸出量血氧計對高鐵血紅蛋白的讀取表現。這是用來確定影響測定高鐵血紅蛋白的預測價值的。在高鐵血紅蛋白升高的情況下，評價 SaO_2 讀數的偏移、精密度和均方根誤差。
結果：在 2 組中，觀察範圍 SaO_2 為 66.2%－99% 和高鐵血紅蛋白為 0.6%－14.4%(170 次抽血)。在全部 SaO_2 範圍內，Masimo 高鐵血紅蛋白讀數偏差和精確度是 7.7% ± 13.0%。SaO_2 範圍在 95%－100%時最準確(1.9% ± 2.5%)，發展到 70%－80%的範圍時最不精確(24.8% ± 15.6%)。SaO_2 每下降 5 個點時，高鐵血紅蛋白讀數錯誤的發生率增高>5%($P < 0.05$)。Masimo 的 SpO_2 讀數在 SaO_2 範圍為 95%－100%且高鐵血紅蛋白範圍 4%－8.3%時偏差了−6.3% ± 3.0%。在 SaO_2 <90%和高鐵血紅蛋白 4%－15%時，Radical-7 和 Nonin 9700 脈搏血氧計都精確地檢測到了下降，但是 SaO_2 >95%時也會顯示低的 SpO_2 讀數。
結論：當 SaO_2 下降 <95%的時候，Radical-7 的高鐵血紅蛋白讀數逐漸越來越不準確，有時候會高估實際值 10%－40%。升高的高鐵血紅蛋白會使 SpO_2 讀數低估了 SaO_2，近似于高飽和度時的普通 2 波長脈搏血氧計。當發生低氧血症(SaO_2 <90%)且高鐵血紅蛋白水準高達 15%的時候，兩種儀器的 SpO_2 讀數都趨向於下降。
（唐亮 譯 馬皓琳 李士通 校）

BACKGROUND: Methemoglobin in the blood cannot be detected by conventional pulse oximetry, although it can bias the oximeter's estimate (SpO_2) of the true arterial functional oxygen saturation (SaO_2). A recently introduced “Pulse CO-Oximeter” (Masimo Rainbow SET® Radical-7 Pulse CO-Oximeter, Masimo Corp., Irvine, CA) is intended to additionally monitor noninvasively the fractional carboxyhemoglobin and methemoglobin content in blood. The purpose of our study was to determine whether hypoxia affects the new device's estimated methemoglobin reading accuracy, and whether the presence of methemoglobin impairs the ability of the Radical-7 and a conventional pulse oximeter (Nonin 9700, Nonin Medical Inc., Plymouth, MN) to detect decreases in SaO_2.

METHODS: Eight and 6 healthy adults were included in 2 study groups, respectively, each fitted with multiple sensors and a radial arterial catheter for blood sampling. In the first group, IV administration of approximately 300 mg sodium nitrite increased subjects' methemoglobin level to a 7% to 8% target and hypoxia was induced to different levels of SaO_2 (70%–100%) by varying fractional inspired oxygen. In the second group, 15% methemoglobin at room air and 80% SaO_2 were targeted. Pulse CO-oximeter readings were compared with arterial blood values measured using a Radiometer multiwavelength hemoximeter. Pulse CO-oximeter methemoglobin reading performance was analyzed by observing the incidence of meaningful reading errors at the various hypoxia levels. This was used to determine the impact on predictive values for detecting methemoglobinemia. SpO_2 reading bias, precision, and root mean square error were evaluated during conditions of elevated methemoglobin.

RESULTS: Observations spanned 66.2% to 99% SaO_2 and 0.6% to 14.4% methemoglobin over the 2 groups (170 blood draws). Masimo methemoglobin reading bias and precision over the full SaO_2 span was 7.7% ± 13.0%. Best accuracy was found in the 95% to 100% SaO_2 range (1.9% ± 2.5%), progressing to its worst in the 70% to 80% range (24.8% ± 15.6%). Occurrence of methemoglobin readings in error >5% increased over each 5-point decrease in SaO_2 ($P < 0.05$). Masimo SpO_2 readings were biased −6.3% ± 3.0% in the 95% to 100% SaO_2 range with 4% to 8.3% methemoglobin. Both the Radical-7 and Nonin 9700 pulse oximeters accurately detected decreases in SaO_2 <90% with 4% to 15% methemoglobin, despite displaying low SpO_2 readings when SaO_2 was >95%.

CONCLUSIONS: The Radical-7's methemoglobin readings become progressively more inaccurate as SaO_2 decreases <95%, at times overestimating true values by 10% to 40%. Elevated methemoglobin causes the SpO_2 readings to underestimate SaO_2 similar to conventional 2-wavelength pulse oximeters at high saturation. SpO_2 readings from both types of instruments continue to trend downward during the development of hypoxemia (SaO_2 <90%) with methemoglobin levels up to 15%.

肺泡而非靜脈右旋氯胺酮抑制大鼠肺泡鈉轉運和肺液體清除率

Alveolar but Not Intravenous S-Ketamine Inhibits Alveolar Sodium Transport and Lung Fluid Clearance in Rats

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背景：右旋氯胺酮(S-ketamine)常用于鎮痛鎮靜，特別是敗血症和心血管不穩定情況下。由於右旋氯胺酮阻斷神經元和骨骼肌電壓門控鈉離子(Na+)通道，因此可以推測右旋氯胺酮也能阻斷決定肺泡液體清除率(AFC)的肺泡上皮鈉通道。我們研究了經肺泡和經靜脈給予右旋氯胺酮對跨肺泡膜 Na+轉運和 AFC 的影響，並觀察右旋氯胺酮靜脈注射是否會因為內毒素血症引起的肺炎而進入肺泡腔。
方法：在培養的大鼠肺泡 II 型 (ATII) 細胞中加入右旋氯胺酮和/或 Na+通道阻滯劑阿米洛利(100 μM)，並在體外擴散池(Ussing chambers)中通過測量短路電流（ISC）反映跨上皮轉運率。在採用含有或不含有阿米洛利的灌流液緩慢灌注的麻醉大鼠肺中測量肺泡液體清除率(AFC)。右旋氯胺酮靜脈注射或加入灌注液中。採用靜脈注射脂多糖(7.5 mg/kg)誘導內毒素血症所致輕度肺損傷模型，該模型或許使靜脈注射的右旋氯胺酮易於到達肺泡表面。
結果：不管肺頂端(−18.9%± 1.4%; $P < 0.001$) 還是底外側(−20.4% ± 3.7%; $P < 0.001$) 給藥，右旋氯胺酮(25 μg/mL)均能降低 ATII 細胞的 ISC。對於阿米洛利預處理的 ATII 細胞，頂端或者底外側給予右旋氯胺酮均不能降低 ISC。對照組大鼠每 30 min 的 AFC 大約為 8%。灌注液中的右旋氯胺酮(5 μg/mL)則通過降低阿米洛利敏感性跨上皮鈉轉運，而使得 AFC 降低至 1.1% ± 1.5% ($P = 0.04$)。靜脈注射右旋氯胺酮(20 mg/kg)不影響 AFC($P = 0.31$)。在脂多糖誘導的炎症情況下，靜脈注射右旋氯胺酮後支氣管肺泡灌洗液中的右旋氯胺酮濃度仍低於抑制 AFC 的濃度。
結論：儘管右旋氯胺酮處理大鼠肺泡上皮降低了阿米洛利敏感性跨肺泡的 Na+轉運和 AFC，但是，即使在輕度肺損傷的情況下，靜脈注射臨床劑量右旋氯胺酮也不影響 AFC。
(江繼宏 譯 馬皓琳 李士通 校）

BACKGROUND: S-ketamine is frequently used for analgosedation, especially during sepsis and cardiovascular instability. Because S-ketamine blocks voltage-gated sodium (Na^+) channels in neurons and skeletal muscle, it is conceivable that S-ketamine also blocks alveolar epithelial Na^+ channels that are crucial for alveolar fluid clearance (AFC). We studied the effects of alveolar and IV S-ketamine on transalveolar Na^+ transport and AFC, and investigated whether IV S-ketamine enters the alveolar space in response to endotoxemia-induced pulmonary inflammation.
METHODS: Cultured rat alveolar type II (ATII) cells were exposed to S-ketamine and/or the Na^+ channel blocker amiloride (100 μM) and transepithelial transport indicated by short circuit current (ISC) was measured in Ussing chambers. AFC was measured in fluid-instilled lungs of anesthetized rats with or without amiloride added to the instillate. S-ketamine was either added to the instillate or injected IV. To induce mild lung injury

that might favor the appearance of IV S-ketamine at the alveolar surface, endotoxemia was induced by IV lipopolysaccharide (7.5 mg/kg).
RESULTS: In ATII cells, S-ketamine (25 μg/mL) caused a decrease of ISC regardless of apical (−18.9%± 1.4%; $P < 0.001$) or basolateral (−20.4% ± 3.7%; $P < 0.001$) application. In ATII cells pretreated with amiloride, addition of apical or basolateral S-ketamine did not decrease ISC. AFC was approximately 8% per 30 minutes in control rats. S-ketamine (5 μg/mL) in the instillate reduced AFC to 1.1% ± 1.5% ($P = 0.04$) by decreasing amiloride-sensitive transepithelial Na^+ transport. Intravenous S-ketamine (20 mg/kg) did not affect AFC ($P = 0.31$). In the presence of lipopolysaccharide-induced inflammation, the concentration of IV-injected S-ketamine in bronchoalveolar lavage fluid remained below the concentration that inhibited AFC.
CONCLUSIONS: Although exposure of the rat alveolar epithelium to S-ketamine decreases amiloride-sensitive transalveolar Na^+ transport and AFC, IV S-ketamine at clinically relevant bolus concentrations does not affect AFC, even in the presence of mild lung injury.

患有惡性高熱和中央軸空病的瑞典患者中 RYR1 突變基因的功能特性

Functional Properties of RYR1 Mutations Identified in Swedish Patients with Malignant Hyperthermia and Central Core Disease

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背景：經體外收縮測試得到的惡性高熱易感性診斷往往只是在遠離患者生活的專科實驗室裏完成的。因此，我們設計了一個方案用於得到 RYR1-cDNA 的基因篩選和從當地初級醫療中心收集到外周血標本分離的 B 淋巴細胞中新確定的蘭尼城受體-1（RYR1）基因變種的功能性測試。
方法：分離 B 淋巴細胞用於提取 RYR1-mRNA 和基因組 DNA，並用於在 5 名攜帶未分類 RYR1 突變的患者體內建立類 B 淋巴母細胞系。用類 B 淋巴母細胞系來研究靜態胞質鈣濃度、來自胞質網狀組織 Ca-ATP 酶抑制劑毒胡蘿蔔素誘發的鈣瞬變高峰以及蘭尼城受體激動劑 4-氯間甲酚誘發的劑量依賴性的鈣釋放。
結果：通過提取 mRNA 用於合成 cDNA,以及在所有的標本中建立 B 淋巴細胞株都是可能的。和對照組相比，所有攜帶 RYR1 潛在突變基因的 B 淋巴細胞系與對照相比較，都顯示了靜息期胞漿鈣濃度的明顯增高及引起鈣釋放的 4-氯間甲酚濃度的下降。
結論：常溫下通過長途平信運送的用於提取 DNA 和 RNA 以及建立 B 類 B 淋巴母細胞系的外周血標本是穩定的。隱藏有新確定的氨基酸取代基的 B 細胞功能的測試表明，他們改變細胞內鈣離子的內穩態，並且是惡性高熱發生的最可能因素。
（楊秀娟 譯 馬皓琳 李士通校）

BACKGROUND: A diagnosis of malignant hyperthermia susceptibility by in vitro contraction testing can often only be performed at specialized laboratories far away from where patients live. Therefore, we have designed a protocol for genetic screening of the RYR1-cDNA and for functional testing of newly identified ryanodine receptor 1 (*RYR1*) gene variants in B lymphocytes isolated from peripheral blood samples drawn at local primary care centers.
METHODS: B lymphocytes were isolated for the extraction of RYR1-mRNA and genomic DNA and for establishment of lymphoblastoid B cell lines in 5 patients carrying yet unclassified mutations in the *RYR1*. The B lymphoblastoid cell lines were used to study resting cytoplasmic calcium concentration, the peak calcium transient induced by the sarco(endo)plasmic reticulum Ca-ATPase inhibitor thapsigargin, and the dose-dependent calcium release induced by the ryanodine receptor agonist 4-chloro-*m*-cresol.
RESULTS: It was possible to extract mRNA for cDNA synthesis and to create B lymphocyte clones from all samples. All B lymphoblastoid cell lines carrying *RYR1* candidate mutations showed significantly increased resting cytoplasmic calcium levels as well as a shift to lower concentrations of 4-chloro-*m*-cresol inducing calcium release compared with controls.
CONCLUSIONS: Peripheral blood samples are stable regarding RNA and DNA extraction and establishment of lymphoblastoid B cell lines after transportation at ambient temperature over large distances by ordinary mail. Functional tests on B cells harboring the newly identified amino acid substitutions indicate that they alter intracellular Ca^{2+} homeostasis and are most likely causative of malignant hyperthermia.

周樹脂毒聯合抗抑鬱藥優先延長大鼠坐骨神經的感覺/傷害性刺激阻滯

Resiniferatoxin Combined with Antidepressants Preferentially Prolongs Sensory/Nociceptive Block in Rat Sciatic Nerve

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背景：當前外周神經阻滯技術具有較大的限制，包括運動纖維和感覺纖維之間缺乏分化以及局麻藥的潛在毒性。最近的研究提示通過一個短暫受體潛在的類香草精 1 型活化劑（辣椒辣素）以及局麻藥得到感受傷害選擇性神經阻滯。我們假設結合強效短暫的受體潛在的類香草精 1 型活化劑周樹脂毒（RTX）以及選擇性抗抑鬱藥（阿米替林、多慮平、氟西汀和強效鈉離子通道阻滯劑）可以延長並優先感覺神經阻滯。
方法：異氟醚麻醉大鼠，0.2ml 阿米替林、多慮平或者氟西汀注射於僅靠外科暴露的坐骨神經（每組 *n*=8）。一些動物接受包括 RTX 的第二次注射（每組 *n*=8）。

通過運動功能的神經行為學測試（伸肌突伸）以及疼痛反射（機械性回縮）評價神經阻滯的效果。
結果：單純運用 RTX 產生疼痛選擇性坐骨神經阻滯，而抗抑鬱藥物產生感受傷害以及運動阻滯。聯合運用 RTX 以及抗抑鬱藥可得到主要為疼痛神經的阻滯。對比單用抗抑鬱藥或 RTX，聯合使用可延長疼痛神經阻滯並優於運動阻滯。
討論：聯合運用 RTX 以及抗抑鬱藥物較各自單用可顯著延長大鼠坐骨神經中的外周疼痛阻滯。然而，兩種藥物給藥法也誘出延長運動功能的阻滯，但其效果小於疼痛感覺阻滯，提示了存在非短暫的受體潛在的類香草精 1 型活化劑的機制。RTX 影響疼痛信號傳導/傳遞的機制並沒有得到完全解釋。
（龔寅 譯 馬皓琳、李士通 校 ）

BACKGROUND: Current techniques of peripheral nerve block have major limitations, including lack of differentiation between motor and sensory fibers and potential toxicity of local anesthetics. Recent studies have suggested that a nociceptive-selective nerve block can be achieved via a transient receptor potential vanilloid type 1 activator (capsaicin) along with local anesthetics. We hypothesized that the combination of potent transient receptor potential vanilloid type 1 agonist resiniferatoxin (RTX) and selected antidepressants (amitriptyline, doxepin, and fluoxetine, also potent sodium channel blockers) would produce prolonged and predominantly sensory nerve block.
METHODS: Rats were anesthetized with isoflurane, and 0.2 mL of amitriptyline, doxepin, or fluoxetine was deposited next to the surgically exposed sciatic nerves (n = 8 per group). Some animals received a second injection containing RTX (n = 8 per group). The effect of nerve block was assessed by neurobehavioral tests of the motor function (extensor postural thrust) and the nocifensive reaction (mechanical pinch).
RESULTS: A single application of RTX produced nociceptive-selective sciatic nerve block, whereas antidepressants produced nociceptive and motor block. The combined administration of RTX and antidepressant resulted in a predominantly nociceptive nerve block. Compared with antidepressants or RTX alone, the combination prolonged the nociceptive nerve block more than the motor block.
CONCLUSIONS: The combined application of RTX and antidepressants produced a markedly prolonged nociceptive peripheral nerve block in rat sciatic nerves compared with either agent alone. However, the 2-drug regimen also elicited prolonged blockade of the motor function, although disproportionately less compared with the nociceptive modality, suggesting the existence of nontransient receptor potential vanilloid type 1–mediated mechanisms. The mechanisms through which RTX affects nociceptive signal transduction/transmission have yet to be fully elucidated.

多點注射腋路臂叢阻滯：肥胖對失敗率和急性併發症發生率的影響

Multiple Injection Axillary Brachial Plexus Block: Influence of Obesity on Failure Rate and Incidence of Acute Complications

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背景：在區域麻醉中，肥胖常常伴隨著更高的失敗率，但是目前還沒有人評估過特殊的阻滯方法在肥胖患者中的應用效果。我們假設肥胖降低了腋路臂叢阻滯的成功率。

方法：我們進行了一項前瞻性的研究，由經驗豐富的麻醉醫生對擇期行上肢手術的患者實施腋路臂叢阻滯。採用三點注射法，分別用 0.5%羅呱卡因 6mL、10mL 和 20mL 阻滯肌皮神經、正中神經和橈神經。通過末梢運動反應（腕關節或手指）判斷正中神經和繞神經的阻滯效果。無需輔助用藥而手術能順利進行即為麻醉成功。記錄麻醉急性併發症。離開麻醉後監護室前，記錄患者對麻醉的滿意度。

結果：在 605 例患者中，有 85 例為肥胖患者（BMI≥30kg/m^2）。總體的成功率為 97%，其中，肥胖患者成功率為 91%，非肥胖患者為 98%（P = 0.003）。肥胖患者中（7%）需要肘部輔助神經阻滯的人數比非肥胖患者多（2%；P = 0.007）。肥胖患者（27%）急性併發症（主要是穿破血管）的發生率高於非肥胖患者（27%比 9%；P < 0.001）。肥胖患者對麻醉的滿意度為 87%，而非肥胖患者為 94%（P = 0.03）。

結論：肥胖增加了腋路臂叢阻滯的失敗率和急性併發症的發生率。此外，更多的肥胖患者對麻醉效果不滿意。

（徐妍君 譯　馬皓琳 李士通 校）

BACKGROUND: Obesity has been associated with an increased failure rate in regional anesthesia, but specific block techniques have not been evaluated. We hypothesized that obesity decreases the success rate of axillary brachial plexus block.

METHODS: We prospectively studied axillary brachial plexus blocks performed by experienced anesthesiologists in patients scheduled for upper limb surgery. A triple-injection technique was given to block the musculocutaneous and the median nerves with 6 mL and 10 mL ropivacaine 0.5%, respectively, and the radial nerve with 20 mL ropivacaine 0.5%. For the median and radial nerves, distal motor responses (wrist or fingers) were identified. Success was defined as adequate anesthesia allowing surgery to be performed without additional medications. Acute complications were noted. Before leaving the postanesthesia care unit, patient's satisfaction with anesthesia was collected.

RESULTS: Of 605 patients, 85 were obese (body mass index ≥30 kg/m^2). The success rate was 97% overall, 91% in the obese and 98% in the non-obese patients (P = 0.003). Additional nerve blocks at the elbow were performed more frequently in obese (7%) than in non-obese patients (2%; P = 0.007). Acute complications (mainly vascular puncture) were more frequent in obese than in non-obese patients (27% vs 9%; P < 0.001). Patient satisfaction was 87% in the obese and 94% in the non-obese patients (P = 0.03).

CONCLUSIONS: Obesity increased the failure rate and immediate complications of axillary brachial plexus block. Furthermore, more obese patients were dissatisfied with their anesthesia.

中樞阿片受體啟動介導心臟缺血再灌注損傷的保護作用

Activation of Central Opioid Receptors Induces Cardioprotection Against Ischemia-Reperfusion Injury

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背景：鞘內注射小劑量嗎啡可以產生類似於靜脈注射嗎啡合併缺血預處理的心臟保護作用。作者研究了鞘內注射嗎啡預處理（ITMPC）時中樞和外周阿片受體的相對作用。
方法：48 只開胸後麻醉中的 SD 雄性大鼠，成功放置鞘內導管後，被分配到 7 個治療組中的一組（每組 6-7 個）。ITMPC 通過連續 3 次鞘內注射嗎啡預處理 5 分鐘(每次 1.0 μg/kg)獲得。結合靜脈(靜脈注射碘化納絡酮+ ITMPC)或者鞘內(鞘內注射碘化納絡酮+ ITMPC)注射碘化納絡酮，重複應用預處理。另外，在不用鞘內注射嗎啡預處理下，採用相同的方法注射化合物(靜脈+鞘內注射碘化納絡酮)。鞘內注射生理鹽水和缺血預處理分別作為陰性和陽性對照。心肌缺血再灌注損傷通過阻斷左主冠狀動脈 30min 後再灌注 2h 獲得。由 2,3,5 -三苯基四唑染色來確定缺血危險區比例，即心肌梗死面積。
結果：與對照組(48% ± 9%)相比，缺血預處理組(22% ± 3%)和鞘內嗎啡預處理組(26% ± 5%)的梗死面積/危險區域(22% ± 3%)明顯降低($P < 0.01$)。此外鞘內注射碘化納絡酮還逆轉了鞘內注射嗎啡預處理的心臟保護作用(45% ± 4%)，而靜脈用藥對鞘內注射嗎啡預處理沒有任何影響(28% ± 9%, $P < 0.01$)。
結論：鞘內注射嗎啡預處理可以通過啟動中樞阿片受體來產生心臟保護作用，外周阿片受體沒有明顯的作用。
（黃丹　譯　陳傑　校）

BACKGROUND: Small doses of intrathecal morphine provide cardioprotection similar to that conferred by IV morphine and ischemic preconditioning (IPC). We investigated the relative role of central versus peripheral opioid receptors in intrathecal morphine preconditioning (ITMPC).
METHODS: Forty-eight anesthetized, open-chest, male Sprague-Dawley rats were assigned to 1 of 7 treatment groups ($n = 6–7$) after successful intrathecal catheter placement. ITMPC was achieved by 3 consecutive 5-min intrathecal infusions of morphine (1.0 μg/kg each). This was repeated in the presence of either IV (IV naloxone methiodide + ITMPC) or intrathecally (intrathecal naloxone methiodide [ITNM] + ITMPC) administered naloxone methiodide. This compound was also given via these same routes in the absence of ITMPC (IV naloxone methiodide + ITNM). Intrathecal normal saline and IPC were used as negative and positive controls, respectively. Myocardial ischemia and reperfusion injury were induced by 30 min of left main coronary artery occlusion followed by 2 h of reperfusion. Myocardial infarct size, as a percentage of the area-at-risk, was determined by 2,3,5-triphenyltetrazolium staining.
RESULTS: The infarct size/area-at-risk were significantly reduced in the IPC (22% ± 3%) and ITMPC (26% ± 5%) groups compared with the control group (48% ± 9%) ($P < 0.01$). The addition of ITNM reversed the cardioprotective effects of ITMPC (45% ± 4%), whereas IV administration of the drug did not have any effect on ITMPC (28% ± 9%, $P < 0.01$).

CONCLUSIONS: Intrathecally administered morphine can produce cardioprotective effects via the activation of central opioid receptors, without the apparent involvement of peripheral opioid receptors.

胸腹主動脈手術中腰部腦脊液引流：基本原理和操作指南

Lumbar Cerebrospinal Fluid Drainage for Thoracoabdominal Aortic Surgery: Rationale and Practical Considerations for Management

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截癱仍是胸腹主動脈瘤手術的最嚴重的併發症之一，與併發症發生率和死亡率增加密切相關。現代主動脈修復技術使用各種方法來減少手術相關的脊髓缺血危險。這些方法之一是通過腰部腦脊液（CSF）的引流來優化脊髓血流量。單獨或與其他措施相結合，腦脊液引流仍然是最常用的脊髓保護技術之一。儘管沒有降低脊髓損傷有效性的確切證據，但是有令人信服的資料支援這項技術的運用。然而，腦脊液引流的潛在優點，必須兼顧其風險，包括插入時神經損傷，椎管內壓迫軸索性血腫形成，由於引流過多導致的顱內出血和感染等。可以通過瞭解其使用的基本原理及遵守實踐指南來實現最佳效益風險比。
（陳毓雯　譯　陳傑　校）

Paraplegia remains one of the most devastating 災難性 complications of thoracoabdominal aortic surgery and is associated with a significant increase in both morbidity and mortality. Modern aortic repair techniques use many modalities 方式 aimed at reducing the risk of spinal cord ischemia inherent with surgical management. One of these modalities that acts via optimizing spinal cord blood flow is lumbar cerebrospinal fluid (CSF) drainage. Either alone or in combination with other interventions, CSF drainage remains one of the most frequently used spinal cord protection techniques. Despite no definitive proof of efficacy for reducing spinal cord injury, there are compelling 有說服力的 data supporting its use. However, the potential benefit of CSF drainage must be balanced against the risks associated with its use, including nerve injury during insertion, compressive neuraxial hematoma formation, intracranial hemorrhage due to excessive drainage, and infection. The optimal benefit to risk ratio can be achieved by understanding the rationale for its use and following practical management guidelines.

吸入麻醉藥的潛在全球變暖效應：臨床應用

Global Warming Potential of Inhaled Anesthetics: Application to Clinical Use

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背景：吸入麻醉藥是公認的溫室氣體。本文通過計算其在常見的臨床使用過程中的相當的二氧化碳總排放量來比較不同的吸入麻醉藥對環境的影響。
方法：作者先確定七氟醚和異氟醚紅外吸收截面積。運用以前公佈的地氟醚，七氟醚，異氟醚的紅外線吸收資料計算 20 年全球變暖潛能值（GWP20），且確定大氣中各氣體最適存在週期。在每小時最小肺泡濃度（MAC）下使用每種麻醉劑的總量乘以計算出的 GWP_{20}，表示為每克“二氧化碳當量”（CDE20）。計算時根據目前臨床吸入麻醉的方法，將分別計算由空氣/氧氣作為載體或 N_2O/氧氣的混合氣體作為載體時二氧化碳當量數值。
結果：對吸入麻醉藥 GWP20 計算值分別為：七氟醚 349，異氟醚 1401，地氟醚 3714。2 L/h 新鮮氣流量下 1MAC 每小時的 CDE_{20} 的預測值分別為：七氟醚 6980 g，異氟醚 15,551 g，地氟醚 187186g。這些麻醉藥之間的比率為七氟醚 1，異氟醚 2.2，地氟醚 26.8。當 60%N_2O 與 40%氧氣的混合氣體替代空氣/氧氣作為載體，同時吸入麻醉藥調整為每小時 1MAC，七氟醚 CDE20 值分別高出 5.9 倍，異氟醚 CDE20 值高出 2.9 倍，而地氟醚 CDE20 值降低，為 0.4 倍。以 100 年為時間水準運用 60%N_2O 作為載體時，七氟醚 CDE_{100} 值比空氣/氧氣作為載體高出 19 倍，異氟醚的值高出 9 倍，地氟醚的值無差異。
結論：根據比較結果和臨床環境，地氟醚對全球變暖的影響超過七氟醚與異氟醚。應用七氟醚或異氟醚，將 N_2O 作為載體將產生更多的溫室氣體。此外，60%氧化亞氮與吸入麻醉劑混合使用，在相同 MAC 的麻醉下大幅度增加七氟醚和異氟醚對環境的影響，而地氟醚對環境的影響降低。氧化亞氮會破壞臭氧層及使全球升溫趨勢；且影響的持續時間更長，與地氟醚混合使用可能不是一個環保的折衷方案。根據計算研究，避免 N_2O 及不必要的高流量氣體可降低吸入麻醉對環境的影響。
（陳毓雯　譯　陳傑　校）
BACKGROUND: Inhaled anesthetics are recognized greenhouse gases. Calculating their relative impact during common clinical usage will allow comparison to each other and to carbon dioxide emissions in general.
METHODS: We determined infrared absorption cross-sections for sevoflurane and isoflurane. Twenty-year global warming potential (GWP_{20}) values for desflurane, sevoflurane, and isoflurane were then calculated using the present and previously published infrared results, and best estimate atmospheric lifetimes were determined. The total quantity of each anesthetic used in 1 minimal alveolar concentration (MAC)-hour was then multiplied by the calculated GWP_{20} for that anesthetic, and expressed as “carbon dioxide equivalent” (CDE_{20}) in grams. Common fresh gas flows and carrier gases, both air/oxygen and nitrous oxide (N_2O)/oxygen, were considered in the calculations to allow these examples to represent common clinical use of inhaled anesthetics.
RESULTS: GWP_{20} values for the inhaled anesthetics were: sevoflurane 349, isoflurane 1401, and desflurane 3714. CDE_{20} values for 1 MAC-hour at 2 L fresh gas flow were: sevoflurane 6980 g, isoflurane 15,551 g, and desflurane 187,186 g. Comparison among these anesthetics produced a ratio of sevoflurane 1, isoflurane 2.2, and desflurane 26.8. When 60% N_2O/40% oxygen replaced air/oxygen as a carrier gas combination, and inhaled anesthetic delivery was adjusted to deliver 1 MAC-hour of anesthetic,

sevoflurane CDE_{20} values were 5.9 times higher with N_2O than when carried with air/O_2, isoflurane values were 2.9 times higher, and desflurane values were 0.4 times lower. On a 100-year time horizon with 60% N_2O, the sevoflurane CDE_{100} values were 19 times higher than when carried in air/O_2, isoflurane values were 9 times higher, and desflurane values were equal with and without N_2O.
CONCLUSIONS: Under comparable and common clinical conditions, desflurane has a greater potential impact on global warming than either isoflurane or sevoflurane. N_2O alone produces a sizable greenhouse gas contribution relative to sevoflurane or isoflurane. Additionally, 60% N_2O combined with potent inhaled anesthetics to deliver 1 MAC of anesthetic substantially increases the environmental impact of sevoflurane and isoflurane, and decreases that of desflurane. N_2O is destructive to the ozone layer as well as possessing GWP; it continues to have impact over a longer timeframe, and may not be an environmentally sound tradeoff for desflurane. From our calculations, avoiding N_2O and unnecessarily high fresh gas flow rates can reduce the environmental impact of inhaled anesthetics.

殘餘神經阻滯:易忘掉的課業.第一部分: 殘餘神經阻滯的定義，發生率和不良生理學反應

Residual Neuromuscular Block: Lessons Unlearned. Part I: Definitions, Incidence, and Adverse Physiologic Effects of Residual Neuromuscular Block

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. Anesth Analg 2010 111:120-128;

在這篇綜述中,作者總結了殘餘神經阻滯的臨床併發症。資料表明，殘餘神經阻滯在麻醉後監護室是一個常見的併發症,將近 40%的患者出現了一次 TOF<0.9。志願者研究表明，低程度的殘餘麻痹（TOF 0.7–0.9）與咽反射受損，誤吸風險增加，上呼吸道肌無力，氣道阻塞，低氧通氣反應（將近 30%），以及肌肉無力的不愉快症狀相關。臨床研究已經證實術中神經肌肉管理與術後不良事件相關。大型資料庫調查發現，術中神經肌肉阻滯劑的應用和殘餘神經神經肌肉阻滯是麻醉相關發病率和死亡率的重要危險因素。此外，觀察和隨機臨床試驗表明，術後早期不完全的神經肌肉功能的恢復可能導致急性呼吸症狀（低氧血症和氣道阻塞），肌肉無力引起的不愉快症狀，麻醉後監護室停留時間延長，氣管拔管延遲，以及術後肺部併發症風險增加。這些近期的資料表明，殘餘神經肌肉阻滯是一個有關患者安全的重要問題，且神經肌肉阻滯的管理影響手術預後。
（懷曉蓉　譯　陳傑　校）
In this review, we summarize the clinical implications of residual neuromuscular block. Data suggest that residual neuromuscular block is a common complication in the postanesthesia care unit, with approximately 40% of patients exhibiting a train-of-four ratio <0.9. Volunteer studies have demonstrated that small degrees of residual paralysis (train-of-four ratios 0.7–0.9) are associated with impaired pharyngeal function and increased risk of aspiration, weakness of upper airway muscles and airway obstruction, attenuation of the hypoxic ventilatory response (approximately 30%), and unpleasant

symptoms of muscle weakness. Clinical studies have also identified adverse postoperative events associated with intraoperative neuromuscular management. Large databased investigations have identified intraoperative use of muscle relaxants and residual neuromuscular block as important risk factors in anesthetic-related morbidity and mortality. Furthermore, observational and randomized clinical trials have demonstrated that incomplete neuromuscular recovery during the early postoperative period may result in acute respiratory events (hypoxemia and airway obstruction), unpleasant symptoms of muscle weakness, longer postanesthesia care unit stays, delays in tracheal extubation, and an increased risk of postoperative pulmonary complications. These recent data suggest that residual neuromuscular block is an important patient safety issue and that neuromuscular management affects postoperative outcomes.

麻醉誘導期間採用高解析度固體測壓計測定食管上、下括約肌壓力：肥胖和非肥胖病人間的比較

High-Resolution Solid-State Manometry of the Upper and Lower Esophageal Sphincters During Anesthesia Induction: A Comparison Between Obese and Non-Obese Patients

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背景：近幾十年肥胖發生率有了顯著增長。肥胖相關的圍術期胃腸道變化對於麻醉醫師有重要的臨床意義。食管下、上括約肌在防止反流和誤吸中起主要作用。麻醉過程中增加的腹內壓對肥胖病人的食管上下括約肌的影響尚未明確。此研究採用高解析度固體測壓計評估麻醉誘導過程中肥胖和非肥胖病人的食管上括約肌、食管下括約肌和屏障壓（BrP）（食管下端壓力－胃內壓）。
方法：使用高解析度固態食管測壓計研究 28 名 18－72 歲的患者。14 名進行腹腔鏡胃旁路手術的病人 BMI 指數大等於 35 kg/m^2，另 14 名進行腹腔鏡膽囊切除術的病人 BMI 指數小等於 30kg/m^2。
結果：兩組食管上括約肌壓力在麻醉誘導時均有下降。兩組食管下括約肌壓力在麻醉誘導時也均下降，且肥胖組較非肥胖組病人下降更為明顯。兩組屏障壓也下降，且肥胖組較非肥胖組下降更為明顯。但兩組所有時刻屏障壓為正值。
結論：兩組麻醉誘導時食管下括約肌和屏障壓均下降，但肥胖組下降更顯著。儘管屏障壓有明顯下降，但所有病人的壓力值均為正值。
（於章傑　譯　陳傑　校）

BACKGROUND: The prevalence of obesity has increased dramatically in recent decades. The gastrointestinal changes associated with obesity have clinical significance for the anesthesiologist in the perioperative period. The lower esophageal sphincter and the upper esophageal sphincter play a central role in preventing regurgitation and aspiration. The effects of increased intra-abdominal pressure during anesthesia on the lower esophageal sphincter and the upper esophageal sphincter in obese patients are unknown. In the present study we evaluated, with high-resolution solid-state manometry, the upper esophageal sphincter, lower esophageal sphincter, and barrier pressure (BrP)

(lower esophageal pressure – gastric pressure) in obese patients during anesthesia induction and compared them with pressures in non-obese patients.
METHODS: We studied 28 patients, ages 18 to 72 years, 14 with a body mass index ≥35kg/m^2, who were undergoing laparoscopic gastric bypass, and 14 with a body mass index ≤30kg/m^2, who were undergoing laparoscopic cholecystectomy, using high-resolution solid-state manometry.
RESULTS: Upper esophageal sphincter pressure decreased during anesthesia induction in both groups. Lower esophageal sphincter pressure decreased in both groups during anesthesia induction, and it was significantly lower in obese patients than in non-obese patients. The BrP decreased in both groups and was significantly lower in the obese group than in the non-obese group. The BrP remained positive at all times in both groups.
CONCLUSION: Lower esophageal sphincter and BrPs decreased in both obese and non-obese patients during anesthesia induction, but were significantly lower in obese patients. Although the BrP was significantly lower, it remained positive in all patients.

行心肺轉流術的嬰幼兒中活化凝血時間值與肝素濃度測定值的關係

Correlations Between Activated Clotting Time Values and Heparin Concentration Measurements in Young Infants Undergoing Cardiopulmonary Bypass

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背景：在嬰幼兒行心肺轉流術（CPB）時檢測活化凝血時間（ACT）的同時檢測肝素濃度可以給我們肝素的應用提供一個更為精確的指導。然而，肝素的標準實驗室測定法（即抗 Xa 因數濃度測定）需要測定的是血漿而不是全血，且醫生並不能馬上得到其測定結果。另外，應用一種自動魚精蛋白滴定儀器——Hepcon 測試儀（即 Hepcon 完整式凝血測試系統支援; 美國美敦力公司提供, 美國明尼蘇達州明尼阿波利斯市, MN)能在床邊進行全血肝素濃度測試。本次研究的目的為在年齡小於 6 月的行 CPB 的嬰幼兒中,將三種不同商家的儀器測試出的 ACT 及床邊運用 Hepcon 測試出的全血肝素濃度和實驗室抗 Xa 因數血漿肝素濃度測定法的結果進行比較。
方法：本次研究的入組患者為年齡小於 6 月擇期行 CPB 下心臟手術的嬰幼兒 44 例。抽取注射肝素首次劑量後 3 分鐘以及 CPB 停機前即刻的血樣作為肝素抗凝指標的檢測樣本。高嶺土-啟動活化凝血時間 ACTs 應用 Hemochron 測定儀 (由美國國際診斷技術公司提供, Edison, NJ), Hepcon 測定儀, 及 i-STAT 測定儀(由 i-STAT 公司提供, 東溫莎, NJ)三種儀器進行測試。全血肝素濃度應用 Hepcon 測定儀進行測試。血漿肝素濃度應用實驗室抗 Xa 因數發色底物法測定。
結果：在肝素首次劑量後即刻的測量值中，三組 ACT 值均與血漿肝素濃度不相關。在 CPB 停機前即刻，只有 i-STAT 儀器測定的 ACT 值與其有一定相關性。相

反，在兩個測試時間點，床邊測定全血肝素濃度值與實驗室血漿肝素濃度值相當一致（一致性相關係數分別為 0.30 和 0.67）。通過抗 Xa 因數測定的血漿肝素濃度值比 Hepcon 測定儀測定的全血肝素濃度值要高一些。
結論：行 CPB 的年齡小於 6 月的嬰幼兒中，單應用 ACT 值作為唯一評判肝素抗凝的指征應尤為謹慎。通常，ACT 與血漿肝素濃度相關性並不大。只有應用 i-STAT 儀器進行 ACT 測試時在停機前即刻 ACT 與血漿肝素有一定相關性。而應用 Hepcon 儀器進行床邊全血肝素濃度測定與應用抗 Xa 因數測定值基本相一致。研究資料表明了臨床上在嬰幼兒中床邊進行肝素濃度測試及時、便捷、精確。
（趙嫣紅 譯 陳傑　校）
BACKGROUND: Monitoring heparin concentration along with the activated clotting time (ACT) may provide a more accurate guide for the administration of heparin to infants during cardiopulmonary bypass (CPB). However, standard laboratory assays of heparin concentration (antifactor Xa heparin concentration) require plasma instead of whole blood, and results are not immediately available to clinicians. Alternatively, measurements of whole blood heparin concentration may be performed at the bedside using an automated protamine titration device, the Hepcon instrument (Hepcon Hemostasis Management System Plus; Medtronics, Minneapolis, MN). The purpose of this investigation was to compare ACT measurements from 3 commercially available instruments and bedside measurements of whole blood heparin concentration using the Hepcon instrument with laboratory measurements of antifactor Xa plasma heparin concentration in infants younger than 6 months of age undergoing CPB.
METHODS: Forty-four pediatric patients younger than 6 months of age scheduled for elective cardiac surgery requiring CPB were enrolled in this prospective study. Blood samples were drawn 3 minutes after the initial heparin bolus and immediately before the termination of CPB to obtain measurements of heparin anticoagulation. Kaolin-activated ACTs were performed with the Hemochron (International Technidyne Corporation, Edison, NJ), Hepcon, and i-STAT (i-STAT Corporation, East Windsor, NJ) instruments. Whole blood heparin concentration was measured using the Hepcon instrument. Plasma heparin concentration was measured using an antifactor Xa chromogenic substrate assay 發色底物法
RESULTS: Immediately after the initial heparin bolus, none of the ACT values correlated with plasma heparin concentration. When measured immediately before the termination of CPB, only the i-STAT ACT showed a moderate correlation. Conversely, bedside measurements of whole blood heparin concentration showed satisfactory agreement with laboratory measurements of plasma heparin concentration at both time points (concordance correlation coefficients 0.30 and 0.67, respectively). There is a bias in that antifactor Xa-measured plasma heparin concentration tends to be higher than Hepcon-measured whole blood heparin concentration.
CONCLUSIONS: In infants younger than 6 months old undergoing CPB, caution is warranted 恰當 when using ACT values as the sole 單獨的 indication of adequate heparin anticoagulation. In general, ACT prolongation correlates poorly with plasma heparin concentration. Only i-STAT ACT values showed a moderate correlation when measured immediately before the termination of CPB. Alternatively, bedside measurements of whole blood heparin concentration measured by the Hepcon instrument agreed well with antifactor Xa laboratory measurements. Our data support the clinical utility of bedside

measurements of heparin concentration to provide timely, convenient, and accurate measurements of heparin concentration in these infants.

有與沒有顱內壓監測的幼豬大腦的無創自我調節功能監測

Noninvasive Autoregulation Monitoring with and without Intracranial Pressure in the Naïve Piglet Brain

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背景：對於那些沒有有創顱內壓監測的危重病人，其腦血管的自我調節功能的監測是很有必要的。沒有顱內壓監測，則動脈血壓可以代替腦灌注壓來測量各動脈壓力跨度範圍內腦血流的限制情況。本研究比較了低血壓模型中使用動脈壓和腦灌注壓對於自我調節功能監測的差異。

方法：給予試驗幼豬(5-7 天齡) 致命性的低血壓來確定新生大腦的顱內壓，並判斷其自我調節的最低限度(LLA)。試驗共有 25 只幼豬，連續監測顱內壓、局部腦血氧飽和度（rSO2）、腦皮層紅細胞流量（多普勒）。之後用兩種方法結合腦氧合指數（Cox）來評價自我調節功能：動脈血壓和局部腦血氧飽和度（COX_{ABP}）間的線性關係以及腦灌注壓和局部腦血氧飽和度（COX_{CPP}）間的線性關係。從紅細胞流量對動脈血壓的圖表中可以判斷自我調節的最低限度。本試驗以 COX_{ABP} 和 COX_{CPP} 的平均值為基點，以 5mmHg 作為最小間距，繪製這兩種方法的受試者操作特徵曲線。

結果：為了確定自我調節的最低限度, COX_{ABP} 和 COX_{CPP} 兩種方法得出相同的受試者操作特徵曲線區域為 0.91（95%CI：0.88-0.95）。但兩種方法的閾值不一樣：判定動脈血壓低於自我調節的最低限度時，其 COX_{ABP} 的閾值是 0.5，敏感度 89%（95%CI，81%-94%），特異度 81%（95%CI，73-88%）。而對於 COX_{CPP} 來說，其閾值是 0.42，敏感度同樣是 89%（95%CI，81%-94%），而特異度為 77%（95%CI，69-84%）。

討論：對新生大腦使用動脈血壓而不是腦灌注壓進行自我調節監測，同時結合 Cox 值，用來區分動脈壓是高於還是低於自我調節的最低限度，這種方法的閾值有所升高。然而兩種方法的準確性相似。這些發現說明了對於沒有顱內壓監測的病人可以用近紅外光譜來監測自我調節功能。

（張婷　譯　陳傑　校）

BACKGROUND: Cerebrovascular autoregulation monitoring is often desirable for critically ill patients in whom intracranial pressure (ICP) is not measured directly. Without ICP, arterial blood pressure (ABP) is a substitute for cerebral perfusion pressure (CPP) to gauge the constraint of cerebral blood flow across pressure changes. We compared the use of ABP versus CPP to measure autoregulation in a piglet model of arterial hypotension.

METHODS: Our database of neonatal piglet (5–7 days old) experiments was queried for animals with naïve ICP that were made lethally hypotensive to determine the lower limit of autoregulation (LLA). Twenty-five piglets were identified, each with continuous recordings of ICP, regional cerebral oximetry (rSO_2), and cortical red cell flux (laser Doppler). Autoregulation was assessed with the cerebral oximetry index(COx) in 2 ways: linear correlation between ABP and rSO_2 (COx_{ABP}) and between CPP and rSO_2 (COx_{CPP}). The lower limits of autoregulation were determined from plots 作圖 of red cell flux versus ABP. Averaged values of COx_{ABP} and COx_{CPP} from 5 mm Hg ABP bins 箱? were used to show receiver operating characteristics for the 2 methods.
RESULTS: COx_{ABP} and COx_{CPP} yielded 生產 identical receiver operating characteristic curve areas of 0.91 (95% confidence interval [CI], 0.88–0.95) for determining the LLA. However, the thresholds for the 2 methods differed: a threshold COx_{ABP} of 0.5 was 89% sensitive (95% CI, 81%–94%) and 81% specific (95% CI, 73%–88%) for detecting ABP below the LLA. A threshold COx_{CPP} of 0.42 gave the same 89% sensitivity (95% CI, 81%–94%) with 77% specificity (95% CI, 69%–84%).
CONCLUSIONS: The use of ABP instead of CPP for autoregulation monitoring in the naïve brain with COx results in a higher threshold value to discriminate ABP above from ABP below the LLA. However, accuracy was similar with the 2 methods. These findings support and refine the use of near-infrared spectroscopy 近紅外光譜 to monitor autoregulation in patients without ICP monitors.

齧齒類動物模型中羅呱卡因外周神經注射傷

Ropivacaine-Induced Peripheral Nerve Injection Injury in the Rodent Model

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背景：神經內注射局麻藥總是與神經損傷相關。作者本研究的目的是探索羅呱卡因神經內注射所引起的大鼠坐骨神經的組織學改變。
方法：54 只雄性成年 Lewis 大鼠隨機分成 9 組，每組 6 只。將 50 微升生理鹽水或 10%苯酚或 0.75%羅呱卡因分別進行神經束內、神經束外或者神經外（局部）注射。2 周後將動物處死，分別用光學顯微鏡、定量組織學檢查和電子顯微鏡評估注射部位的坐骨神經。
結果：橫斷面評估發現，神經束外及神經外注射羅呱卡因均可引起神經束膜的損傷，並伴隨著神經組織中心脫髓鞘，外周環繞著水腫的神經內膜。神經束內注射羅呱卡因引起的脫髓鞘改變呈契形，中央軸突消失，並伴有一些再生組織，邊緣是正常有髓鞘區域，它們的周圍是水腫的神經內膜。定量組織學檢查顯示，神經束外注射引起的神經損傷較神經外注射嚴重得多，但仍輕於神經束內注射者。從數量上來說，注射羅呱卡因的標本較注射生理鹽水的標本其神經密度低得多。從電鏡下還可以發現神經組織呈華勒樣變性，以及神經周圍水腫。

結論：本次研究表明，向大鼠模型以神經束內或神經束外的方式注射羅呱卡因後，可引起嚴重的組織學異常，包括神經束膜水腫和華勒樣變性後發生的軸突的破壞。神經束外和神經外注射引起的損傷是相似的，儘管比神經束內注射的組織學損傷輕微。如果本次研究中觀察到的組織學異常真實存在，那麼將來還需進一步的工作以研究其對功能的影響。

（周姝婧　譯　陳傑　校）

BACKGROUND: Intraneural administration of local anesthetics has been associated with nerve damage. We undertook the present study to investigate histological changes induced by ropivacaine injection into rat sciatic nerve.

METHODS: Fifty-four adult male Lewis rats were randomly distributed into 9 groups, 6 animals per group. Fifty microliters of normal saline, 10% phenol, or 0.75% ropivacaine were administered by intrafascicular injection, extrafascicular injection, or extraneural (topical) placement. At 2 weeks, animals were killed and the sciatic nerve at the injection site was evaluated with light microscopy, quantitative histomorphometry, and electron microscopy.

RESULTS: On cross-sectional evaluation, extrafascicular ropivacaine injection and extraneural placement of ropivacaine were both associated with damage to the perineurium, with focal demyelination surrounded by edematous endoneurium. Intrafascicular injection of ropivacaine resulted in a wedge-shaped region of demyelination and focal axonal loss with some regeneration, bordered by a region of normally myelinated axons in a background of edematous endoneurium. Extrafascicular injection resulted in more significant damage than extraneural placement of ropivacaine, but less than intrafascicular injection as shown with quantitative histomorphometry. Quantitatively, ropivacaine-injured specimens had significantly lower nerve density than saline-injured specimens. Wallerian degeneration and perineural edema were also demonstrated qualitatively with electron microscopy.

CONCLUSIONS: This study demonstrates that, in the rat model, ropivacaine is associated with marked histological abnormality, including edema of the perineurium and axonal destruction with wallerian degeneration, when injected into or extraneurally placed onto a nerve. Extrafascicular injection and extraneural placement were associated with similar, although milder, histological damage than intrafascicular injection. Further work is needed to investigate the functional implications, if any, of the histological abnormalities observed in this study.

頸叢神經阻滯局麻液中輔以芬太尼對局麻時效的影響：一項隨機、對照研究

The Addition of Fentanyl to Local Anesthetics Affects the Quality and Duration of Cervical Plexus Block: A Randomized, Controlled Trial

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背景：頸叢神經阻滯常被認為是非完善的感覺阻滯，在這項隨機，雙盲，對照試驗中，作者比較了頸動脈內膜剝脫術（CEA）中，局麻液中加芬太尼後是否改善頸叢神經阻滯效果。
方法：77 名擇期行 CEA 術的成年患者，行頸深叢神經阻滯，隨機分組：實驗組局麻液中加入芬太尼 1 mL (50 μg)，對照組加入生理鹽水鹽 1 mL，分別與 0.5%布比卡因 10 mL 和 2%利多卡因 4 mL 配置成混合液。同時用 0.5%布比卡因 10 mL 和 2%利多卡因 4 mL 行頸淺叢阻滯。用視覺類比評分評估疼痛(0–10; 0 =無痛, 10 = 疼痛難以忍受), 對於術中疼痛評分大於 3 者給予異丙酚 20mg 靜脈推注。記錄術中所需藥物追加時間以及從術後 24 小時內所需鎮痛情況，P <0.05 認為有統計學意義。
結果：芬太尼組術中追加異丙酚(4 of 38, 10.5%)明顯少於對照組(26 of 39, 66.7%; *P* < 0.001)。儘管兩組的阻滯起效時間無明顯差異(各自的中位數為 12 [9–18] vs 15 [9–18] 分; *P* = 0.18)。 但相比對照組，芬太尼組需要異丙酚的量更少(分別是中位數 0 [0–60] vs 60 [0–160] mg; *P* < 0.001), 需要術後鎮痛的發生率更低(分別是 22 /38 例, 57.9% vs 35 / 39 例, 89.7%,; *P* = 0.002)，術後首次鎮痛的時間更晚 (分別是 中位時間 5.8h [1.9–15.6] 和 3.1 [1.0–11.7] h; *P* < 0.001)。
結論：在 CEA 中，以局部麻醉藥中輔助芬太尼可以改善頸叢阻滯的作用，並延長阻滯時間。
BACKGROUND: Cervical plexus block is frequently associated with unsatisfactory sensory blockade. In this randomized, double-blind, placebo-controlled trial, we examined whether the addition of fentanyl to local anesthetics improves the quality of cervical plexus block in patients undergoing carotid endarterectomy (CEA).
METHODS: Seventy-seven consecutive adult patients scheduled for elective CEA were randomized to receive either fentanyl 1 mL (50 μg) or saline placebo 1 mL in a mixture of 10 mL bupivacaine 0.5% and 4 mL lidocaine 2% for deep cervical plexus block. Superficial cervical plexus block was performed using a mixture of 10 mL bupivacaine 0.5% and 5 mL lidocaine 2%. Pain was assessed using the verbal rating scale (0–10; 0 = no pain, 10 = worst pain imaginable), and propofol in 20-mg IV bolus doses was given to patients reporting verbal rating scale >3 during the procedure. Rescue medication consumption during surgery and analgesia requirements over the next 24 hours, as well as onset of sensory blockade, were recorded. A *P* value <0.05 was regarded as statistically significant.
RESULTS: Fewer patients in the fentanyl group (4 of 38, 10.5%) required propofol compared with the placebo group (26 of 39, 66.7%; *P* < 0.001). In comparison with the placebo group, the fentanyl group consumed less propofol (median 0 [0–60] vs 60 [0–160] mg, respectively; *P* < 0.001), required postoperative analgesia less frequently (22 of 38 patients, 57.9% vs 35 of 39 patients, 89.7%, respectively; *P* = 0.002), and requested the first analgesic after surgery later (median 5.8 [1.9–15.6] vs 3.1 [1.0–11.7] hours, respectively; *P* < 0.001), whereas the onset time of sensory blockade was similar in both groups (median 12 [9–18] vs 15 [9–18] minutes, respectively; *P* = 0.18).
CONCLUSIONS: The addition of fentanyl to local anesthetics improved the quality and prolonged the duration of cervical plexus block in patients undergoing CEA.

吸入一氧化碳可通過介導熱休克反應預防豬體外迴圈術後急性腎損傷的發生

Inhaled carbon monoxide prevents acute kidney injury in pigs after cardiopulmonary bypass by inducing a heat shock response.

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背景：體外迴圈術（CPB）與急性腎損傷（AKI）之間可能存有一定聯繫。而吸入一氧化碳（CO）具有細胞及器官的保護作用。此研究擬通過以吸入 CO 為預處理措施預防體外迴圈相關性急性腎損傷的發生。
方法：將試驗用豬（n=38）非隨機地分配至假 CPB 組、標準 CPB 組、假或標準 CPB 前 CO 預處理組（250ppm、1 小時）、吸入 CO 和進行 CPB 前槲皮素（熱休克反應抑制劑）或腎小管間質性原卟啉-IX（SnPPIX、內源性 CO 衍生抑制劑）預處理組。初步的試驗資料為 AKI 的相關指標（包括尿素、尿酸、肌酐、半胱氨酸蛋白酶抑制劑 C、中性粒細胞明膠酶相關脂質運載蛋白、白介素-6、腫瘤壞死因數-α 等），其均取 CPB 後 120 分鐘時予以檢測。進一步的檢測資料為熱休克蛋白（HSP）-70 及血紅素氧化酶-1 蛋白的表達量，其可作為 CO 介導的熱休克反應的指示劑。
結果：CO 預處理減輕了體外迴圈相關性急性腎損傷的發生（P 均＜0.001），主要表現在（1）血清中半胱氨酸蛋白酶抑制劑 C（64±14 對 28±9 ng/mL）、中性粒細胞明膠酶相關脂質運載蛋白（391±65 對 183±56 ng/mL）、腎腫瘤壞死因數-α（450±73 對 179±110 pg/mL）、及白介素-6（483±102 對 125±67 pg/mL）的濃度均明顯提高；（2）腎臟天冬氨酸特異性半胱氨酸蛋白酶-3 活性（550±66 對 259±52 相對螢光單位）顯著增加及（3）急性腎損傷的組織學證據。上述效應均伴隨有 HSP-70 的啟動（196±64 對 554±149 ng/mL，P＜0.001）。熱休克反應抑制劑槲皮素預處理可消除 CO 相關的生化和組織學腎保護效應（P 均＜0.001），而血紅素氧化酶抑制劑 SnPPIX 僅能部分抵抗 CO 相關的腎保護作用及阻礙熱休克反應啟動。
結論：組織學受損表現的減少及半胱氨酸蛋白酶抑制劑 C 濃度的下降證實 CPB 前行 CO 預處理具有明確的腎保護依據。研究發現，CO 的抗炎及抗細胞凋亡效應與 HSP-70 的受啟動化相伴產生，而槲皮素對其效應的逆轉作用提示 CPB 前行 CO 預處理的腎保護措施是通過啟動腎臟的熱休克反應而介導的。
（范羽譯 薛張綱校）

BACKGROUND: Cardiopulmonary bypass (CPB) may be associated with acute kidney injury (AKI). Inhaled carbon monoxide (CO) is cyto- and organ-protective. We hypothesized that pretreatment with inhaled CO prevents CPB-associated AKI.
METHODS: Pigs (n = 38) were nonrandomly assigned to SHAM, standard CPB, pretreatment with inhaled CO (250 ppm, 1 hour) before SHAM or CPB, to pretreatment with quercetin (an inhibitor of the heat shock response), and to pretreatment with SnPPIX (an inhibitor of endogenously derived CO), before CO inhalation and CPB. The primary outcome variables were markers of AKI (urea, uric acid, creatinine, cystatin C, neutrophil gelatinase-associated lipocalin, interleukin-6, tumor necrosis factor-alpha), which were determined 120 minutes after CPB. Secondary outcome variables were heat shock protein

(HSP)-70 and heme oxygenase-1 protein expressions as indicators of CO-mediated heat shock response.
RESULTS: Pretreatment with inhaled CO attenuated (all P < 0.001) CPB-associated, (1) increases in serum concentrations of cystatin C (64 +/- 14 vs 28 +/- 9 ng/mL), neutrophil gelatinase-associated lipocalin (391 +/- 65 vs 183 +/- 56 ng/mL), renal tumor necrosis factor-alpha (450 +/- 73 vs 179 +/- 110 pg/mL), and interleukin-6 (483 +/- 102 vs 125 +/- 67 pg/mL); (2) increase in renal caspase-3 activity (550 +/- 66 vs 259 +/- 52 relative fluorescent units); and (3) histological evidence of AKI. These effects were accompanied by activation of HSP-70 (196 +/- 64 vs 554 +/- 149 ng/mL, P < 0.001). Pretreatment with the heat shock response inhibitor quercetin counteracted the CO-associated biochemical and histological renoprotective effects (all P < 0.001), whereas the heme oxygenase inhibitor SnPPIX only partially counteracted the CO-associated renoprotection and the activation of the heat shock response.
CONCLUSIONS: CO treatment before CPB was associated with evidence of renoprotection, demonstrated by fewer histological injuries and decreased cystatin C concentrations. The findings that the antiinflammatory and antiapoptotic effects of CO were accompanied by activation of HSP-70, which in turn were reversed by quercetin, suggest that renoprotection by pretreatment with inhaled CO before CPB is mediated by activation of the renal heat shock response.

主動脈瓣修補術中的經食管超聲心動圖評估

Transesophageal echocardiographic evaluation during aortic valve repair surgery.
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對於主動脈瓣膜疾病和主動脈狹窄的患者，最經典的治療方法是主動脈瓣置換術。與之相比，主動脈關閉不全修復術是一種新興的具有可行性並且具有吸引力的方法，它對於伴或不伴有主動脈根部病變均適用。主動脈瓣是主動脈根部的組成部分之一。因此，當一個或更多的主動脈根部的組成部分發生病變時，主動脈關閉不全的病情就會進展。術中的經食管超聲心動圖評估方法可以分析主動脈返流的機制以及鑒別可修復與不可修復的主動脈瓣病變。修復術後即刻的經食管超聲心動圖能提供很多重要資訊，包括修復的品質，修復的耐久性以及發現與主動脈關閉不全有關的多種因素徵象。
（黃劍譯 薛張綱校）
For patients with aortic valve (AV) disease, the classic treatment has been AV replacement and this remains true for aortic stenosis. In contrast, repair of isolated aortic insufficiency (AI), with or without aortic root pathology, is emerging as a feasible and attractive option to replacement. The AV is one of the elements of the aortic root. As such, AI can develop if one or more elements of the aortic root are diseased. Intraoperative transesophageal echocardiographic evaluation permits analysis of the mechanisms of aortic regurgitation as well as differentiation between repairable and unrepairable AV pathology. Immediate postrepair transesophageal echocardiography

provides important information about the quality and durability of repair and identifies variables associated with recurrent AI.

大麻素配體 MDA19 治療神經性疼痛的藥理特性

Pharmacological Characterization of a Novel Cannabinoid Ligand, MDA19, for Treatment of Neuropathic Pain

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背景：大麻素受體 2（CB_2）激動劑治療神經性疼痛潛在靶點的研究近來引起關注。通過研究，我們描繪一個 CB_2 激動劑複合物 N'-[（3Z）-1-（1-己基）-2-氧代-1,2 二氫 3H-吲哚-3-亞基]的藥理學曲線。
方法：我們對人和大鼠的 CB_1 和 CB_2 受體採用放射性配體結合分析以及多次體外功能性分析。評估 MDA19 對逆轉大鼠及 $CB_2^{+/+}$和 $CB_2^{-/-}$小鼠各式神經性疼痛的作用。
結果：MDA19 對人 CB_2 受體的親和力是 CB_1 受體的 4 倍（K_1=43.3±10.3vs162.4±7.6 nM)，對大鼠 CB_2 受體的親和力是 CB_1 受體的近 70 倍 (K_1=16.3±2.1vs1130±574nM）。在鳥苷三磷酸（GTP）γ[^{35}S]功能性分析中，MDA19 對人類 CB_1 和 CB_2 受體及大鼠 CB_1 受體有激動作用，對大鼠 CB_2 受體起反激動作用。3,5-環單磷酸腺苷（cAMP）中，MDA19 對大鼠 CB_1 受體有激動作用，對大鼠 CB_2 受體沒有作用。 對細胞外信號調節激酶 1 和 2 的活化分析顯示 MDA19 對大鼠 CB_2 受體有激動作用。MDA19 可減弱 $CB_2^{+/+}$小鼠由脊神經離斷或紫杉醇劑量依賴所致的異常疼痛，對 $CB_2^{-/-}$小鼠無此作用，表明 MDA19 通過 CB_2 受體發揮作用。MAD19 不影響大鼠的運動能力。
結論：MDA19 在體外功能性研究中對大鼠 CB_2 受體有顯著作用，在體內類似蛋白激動劑對 CB_1/CB_2 有激動作用。MDA19 在減緩神經性疼痛而不對中樞神經系統產生副作用方面有很大優勢。
（毛慧譯，薛張綱校）

BACKGROUND: Cannabinoid receptor2 (CB_2) agonists have recently gained attention as potential therapeutic targets in the management of neuropathic pain. In this study, we characterized the pharmacological profile of the novel compound *N*□-[(3*Z*)-1-(1-hexyl)-2-oxo-1,2-dihydro-3*H*-indol-3-ylidene]benzohydrazide (MDA19), a CB_2 agonist.
METHODS: We used radioligand binding assays and multiple in vitro functional assays at human and rat CB_1 and CB_2 receptors. The effects of MDA19 in reversing neuropathic pain were assessed in various neuropathic pain models in rats and in $CB_2^{+□/□+}$and $CB_2^{-/-}$ mice.
RESULTS: MDA19 displayed 4-fold-higher affinity at the human CB_2 than at the human CB_1 receptor (K_1=43.3±10.3vs162.4±7.6 nM) and nearly 70-fold-higher affinity at the rat CB_2 than at the rat CB_1 receptor (K_1=16.3±2.1vs1130±574nM). In guanosine triphosphate (GTP)γ□[^{35}S] functional assays, MDA19 behaved as an agonist at the

human CB_1 and CB_2 receptors and at the rat CB_1 receptor but as an inverse agonist at the rat CB_2 receptor. In 3□,5□-cyclic adenosine monophosphate (cAMP) assays, MDA19 behaved as an agonist at the rat CB_1 receptor and exhibited no functional activity at the rat CB_2 receptor. In extracellular signal-regulated kinases 1 and 2 activation assays, MDA19 behaved as an agonist at the rat CB_2 receptor. MDA19 attenuated tactile allodynia produced by spinal nerve ligation or paclitaxel in a dose-related manner in rats and $CB_2^{+/+}$mice but not in $CB_2^{-/-}$ mice, indicating that CB_2 receptors mediated the effects of MDA19. MDA19 did not affect rat locomotor activity.
CONCLUSIONS: We found that MDA19 exhibited a distinctive in vitro functional profile at rat CB_2 receptors and behaved as a CB_1/CB_2 agonist in vivo, characteristics of a protean agonist.
MDA19 has potential for alleviating neuropathic pain without producing adverse effects in the central nervous system.

我們所知的殘餘肌松。第二部分：減少殘餘肌松風險的方法

Residual Neuromuscular Block: Lessons Unlearned. Part II: Methods to Reduce the Risk of Residual Weakness

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本綜述第二部分的目的是觀察可在術後早期供臨床醫生運用的最優的神經肌肉管理策略。現代觀點認為臨床頻繁使用的判斷神經肌肉功能的測試如抬頭或握拳不能可靠的排除殘餘肌松的存在。當使用定性（視和觸）的神經肌肉監測如四個成串刺激，雙重爆發刺激或強直刺激，四個成串刺激比值為0.6至1.0之間視臨床醫生常常不能發現衰減。而且定性監測在術後殘餘肌松方面的作用意見仍不一。然而，強有力的證據顯示加速度法監測（定量法）能更好的檢測較少的殘餘肌松（四個成串刺激比值>0.6）。中效的神經肌肉阻滯劑與長效神經肌肉阻滯劑相比能減少但不能消除殘餘肌松的風險。而且，抗膽鹼酯酶藥物應用早（拔管前15-20分鐘）、神經阻滯較淺時（四個成串刺激比值為4）更有可能使神經肌肉功能完全恢復。最後，新近發明的起效快、作用時程短的神經肌肉阻滯藥物和有效拮抗較深程度神經肌肉阻滯的選擇性的神經肌肉逆轉藥物可能在預防術後殘餘肌松及其併發症方面為臨床醫生提供新的選擇。
(姚敏敏譯 薛張綱校)
The aim of the second part of this review is to examine optimal neuromuscular management strategies that can be used by clinicians to reduce the risk of residual paralysis in the early postoperative period. Current evidence has demonstrated that frequently used clinical tests of neuromuscular function (such as head lift or hand grip) cannot reliably exclude the presence of residual paralysis. When qualitative (visual or tactile) neuromuscular monitoring is used (train-of-four [TOF], double-burst, or tetanic stimulation patterns), clinicians often are unable to detect fade when TOF ratios are

between 0.6 and 1.0. Furthermore, the effect of qualitative monitoring on postoperative residual paralysis remains controversial. In contrast, there is strong evidence that acceleromyography (quantitative) monitoring improves detection of small degrees (TOF ratios >0.6) of residual blockade. The use of intermediate-acting neuromuscular blocking drugs (NMBDs) can reduce, but do not eliminate, the risk of residual paralysis when compared with long-acting NMBDs. In addition, complete recovery of neuromuscular function is more likely when anticholinesterases are administered early (>15–20 minutes before tracheal extubation) and at a shallower depth of block (TOF count of 4). Finally, the recent development of rapid-onset, short-acting NMBDs and selective neuromuscular reversal drugs that can effectively antagonize deep levels of blockade may provide clinicians with novel pharmacologic approaches for the prevention of postoperative residual weakness and its associated complications.

嚴重敗血症患者的皮膚膠原蛋白合成降低

Skin Collagen Synthesis Is Depressed in Patients with Severe Sepsis

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背景：皮膚是保持內環境穩定的重要屏障。足夠的再生能力對於克服污染菌侵襲所帶來的對內穩態的挑戰是非常重要的。已知在發生敗血症時凝血功能和炎症被啟動來恢復內穩態，但不知道敗血症是否也影響組織再生如皮膚膠原蛋白的合成。

方法：在這項前瞻性觀察研究中，我們測量敗血症患者水皰液中膠原蛋白 1 和膠原蛋白 3 的氨基酸多肽的合成。 水皰液來自於腹部皮膚實驗誘導所得的水皰的四個時相：第一個器官功能衰竭內的 48 小時、第 5 天以及 3 個月和 6 個月後。 44 個重度膿毒症患者收入組內。 年齡中位數為 63 歲（四分之一位數和四分之三位數為 53 歲和 71 歲）。 入選者的急性生理學和慢性健康評估得分的中位數為 26（22-30）。 30 天的死亡率為 25%。 15 名健康成人入選對照組。

結果：與對照組相比，膠原蛋白 1 和膠原蛋白 3 的中位數水準在敗血症患者的水皰第一時相降低（分別為 40.8 μg/L [P1/4 和 P3/4, 22.2–77.1 μg/L], $P = 0.028$ 和 69.9 μg/L [32.4–112.7 μg/L], $P < 0.001$），第二時相也相對對照組降低(分別為 38.8 μg/L [19.9–68.5 μg/L], $P < 0.001$ 和 90.0 [35.1–138.8 μg/L], $P < 0.001$)。倖存者顯示膠原蛋白 1 和膠原蛋白 3 在三個月後超表達，在六個月後正常表達。

結論：嚴重敗血症時皮膚膠原蛋白合成抑制，並在發病後的三個月和六個月發生代償反應。

（張玥琪譯，薛張綱校）

BACKGROUND: Skin is an essential barrier in maintaining a stable internal environment. Adequate regenerative capacity is crucial to overcome the homeostatic challenges caused by a septic insult. In sepsis, coagulation and inflammation are activated

to restore homeostasis, but it is not known whether sepsis also alters tissue regeneration processes such as skin collagen synthesis.
METHODS: In this prospective observational study, we measured aminoterminal propeptides of collagens I and III (PINP, PIIINP) from blister fluid of sepsis patients. Blister fluid was obtained from experimental blisters induced on intact abdominal skin 4 times: within the first 48 hours from the first organ failure, on the fifth day, and at 3 and 6 months thereafter. Forty-four patients with severe sepsis were enrolled. The median age was 63 years (25th–75th percentile, 53–71 years). The median Acute Physiology and Chronic Health Evaluation II score on admission was 26 (22–30). Thirty-day mortality was 25%. Fifteen healthy adults were used as controls.
RESULTS: Median PIIINP and PINP levels in septic patients were lower in comparison with controls in the first blister (40.8 μg/L [25th–75th percentile, 22.2–77.1 μg/L], P = 0.028 and 69.9 μg/L [32.4–112.7 μg/L], $P < 0.001$, respectively) and in the blister induced on day 5 (38.8 μg/L [19.9–68.5 μg/L], $P < 0.001$ and 90.0 [35.1–138.8 μg/L], $P < 0.001$, respectively). The survivors revealed an overexpression at 3 months, whereas normal values of PIIINP and PINP were reestablished at 6 months.
CONCLUSIONS: Skin collagen synthesis is depressed during severe sepsis and is followed by a compensatory response 3 and 6 months after the onset of sepsis.

氨基已酸在體外抑制新生兒血漿纖維蛋白溶解的有效濃度

The Effective Concentration of Epsilon-Aminocaproic Acid for Inhibition of Fibrinolysis in Neonatal Plasma in Vitro

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介紹：兒科患者，尤其是新生兒，由於他們的止血系統發育不完善，體積小，手術複雜，接受心臟手術後發生出血併發症的風險很高。血管內纖維蛋白啟動是體外迴圈最基本的影響之一，可導致術後止血功能障礙。這一併發症已被認識很久，並且用抗纖溶藥物進行治療，包括賴氨酸同型 ε 氨基乙酸(EACA)。成人的 EACA 治療血漿濃度已經科學的確定，但是新生兒的目前推薦劑量是根據成人的劑量經驗性的得來。因此，我們研究了體外迴圈時 EACA 在新生兒的合適濃度。
方法：我們用從 20 個足月、選擇剖腹產分娩的胎盤臍帶血中分離出的新生兒血漿進行了一項體外研究。在用組織型纖溶酶原啟動劑啟動纖維蛋白溶解前，將各等級濃度的 EACA 加入等分的血漿容器中。然後進行標準白陶土啟動的血栓彈性描記圖檢測，並評估溶解百分比初步結果的變異。這些過程在購買的混合成人正常血漿樣本中重複進行以作對比。
結果：我們發現完全抑制纖溶新生兒所需要的 EACA 濃度明顯低於成人（對 400 和 1000 U/mL 的纖溶酶原啟動物，新生兒 EACA 濃度為 44.2 μg/mL 和 47.8 μg/mL，成人為 94.4 和 131.4 μg/mL，$P < 0.001$）。

結論：我們的資料確定了 EACA 在體外新生兒血中完全抑制纖維蛋白溶解所必需的最低有效濃度。這一濃度顯著低於目前的定量規則確定的濃度值，表明新生兒可能暴露於較臨床所必需的更高水準的 EACA。
（朱蘭芳譯，薛張綱校）

INTRODUCTION: Pediatric patients, particularly neonates, are at high risk for bleeding complications after cardiovascular surgery because of their immature hemostatic system, small size, and the complex operations they require. Activation of intravascular fibrinolysis is one of the principle effects of cardiopulmonary bypass that causes poor postoperative hemostasis. This complication has long been recognized and treated with antifibrinolytic medications, including the lysine analog ε aminocaproic acid (EACA). The therapeutic plasma concentration of EACA has been scientifically determined for the adult population, but the current recommended dosage for neonates has been empirically derived from adult studies. Therefore, we investigated the appropriate concentration of EACA for neonates undergoing bypass.

METHODS: We conducted an in vitro study using neonatal plasma derived from the placenta/cord units from 20 term, elective cesarean deliveries. Graded concentrations of EACA were added to aliquots of the plasma pool before activating fibrinolysis with tissue-type plasminogen activator. Standard kaolin-activated thromboelastograms were then run with the primary outcome variable being estimated percent lysis. These procedures were repeated on samples of commercially available pooled adult normal plasma for comparison.

RESULTS: We found that neonatal plasma required significantly lower concentrations of EACA to completely prevent fibrinolysis than did adult plasma (44.2 μg/mL and 47.8 μg/mL for neonatal plasma and 94.4 and 131.4 μg/mL in adult plasma for 400 and 1000 U/mL of plasminogen activator, respectively, $P < 0.001$).

CONCLUSIONS: Our data establish the minimal effective concentration of EACA necessary to completely prevent fibrinolysis in neonatal blood in vitro. This concentration is significantly less than that targeted by current dosing schemes, indicating that neonates are possibly being exposed to greater levels of EACA than is clinically necessary.

竇椎神經阻滯應用于腰段椎間盤來源疼痛的診斷：一項探索性研究

Blockade of the Sinuvertebral Nerve for the Diagnosis of Lumbar Diskogenic Pain: An Exploratory Study

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在這個探索性研究中，我們評估了竇椎神經阻滯（SVNB）用於診斷腰段椎間盤來源疼痛的敏感性和特異性。椎間盤造影是診斷該類疾病的金標準。15 名椎間盤造影陽性的患者經後路行 SVNB。阻滯成功的定義為≥80%的疼痛緩解或身體限制緩解。SVNB 的靈敏度為 73.3%（95%置信區間為 50.9%-95.7%）。特異性為 40%

（15.2%-64.8%）。結果表明，SVNB 還不能取代椎間盤造影，但鼓勵進一步研究來提高其特異性。
(陳珺珺譯 薛張綱校)
In this exploratory study we evaluated sensitivity and target specificity of sinuvertebral nerve block (SVNB) for the diagnosis of lumbar diskogenic pain. Diskography has been the diagnostic gold standard. Fifteen patients with positive diskography underwent SVNB via interlaminar approach to the posterior aspect of the disk. Success was defined as ≥80% pain reduction or excellent relief of physical restrictions after the block. The sensitivity was 73.3% (95% CI: 50.9%–95.7%). The target specificity was 40% (15.2%–64.8%). The results indicate that SVNB cannot yet replace diskography but encourage future studies to improve its target specificity.

在一項對照研究中研究坐骨神經阻滯持續時間和利多卡因釋放速率的關係

The Relationship Between Functional Sciatic Nerve Block Duration and the Rate of Release of Lidocaine from a Controlled-Release Matrix

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背景：我們希望在圍手術期和術後神經阻滯有較長維持時間。瞭解神經阻滯維持時間和局麻藥釋放速率的關係對於發展局麻藥釋放系統是重要的，從而可以優化神經阻滯持續時間。
方法：在 OSB-L 組，利多卡因的濃度不同，但是以恒定的速率釋放。 在另一組(OST-R),利多卡因的濃度恒定，但是釋放速率不同。試劑植入試驗鼠的坐骨神經附近後，在體及離體測定與抗傷害性刺激及運動阻滯相關的釋放動力學。在平行試驗中，實驗鼠接受了緩慢釋放的利多卡因後，心內注射 4%多聚甲醛，並取神經肌肉組織進行組織學分析。
結果：在這項研究中，我們證明了影響神經阻滯的各種因素中（例如阻滯坐骨神經相關纖維衝動傳導），最重要的因素是利多卡因釋放的速率。在 OSB-L 組（利多卡因的濃度分別為 1.875%, 3.75%, 7.5%和 15% ，以恒定的 5%的速率釋放）,在體試驗中，50%運動阻滯及傷害刺激恢復的平均時間分別為 0.91 ± 0.28 和 1.75 ± 0.61 mg/h。在 OST-R 組（16%的利多卡因分別以 1.875%, 3.75%, 7.5%和 15%的濃度釋放),在體 50%運動阻滯及傷害性刺激恢復的平均時間分別為 2.33 ± 1.39 和 4.34 ± 1.09 mg/h。在 OSB-L 組顯示了增加利多卡因的起始濃度可以劑量依賴性的增加阻滯持續時間，而在 OST-R 組中顯示了釋放速率濃度並不是阻滯持續時間的單獨因素。在植入試劑後 24 小時、3 天、5 天、7 天和 4 周進行組織學研究發現炎症反應的程度和利多卡因含量正相關，但是局限在植入物周圍組織。儘管觀察到炎症反應，抗傷害性刺激及運動阻滯均可以恢復到植入前。

結論：增加利多卡因的起始濃度可以成比例地延長坐骨神經阻滯時間。然而降低每次釋放地速率並不能成比例地延長阻滯時間。相反，間斷給藥可以最大程度延長阻滯持續時間，是優化的方案。
（陳珺珺譯 薛張綱校）

BACKGROUND: Nerve blocks of long duration are often desirable in perioperative and postoperative situations. The relationship between the duration of such blocks and the rate at which a local anesthetic is released is important to know for developing a localized drug delivery system that will optimize block duration.
METHODS: Lidocaine concentration was varied in 1 series of formulations (OSB-L) containing a constant amount of release rate modifier. In another series (OST-R), the release rate modifier was varied while the lidocaine content was held constant. Release kinetics were measured in vitro and correlated to the in vivo duration of antinociceptive and motor block effects when the formulation was implanted next to the rat sciatic nerve. In parallel studies, rats receiving different formulations of slow-release lidocaine were fixed by intracardiac perfusion with 4% paraformaldehyde and nerve-muscle tissue taken for histopathological analysis.
RESULTS: In this study, we have demonstrated that the most important variable for effecting functional nerve block, i.e., the blockade of impulses in the relevant fibers of the sciatic nerve, is the rate of lidocaine release at that time. For the OSB-L formulations (lidocaine concentrations of 1.875%, 3.75%, 7.5%, and 15% at a constant release rate modifier of 5%), the average in vitro release rates at 50% recovery of motor block and nociceptive block were 0.91 ± 0.28 and 1.75 ± 0.61 mg/h, respectively. For the OST-R formulations (16% lidocaine with release rate modifier concentrations of 1.875%, 3.75%, 7.5%, and 15%), the average in vitro release rates at 50% recovery of motor block and nociceptive block were 2.33 ± 1.39 and 4.34 ± 1.09 mg/h, respectively. The OSB-L formulations showed a dose-dependent increase in block duration proportional to an increase in initial lidocaine concentration, whereas the OST-R formulations showed a nonmonotonic relationship between release rate modifier concentration and block duration. The histopathological studies at 24 hours, 3, 5, or 7 days, and 4 weeks after the implantation revealed inflammatory reactions with degrees correlated with lidocaine content, but limited to the connective tissue and muscle immediately surrounding the implanted material. Despite these observed inflammatory reactions, nociceptive and motor block function returned to normal, preimplantation values in all animals.
CONCLUSIONS: Increasing initial lidocaine content proportionately increased the duration of functional sciatic nerve block. However, decreasing the release rate per se does not give a proportional increase in block duration. Instead, there seems to be an optimal, intermediate release rate for achieving the maximum duration of block.