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揮發性麻醉藥異氟醚通過磷酸肌醇-3 激酶/ Akt 信號途徑預防小鼠呼吸機相關肺損傷

The Volatile Anesthetic Isoflurane Prevents Ventilator-Induced Lung Injury via Phosphoinositide 3-Kinase/Akt Signaling in Mice

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背景：機械通氣在動物中可以導致呼吸機相關性肺損傷，在人體中可以導致急性肺損傷/急性呼吸窘迫綜合征。急性肺損傷/急性呼吸窘迫綜合征目前是導致危重病人高患病率和死亡率的重要原因。揮發性麻醉藥在活體中被證實有抗炎和器官保護作用。本文研究揮發性麻醉藥異氟醚在機械性通氣中對肺損傷的影響。
方法：使用 12ml/kg 潮氣量含或不含異氟醚的氣體對 C7BL/6N 小鼠進行通氣 6 小時，隨後根據是否使用特異性的磷酸肌醇-3 激酶/ Akt 信號通路抑制劑 LY294002 進行再次試驗。通過比較組織學、支氣管肺泡灌洗液細胞分離計數以及 ELISA 檢測細胞因數水準等方法評價肺損傷。通過 western blotting 分析肺勻漿中的蛋白表達。
結果：機械通氣導致肺泡壁增厚，細胞侵潤和呼吸機相關肺損傷評分增加。在通氣小鼠的肺泡灌洗液中，中性粒細胞浸入以及細胞因數（如白介素-1β，巨噬細胞炎症蛋白-2 等）釋放增加。在肺組織勻漿中應激蛋白血紅素氧化酶-1 和熱休克蛋白-70 的表達增加。異氟醚通氣可以顯著減少肺損傷、炎症反應以及應激蛋白表達。相反地，在異氟醚通氣中， Akt 蛋白磷酸化增加。在活體中，機械通氣前抑制磷酸肌醇-3 激酶/Akt 信號可以完全逆轉異氟醚的肺保護作用。
結論：在機械通氣中異氟醚的吸入可以通過抑制促炎症反應保護免受肺損傷。該保護作用是通過磷酸肌醇-3 激酶/Akt 信號途徑介導。
（俞芳　譯　陳傑　校）
BACKGROUND: Mechanical ventilation leads to ventilator-induced lung injury in animals, and can contribute to acute lung injury/acute respiratory distress syndrome in humans. Acute lung injury/acute respiratory distress syndrome currently causes an unacceptably high rate of morbidity and mortality among critically ill patients. Volatile anesthetics have been shown to exert anti-inflammatory and organ-protective effects in vivo. We investigated the effects of the volatile anesthetic isoflurane on lung injury during mechanical ventilation.
METHODS: C57BL/6N mice were ventilated with a tidal volume of 12 mL/kg body weight for 6 hours in the absence or presence of isoflurane, and, in a second series, with or without the specific phosphoinositide 3-kinase/Akt inhibitor LY294002. Lung injury was determined by comparative histology, and by the isolation of bronchoalveolar lavage for differential cell counting and analysis of cytokine levels using enzyme-linked immunosorbent assays. Lung homogenates were analyzed for protein expression by Western blotting.

RESULTS: Mechanical ventilation caused increases in alveolar wall thickening, cellular infiltration, and an elevated ventilator-induced lung injury score. Neutrophil influx and cytokine (i.e., interleukin-1β, and macrophage inflammatory protein-2) release were enhanced in the bronchoalveolar lavage of ventilated mice. The expression levels of the stress proteins hemeoxygenase-1 and heat shock protein-70 were elevated in lung tissue homogenates. Isoflurane ventilation significantly reduced lung damage, inflammation, and stress protein expression. In contrast, phosphorylation of Akt protein was substantially increased during mechanical ventilation with isoflurane. Inhibition of phosphoinositide 3-kinase/Akt signaling before mechanical ventilation completely reversed the lung-protective effects of isoflurane treatment in vivo.
CONCLUSIONS: Inhalation of isoflurane during mechanical ventilation protects against lung injury by preventing proinflammatory responses. This protection is mediated via phosphoinositide 3-kinase/Akt signaling.

超聲引導的頸內靜脈置管期間頸正中位的安全性高於頸部旋轉 45°嗎？一項臨床隨機對照試驗結果

Is a Neutral Head Position Safer than 45-Degree Neck Rotation During Ultrasound-Guided Internal Jugular Vein Cannulation? Results of a Randomized Controlled Clinical Trial

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背景：理想的頸內靜脈（internal jugular vein ，IJV）置管時採取的最佳頸部旋轉角度仍不清楚，因爲先前研究使用超聲，但未及刺破靜脈的情況。本文旨在比較超聲引導下的頸內靜脈置管過程中頸正中位（neutral position ，NP，0 度）與旋轉 45 度的安全性。首要評估指標是這兩種體位對超聲引導下的頸內靜脈置管主要併發症的影響，另外還評估了整體併發症、穿刺時間和穿刺過程中的困難。
方法：在一家神經外科醫院進行此項前瞻性隨機對照非盲研究，將接受擇期神經外科手術並需要中心靜脈置管的患者隨機分爲兩組，進行平面外技術超聲引導下的頸內靜脈置管。
結果：總共對 1424 名患者進行了評估，其中 92 名患者被排除，頸部旋轉位組有 670 人，正中位組有 662 人。置管成功率均爲 100%，除了頸內靜脈的位置兩組樣本的人口學資料相似。出現的嚴重併發症只有 10 例：其中 6 例出現在 0 度 NP 組，4 例出現在 45 度旋轉組。併發症的發生率兩組無明顯差異。整體併發症發生率是 13%，女性、ASA≥II、靜脈直徑小、靜脈在外側較深或前外側的患者的發生率更高。穿刺時間的延長與併發症發生率的增加一致。兩組在穿刺時感覺到困難的差異並無統計學意義。
結論：由於主要及次要併發症、穿刺時間方面的相似性，採取正中位進行頸內靜脈置管和旋轉 45°體位置管相比安全性並無明顯差異。超聲引導可以幫助我們選擇頸內靜脈置管時的最佳旋轉角度。
（夏蘇雲　譯　陳傑　校）

BACKGROUND: The optimal degree of neck rotation during internal jugular vein (IJV) cannulation remains undetermined because previous studies suggested using sonography, but without puncturing the vein. We assessed whether a neutral position (NP) of the head (0 degrees) during ultrasound-guided cannulation of the IJV was safer than rotating the neck to 45 degrees head turned. The effect of these 2 positions during ultrasound-guided cannulation on major complications was the primary outcome. Overall complications, venous access time, and perception of difficulty during the procedure were also evaluated.
METHODS: A prospective, randomized, controlled, nonblinded study was conducted in a tertiary neurosurgical hospital. Patients undergoing major elective neurosurgical procedures requiring a central venous line were randomly allocated to 2 groups; ultrasound-guided cannulation of the IJV was then performed using an out-of-plane orientation.
RESULTS: One thousand four hundred twenty-four patients were evaluated, but 92 were excluded; 670 were allocated to the head turned group and 662 to the NP group. Cannulation was 100% successful. Demographic data were similar in the 2 groups except for IJV positions. There were only 10 major complications: 6 in the 0-degree NP group and 4 in the 45-degree head turned group. The frequency of these complications was not different between the 2 groups. The overall complication rate was 13%, and was higher in women, in patients with ASA physical status ≥II, and in patients with a smaller diameter vein, or when the vein was located deeper and lateral or in the anterolateral position. An increased venous access time was associated with an increased rate of overall complications. The perception of difficulty performing the procedure with the head placed in the 2 positions was not statistically different in either group.
CONCLUSION: A head NP was as safe as a 45-degree neck rotation during ultrasound-guided IJV cannulation with regard to both major and minor complications, and venous access time was similar. Ultrasound guidance helps determine optimal head rotation for IJV cannulation.

ICU 中床邊監測平均灌注壓（Pmsf）和臨界閉合壓（Pcc）確定血管瀑布現象

Determination of Vascular Waterfall Phenomenon by Bedside Measurement of Mean Systemic Filling Pressure and Critical Closing Pressure in the Intensive Care Unit

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背景：平均灌注壓（Pmsf）資料可以在床邊通過在屏氣過程中測量中心靜脈壓（Pcv）和心輸出量（CO）得到。臨界閉合壓（Pcc）的資料也可以通過同樣的方法測得動脈壓（Pa）和 CO 得出。當 Pcc 大於 Pmsf，就會出現血管瀑布現象。此項研究通過床邊測量 Pmsf 和 Pcc 來評定血流瀑布現象的存在以及與其相關的血管阻力的計算。
方法：10 例心臟術後機械通氣的患者中，通過屏氣短暫增加 Pcv 和降低 Pa 和 CO 到 4 個不同的穩定狀態。對於每個患者，Pcv 和 CO 的值被繪製成靜脈回流曲線從而得出 Pmsf。同樣，Pa 和 CO 也可繪製成心室輸出曲線而得出 Pcc。在每個病人擴容前以及用 0.5L 膠體擴容後分別進行以上測量，同時計算血管阻力。
結果：對於每個病人， Pcv 和 CO 之間及 Pa 和 CO 之間的 4 次測量都有線性關係。Pmsf 的基礎值爲 18.7 ± 4.0 mm Hg，與 Pcc 的 45.5 ± 11.1 mm Hg 有明顯不同。Pmsf 和 Pcc 的差

異達 26.8 ± 10.7 mm Hg，說明了全身血管瀑布現象的存在。擴容增加 Pmsf（26.3 ± 3.2 mm Hg），Pcc（51.5 ± 9.0 mm Hg）和 CO（5.5 ± 1.8 to 6.8 ± 1.8 L/ min）。動脈（Pcc 的上游）和靜脈（Pmsf 的下游）血管阻力是 8.27 ± 4.45 and 2.75 ± 1.23 mm Hg ·min ·L^{-1}，兩者之和是 11.01 mm Hg ·min ·L^{-1}，與全身系統血管阻力 16.56 ± 8.57 mm Hg ·min ·L^{-1} 有明顯差異。動脈阻力與全身阻力相關。

結論：心臟手術病人血管壓力梯度表明了血管瀑布現象的存在，而且並不被 CO 所影響。所以，全身系統血管阻力的測量可能與評估系統血管張力並不相關。

（範逸辰　譯　陳傑　校）

BACKGROUND: Mean systemic filling pressure (Pmsf) can be determined at the bedside by measuring central venous pressure (Pcv) and cardiac output (CO) during inspiratory hold maneuvers. Critical closing pressure (Pcc) can be determined using the same method measuring arterial pressure (Pa) and CO. If Pcc > Pmsf, there is then a vascular waterfall. In this study, we assessed the existence of a waterfall and its implications for the calculation of vascular resistances by determining Pmsf and Pcc at the bedside.

METHODS: In 10 mechanically ventilated postcardiac surgery patients, inspiratory hold maneuvers were performed, transiently increasing Pcv and decreasing Pa and CO to 4 different steady-state levels. For each patient, values of Pcv and CO were plotted in a venous return curve to determine Pmsf. Similarly, Pcc was determined with a ventricular output curve plotted for Pa and CO. Measurements were performed in each patient before and after volume expansion with 0.5 L colloid, and vascular resistances were calculated.

RESULTS: For every patient, the relationship between the 4 measurements of Pcv and CO and of Pa and CO was linear. Baseline Pmsf was 18.7 ± 4.0 mm Hg (mean ± SD) and differed significantly from Pcc 45.5 ± 11.1 mm Hg ($P < 0.0001$). The difference of Pcc and Pmsf was 26.8 ± 10.7 mm Hg, indicating the presence of a systemic vascular waterfall. Volume expansion increased Pmsf (26.3 ± 3.2 mm Hg), Pcc (51.5 ± 9.0 mm Hg), and CO (5.5 ± 1.8 to 6.8 ± 1.8 L · min^{-1}). Arterial (upstream of Pcc) and venous (downstream of Pmsf) vascular resistance were 8.27 ± 4.45 and 2.75 ± 1.23 mm Hg ·min ·L^{-1}; the sum of both (11.01 mm Hg ·min ·L^{-1}) was significantly different from total systemic vascular resistance (16.56 ± 8.57 mm Hg ·min ·L^{-1}; $P = 0.005$). Arterial resistance was related to total resistance.

CONCLUSIONS: Vascular pressure gradients in cardiac surgery patients suggest the presence of a vascular waterfall phenomenon, which is not affected by CO. Thus, measures of total systemic vascular resistance may become irrelevant in assessing systemic vasomotor tone.

剖宮產術後靜注帕瑞考昔後其主要活性代謝物伐地考昔的母乳轉移:單純集聚資料分析法和非線性混合效應模型法的比較

Transfer of Parecoxib and Its Primary Active Metabolite Valdecoxib via Transitional Breastmilk Following Intravenous Parecoxib Use After Cesarean Delivery: A Comparison of Naïve Pooled Data Analysis and Nonlinear Mixed-Effects Modeling

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背景: 剖宮產術後數日通常運用多模式鎮痛, 包括非阿片類鎮痛藥的使用。由於母乳餵養小孩在此期間接受母乳，所以有必要瞭解究竟有多少母體的麻醉藥被嬰兒接受。爲此本試驗目的爲評估在母體剖宮產術後單次靜脈注射帕瑞考昔，嬰兒對帕瑞考昔及其活性代謝物伐地考昔的暴露程度 。

方法:40 名婦女及其嬰兒參與研究，在嬰兒出生平均 41 小時後，母體靜注帕瑞考昔(40mg)。在之後的 24 小時收集母乳(4 個樣本)和血漿(1 個樣本)，用液相色譜 - 串聯質譜分析法測定藥物含量。嬰兒的資料收集在帕瑞考昔注射後的第二天。使用單純集聚資料的標準方法評估母乳的帕瑞考昔和伐地考昔的絕對(AID)和相對嬰兒劑量(RID)，這樣也可以計算出母乳/血漿(M/P) 濃度比。非線性混合效應模型也被用來將伐地考昔的母乳和血漿的資料集與房室模型配對，並預測 M/P，AID 和 RID。

結果:帕瑞考昔和伐地考昔的 M/P 比（中位數[四分位範圍，IQR]）分別爲 0.5（0.15 至 1.15）和 0.14（0.11 至 0.18）。運用單純集聚資料分析，帕瑞考昔的 AID（母乳中的藥物濃度×每天的乳汁攝入量/公斤）爲 0.24(0.05 至 1.85) 微克/公斤/天，伐地考昔的 AID 爲 1.82（1.12 至 2.73）微克/公斤/天。帕瑞考昔的 RID 是 0.04（0.01 至 0.43）%的產婦體重調整劑量（24 小時內一次劑量），伐地考昔的爲 0.47（0.29 至 0.69）%（與帕瑞昔布等值）。伐地考昔的房室模型單獨生產的平均（個體差異）M/P 爲 0.149（26%），中位數（IQR）AID 爲 1.47（0.96 至 2.03）微克/公斤/天，中位數（IQR）RID 爲 0.39（0.28 至 0.47）%。新生兒神經和適應能力評分（平均= 34，95%CI 爲 33%至 35%）爲正常的預期得 35 分一致。

結論: 單純集聚資料分析法和非線性混合效應模型法都給出了相似的結論，帕瑞昔布和伐地考昔的 RID 很低。本研究結論顯示：給予剖宮產術後哺乳的婦女靜注 40 毫克一次劑量的 COX-2 抑制劑帕瑞考昔，不會對母乳餵養的嬰兒造成不良影響。

（俞劼晶　譯　陳傑　校）

BACKGROUND: Multimodal analgesia, including nonopioid analgesics, is usually used for several days after cesarean delivery. Because the breastfed infant receives transitional milk during this same period, it is important to know how much of a maternal analgesic drug is received by the infant. We designed this study to estimate infant exposure to parecoxib and its active metabolite valdecoxib (a cyclooxygenase-2 inhibitor) after a single IV maternal dose of parecoxib after cesarean delivery.

METHODS: Forty women and their infants participated in the study. Parecoxib (40 mg) was administered IV at a mean of 41 hours after birth. Milk (4 samples) and plasma (1 sample) were collected from the women over the subsequent 24 hours and drug content was measured by liquid chromatography-tandem mass spectrometry. The infants were assessed the day after parecoxib dosing. Absolute (AID) and relative infant doses (RID) of both parecoxib and valdecoxib through milk were estimated by standard methods using the naïve pooled datasets, and where possible milk/plasma (M/P) concentration ratios were calculated. Nonlinear mixed-

effects modeling was also used to fit the valdecoxib milk and plasma datasets to a compartmental model and to predict M/P, AID, and RID.
RESULTS: M/P ratios (median [interquartile range; IQR]) were 0.5 (0.15 to 1.15) for parecoxib and 0.14 (0.11 to 0.18) for valdecoxib. Using the naïve pooled datasets, AID (drug concentration in milk×daily milk intake/kg) was 0.24 (0.05 to 1.85) μg/kg/day for parecoxib, and 1.82 (1.12 to 2.73) μg/kg/day, for valdecoxib. RID was 0.04 (0.01 to 0.43) % of the weight-adjusted maternal dose (one dose in 24 hours) for parecoxib and 0.47 (0.29 to 0.69) % for valdecoxib (as parecoxib equivalents). Compartmental modeling of valdecoxib alone produced a mean (interindividual variability) M/P of 0.149 (26%), median (IQR) AID of 1.47 (0.96 to 2.03) μg/kg/day, and median (IQR) RID of 0.39 (0.28 to 0.47) %. Neonatal neurologic and adaptive capacity scores (mean=34, 95% CI 33 to 35) were consistent with a normal expected score of 35.
CONCLUSIONS: Both the naïve pooling of data and the modeling analyses gave similar results. The RID of both parecoxib and valdecoxib was low. We conclude that a single 40 mg IV dose of the cyclooxygenase-2 inhibitor parecoxib administered to lactating women after cesarean delivery is unlikely to cause adverse effects in breastfed infants.

專題：容積二氧化碳圖用於死腔測量的原理

Special Article: Rationale of Dead Space Measurement by Volumetric Capnography

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死腔是潮氣量的一部分，因其沒有接觸到流經肺毛細血管的血液而不參與氣體交換。通常使用容積二氧化碳圖，即一種相對潮氣量作出呼出氣 CO2 的曲線來計算，它是一種評估特定通氣參數無效的床邊監測方式。如今，可通過一個完全無創性和每拍呼吸的方式，比如平均肺泡二氧化碳分壓（$PACO_2$），來測量 Bohr 死腔，而其中 $PACO_2$ 現在可以從二氧化碳描記圖直接確定。Enghoff 對玻爾公式修改的價值（使用動脈血二氧化碳分壓而不是肺泡 CO_2 因爲它是受各種肺通氣/灌注不匹配原因（從真死腔到分流）的影響。因此，玻爾和 enghoff 公式取得的結果有不同的生理意義，臨床醫生在分析病人的資料時必須意識到這種差異。本文描述了容積二氧化碳圖測量死腔的原理並討論其臨床意義以及對其的誤解。
（龔寅　譯　陳傑　校）
Dead space is the portion of a tidal volume that does not participate in gas exchange because it does not get in contact with blood flowing through the pulmonary capillaries. It is commonly calculated using volumetric capnography, the plot of expired carbon dioxide (CO_2) versus tidal volume, which is an easy bedside assessment of the inefficiency of a particular ventilatory setting. Today, Bohr's original dead space can be calculated in an entirely noninvasive and breath-by-breath manner as the mean alveolar partial pressure of CO_2 ($PACO_2$) which can now be determined directly from the capnogram. The value derived from Enghoff's modification of Bohr's formula (using $PaCO_2$ instead of $PACO_2$) is a global index of the inefficiency of gas exchange rather than a true “dead space” because it is influenced by all causes of ventilation/perfusion mismatching, from real dead space to shunt. Therefore, the results obtained

by Bohr's and Enghoff's formulas have different physiological meanings and clinicians must be conscious of such differences when interpreting patient data. In this article, we describe the rationale of dead space measurements by volumetric capnography and discuss its main clinical implications and the misconceptions surrounding it.

輸注脂肪乳劑對豬死後羅派卡因濃度的影響：對一名軍人死因的解讀

The Effect of Lipid Emulsion Infusion on Postmortem Ropivacaine Concentrations in Swine: Endeavoring to Comprehend a Soldier's Death

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背景：現在主張脂肪乳可用於局麻藥中毒時的搶救藥。但至今還沒有一項研究測量脂肪乳對死後局麻藥血清濃度的影響。
方法：本實驗中使用的是約克夏豬（n=11），進行常規監護。給實驗豬按 1.5mg/kg/min 注射羅派卡因直至死亡（心臟停搏）。輸注前及輸注中每隔 5 分鐘採集血液樣本，用於血氣分析，並通過高效液相色譜法檢測血漿游離、結合以及總的羅派卡因濃度。在平均動脈壓達到 50mmHg 時，5 只實驗豬僅給予羅派卡因，而另外 6 只豬給予羅派卡因和單劑量 20%脂肪乳（1mg/kg）的混合液。直至心臟停搏後停止輸注羅派卡因，不予任何心肺復蘇。記錄總的羅派卡因濃度以及實驗開始至死亡的時間。給予實驗豬降溫（平均體溫，死後 6 小時 25.5℃±0.8℃）從而反映停屍房的環境條件。分別在心臟停搏時，停搏後 1 小時、3 小時、6 小時採集血樣。另外，在以上各時間點上行開顱和開腹手術以獲取 1.5-3g 大腦、肺、肝臟、腎臟和肌肉組織用於分析。
結果：將對照組和脂肪乳治療組的實驗豬死後血漿羅派卡因濃度進行分析，發現脂肪乳治療組的總羅派卡因濃度（結合和未結合白蛋白的羅派卡因）以及游離羅派卡因濃度（未結合白蛋白）均顯著高於對照組（分別是 $P = 0.0094$ and $P = 0.0063$). 此外，時間對增加死後游離血漿羅派卡因濃度也有顯著作用（P=0.0095）。脂肪乳組與對照組相比，更早發生心臟停搏（P=0.0274）。組織學分析發現，脂肪乳組的實驗豬死後肺、腎臟和大腦組織中的羅派卡因的濃度明顯下降(分別是 $P = 0.0168$, $P = 0.0073$, and $P = 0.0018$)。對照組其組織中的藥物濃度沒有改變。
結論：本實驗的資料顯示，有過局麻藥心臟毒性且給予脂肪乳治療的實驗豬，其死後的血標本的檢測可能無法直接反映死後的藥物濃度。
（張婷　譯　陳傑　校）

BACKGROUND: Lipid emulsion (20%) is advocated as a rescue drug for local anesthetic toxicity. No study has measured the impact of lipid emulsion therapy on postmortem local anesthetic serum levels.
METHODS: We anesthetized Yorkshire swine ($n = 11$) and standard monitors were placed. The swine received 1.5 mg/kg/min IV ropivacaine until death (asystole). Blood samples were drawn before infusion (baseline) and at 5-minute intervals during the infusion for measurement of blood

gases and free, bound, and total serum ropivacaine concentrations via high-performance liquid chromatography. Five swine received ropivacaine only, and 6 swine received ropivacaine plus a single bolus dose of 20% lipid emulsion (1 mg/kg) when the mean arterial blood pressure reached 50 mm Hg. Ropivacaine infusions were terminated at asystole and no resuscitation was initiated. Total ropivacaine dose and time to death were recorded. The swine were cooled (mean temperature, 25.5°C ± 0.8°C at 6 hours postmortem) to reflect morgue conditions. Serum samples were drawn at asystole, 1, 3, and 6 hours postmortem for analysis. Additionally, a craniotomy and laparotomy were performed at those times to remove 1.5 to 3 g each of brain, lung, liver, kidney, and muscle for analysis.

RESULTS: Analysis of the postmortem serum ropivacaine concentrations in the control and the lipid-treated animals indicated that both the total (bound and not bound to proteins) and free (not bound to proteins) ropivacaine concentrations were significantly higher in the lipid-treated animals ($P = 0.0094$ and $P = 0.0063$, respectively). Furthermore, time had a significant effect on increasing the postmortem free ropivacaine concentrations ($P = 0.0095$). The lipid group had a statistically significant earlier onset of death (asystole) compared with the control group ($P = 0.0274$). Tissue analysis indicated that the ropivacaine concentration significantly decreased postmortem in the lung, kidney, and brain tissues of the lipid-treated animals ($P = 0.0168$, $P = 0.0073$, and $P = 0.0018$, respectively). Tissue drug concentrations in the control animals remained unchanged after death.

CONCLUSIONS: Our data show that postmortem blood samples in swine that experience local anesthetic cardiovascular collapse and are treated with lipid emulsions will result in measurements that cannot be directly extrapolated to premortem drug concentrations.

腦電圖和肌電圖變化率的增加與術中軀體反應發生率增加的關係

Increases in electroencephalogram and electromyogram variability are associated with an increased incidence of intraoperative somatic response.

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背景：BIS-腦電雙頻指數，EMG-體表肌電變化率，以及 CVI-複合變化指數是腦電和肌電變化率監測的三個新指標。我們研究增加這些指數的變化是否與術中軀體反應有關。

方法：這一多中心研究包括來自 4 個不同的中心經過篩選後的 120 名非心臟手術患者。全麻術中維持，2 個中心用丙泊酚和瑞芬，另外 2 個中心用七氟醚和瑞芬，通過調整丙泊酚和七氟醚維持 BIS 在 45-60。臨床醫生一直以來不瞭解 CVI(v2.0)，瑞芬的輸注全憑個人經驗。這次術中所有的軀體反應（包括體動，怪相，睜眼等）都被記錄。每個事件都被劃分爲連續不重疊 10 分鐘爲一段的事件。每一片段都包括體動事件或者未發生事件。對於每個階段，BIS，EMG、CVI、HR、ART 都要監測。爲有效量化區別每個在體動事件和非體動事件中的變數，我們通過計算了每個變數受試者特性曲線(ROC)的曲線下面積。最

終，我們觀察到每個體動事件發生前的 BIS，EMG，CVI 和 HR 波動的時間進程，以及每個變數能區分體動和特殊非體動事件的最早特徵。
結果：分析結果包括來自 105 例手術的 33 個體動事件和 829 個非體動事件。ROC 曲線曲線下面積計算結果 BIS 爲 0.84±0.04，EMG 爲 0.92±0.02，CVI 爲 0.89±0.03，HR 爲 0.77±0.03，ART 爲 0.68±0.05。CVI、BIS、EMG 對體動發生的敏感性高。心率只在體動發生前的幾秒鐘稍有變化。
結論：BIS、EMG、CVI 監測增加術中體動發生的預知，在 10 分鐘的體動和非體動實驗中，相比 HR 和 ART，前三者變化更明顯。進一步講，在體動發生前，相比心率的增加，CVI 增加更早，可更早提示鎮痛不足。
（韓旭譯 薛張綱校）
BACKGROUND: sBIS, the variability of the Bispectral Index (BIS), sEMG, the variability of facial electromyogram power (EMG), and the Composite Variability Index (CVI) are 3 new measures of electroencephalogram and EMG variability. CVI is a single measure of the combined variability in BIS and EMG. We investigated whether increases in these variables are associated with intraoperative somatic responses.
METHODS: This multicenter study included 120 patients undergoing elective, noncardiac surgery from 4 different sites. General anesthesia was maintained using propofol and remifentanil at 2 of the sites and sevoflurane and remifentanil at the 2 other sites. Propofol or sevoflurane was adjusted to maintain BIS between 45 and 60. Clinicians were blinded to CVI (v2.0) at all times, and remifentanil infusions were adjusted at the discretion of the clinician. The times of all intraoperative somatic events, defined as movement, grimacing, or eye opening, were recorded. Offline, the maintenance phase of each case was divided into consecutive, nonoverlapping, 10-minute segments. Segments were identified as containing a somatic event or containing no events. For each segment, mean sBIS, sEMG, and CVI and the heart rate (HR) range and mean arterial blood pressure range were calculated. To quantify how effectively each variable discriminated between somatic event segments and nonevent segments, we computed the area under the receiver operating characteristic (ROC) curve for each variable. Finally, we observed the time course of sBIS, sEMG, CVI, and the HR range before each somatic event and characterized the earliest time before the somatic event at which each variable was able to discriminate between the somatic events and a specified set of nonevents.
RESULTS: The analysis included 33 somatic event segments and 829 nonevent segments from 105 surgical cases. The areas under the ROC curve (±SE) for sBIS, sEMG, and CVI were 0.83 ± 0.04, 0.92 ± 0.02, and 0.89 ± 0.03, respectively. The areas under the ROC curve for HR range and mean arterial blood pressure range were 0.77 ± 0.03 and 0.68 ± 0.05, respectively. CVI, sBIS, and sEMG all demonstrated higher average values before upcoming somatic events when compared with nonevents. HR range only showed a difference within a few seconds before the somatic event.
CONCLUSION: sBIS, sEMG, and CVI, measures of electroencephalogram and EMG variability, increased when intraoperative somatic events occurred. sBIS, sEMG, and CVI discriminated between 10-minute segments that contained a somatic event and those segments that did not contain an event better than changes in HR and mean arterial blood pressure. Furthermore, CVI increases before somatic events began earlier than HR changes and may provide caregivers with an early warning of potentially inadequate antinociception.

通過喉電描記法評估的發音不受利多卡因局部麻醉的影響：一項前瞻性，交叉，隨機，雙盲安慰劑對照研究

Vocalization assessed by electrolaryngography is unaffected by topical lidocaine anesthesia: a prospective, crossover, randomized, double-blind placebo-controlled study.

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背景：預計氣道管理困難時，人們常常推薦使用上呼吸道局部麻醉技術。然而，有發表的報告指出實施局部麻醉導致氣道控制完全喪失，不利影響主要歸因於其對非自主呼吸道保護性反射的干擾，而粗大的運動功能本身一般被認爲是保留的。我們假設，如果運動控制受到影響，當使用局部麻醉劑時發音測量會出現量的變化
方法：我們進行了一項前瞻性，交叉，隨機，雙盲研究，對 24 名健康志願者進行發聲練習 2，同時通過數位光纖鼻內窺鏡記錄他們的聲門外觀。受檢者在不同場合用 3 種不同的試驗溶液漱口（安慰劑，2%利多卡因，4%利多卡因）和重複發聲練習和接受鼻內窺鏡檢查。聲帶之間的角度測量使用® MB-Ruler，喉動描記語音工作室® 軟體用於發音參數分析
結果：對照組和試驗組僅僅在語音品質上有顯著變化，（p =0.014）。安慰劑組和利多卡因組沒有差異
結論：雖然用局部麻醉劑漱口影響發聲，但是這與局部麻醉劑的藥理作用無關。
（賀盼譯 薛張綱校）

BACKGROUND: Topical anesthesia of the upper airway is often recommended when difficulty in airway management is anticipated. There are published reports, however, of administration of topical anesthesia resulting in complete loss of airway control. Adverse effects are mostly attributed to interference with involuntary protective airway reflexes, while gross motor function itself generally is thought to be preserved. We hypothesized that if motor control is affected, measurable quantitative changes in vocalization should follow the use of topical anesthesia
METHODS: A prospective, crossover, randomized, double-blind study was conducted, in which 24 healthy volunteers each performed 2 vocal exercises, while having their glottic appearance recorded digitally via fiberoptic nasendoscopy. Subjects gargled with 3 test solutions on separate occasions (placebo, 2% lidocaine, and 4% lidocaine) and repeated the vocal exercises and nasendoscopy. The angle between the vocal cords was measured using MB-Ruler® , and the Laryngograph Speech Studio® software was used for vocal parameter analysis。
RESULTS: The only significant changes in voice quality occurred between the control and test groups (P = 0.014). No difference could be found between the placebo and lidocaine groups.
CONCLUSIONS: Although gargling with local anesthetic affected vocalization, no pharmacological effect attributable to local anesthetic was observed.

系統回顧和薈萃分析：地塞米松對預防術後噁心和嘔吐與椎管內注射嗎啡的相關性

Dexamethasone for the Prophylaxis of Postoperative Nausea and Vomiting Associated with Neuraxial Morphine Administration: A Systematic Review and Meta-Analysis

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背景：我們進行了系統的回顧，以評估地塞米松在減少術後噁心，嘔吐（PONV）,皮膚瘙癢，加強患者在接受椎管內麻醉，椎管內注射嗎啡鎮痛的療效。
方法：我們查閱了 Medline(1966-2011 年)，科克倫中央登記冊對照試驗，文摘，Web of Science 中所有隨機對照試驗，地塞米松與安慰劑比較在預防患者接受椎管內麻醉其中包含的椎管內注射嗎啡後 PONV 和/或皮膚瘙癢的功效。術後噁心嘔吐，瘙癢，疼痛評分在 4 小時和 24 小時，止吐藥的應用，止癢，止痛的作者均獨立提取資料。
結果：八組隨機對照試驗（4 個剖腹產，4 個腹部子宮切除術）都包含在內。從這些試驗中，768 例患者被分析包括 473 名患者接受地塞米松和 295 名接受安慰劑。地塞米松的劑量範圍從 2.5 至 10mg。地塞米松減少術後噁心（相對風險[95%]可信區間=0.57[0.45,0.72]），嘔吐（相對風險[95%]可信區間=0.56[0.43,0.72]），以及使用止吐藥治療的發生率（相對風險[95%]可信區間=0.47[0.36,0.61]）與安慰劑相比。沒有顯示其止吐效果與劑量的關係。地塞米松也減少了 24 小時視覺類比疼痛評分（11 種程度[0-10]）（平均差[95%]可信區間=-0.30[-0.46,-0.13]）和鎮痛藥的使用率（相對風險[95%]可信區間=0.72[0.52,0.98]）。地塞米松並未減少瘙癢的發生率（相對風險[95%]可信區間=0.98[0.84,1.15]）。漏斗圖和艾格檢測揭示了主要結果存在出版偏倚的證據。
結論：地塞米松是一種有效的止吐劑用於接受剖腹產，腹式子宮切除進行椎管內注射嗎啡的患者。此外，增強止吐和預防術後鎮痛作用的劑量與安慰劑相比較過。然而，地塞米松不能有效預防椎管內注射嗎啡引起的瘙癢。
（胡曉清譯 薛張綱校）
BACKGROUND: We performed a systematic review to assess the efficacy of dexamethasone in reducing postoperative nausea, vomiting (PONV), pruritus, and enhancing postoperative analgesia in patients receiving neuraxial anesthesia with neuraxial morphine.
METHODS: We searched Medline (1966-2011), the Cochrane Central Register of Controlled Trials, EMBASE, and Web of Science for all randomized controlled trials comparing dexamethasone with placebo for the prevention of PONV and/or pruritus in patients receiving neuraxial morphine as part of a neuraxial anesthetic technique. Data were extracted independently by the authors on the incidence of PONV, pruritus, pain scores at 4 and 24 hours, and use of rescue antiemetics, antipruritics, and analgesics.
RESULTS: Eight randomized controlled trials (4 cesarean deliveries, 4 total abdominal hysterectomies) were included. From these trials, 768 patients were analyzed with 473 receiving dexamethasone and 295 receiving placebo. The doses of dexamethasone investigated ranged from 2.5 to 10 mg. Dexamethasone reduced the incidence of postoperative nausea (relative risk, RR [95% confidence interval, CI] = 0.57 [0.45, 0.72]), vomiting (RR [95% CI] = 0.56 [0.43, 0.72]), and the use of rescue antiemetic therapy (RR [95% CI] = 0.47 [0.36, 0.61]) compared with placebo. There was no evidence of dose responsiveness with respect to its antiemetic effect. Dexamethasone also reduced 24-hour visual analog pain scores (measured on an 11-point scale [0-10]) (mean difference [95% CI] = -0.30 [-0.46, -0.13]) and the use of rescue analgesics (RR [95% CI] = 0.72 [0.52, 0.98]). Dexamethasone did not reduce the incidence of pruritus (RR [95% CI] = 0.98 [0.84, 1.15]). Examination of the funnel plots and Egger's test revealed evidence of publication bias in the primary outcomes.

CONCLUSION: Dexamethasone is an effective antiemetic for patients receiving neuraxial morphine for cesarean delivery and abdominal hysterectomy. In addition, the doses used for antiemetic prophylaxis enhanced postoperative analgesia compared with placebo. However, dexamethasone was not effective for the prophylaxis against neuraxial morphine-induced pruritus.

α5γ 酪氨酸（GABA）A 型受體恢復了全麻後認知記憶

Inhibition of α5 γ-Aminobutyric Acid Type A Receptors Restores Recognition Memory After General Anesthesia.

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背景：全麻所致認知障礙持續時間比依照其藥理學特性所預期的長得多。這些麻醉後認知障礙的細胞學機制仍然不清楚。GABA A 型受體是大多麻醉藥的主要作用靶點。尤其是 α5 亞型 GABA 受體參與了麻醉後急性記憶阻斷及術後早期記憶缺失的形成。本研究中，首先我們探索了工作記憶及短期認知記憶在應用異氟醚後是否可以修復；其次研究了異氟醚使用後引起的記憶缺失能否通過抑制 α5 亞型 GABA 受體而得到翻轉；同時我們也研究了 α5 亞型 GABA 受體表達對使用異氟醚處理後記憶缺失的形成是否是必要的。
方法：野生型及 GABA 受體 α5 亞型敲除小鼠用異氟醚（1.3%，1 mac）或七氟醚（2.3%， 1mac）或對照氣體處理一小時。記憶評估用一種新型的目標識別任務。小鼠在吸入異氟醚麻醉結束後 24 小時或 72 小時進行識別任務訓練。工作記憶和短時記憶在訓練後 1 分鐘和 1 小時分別進行檢測。爲了測定抑制 α5 亞型 GABA 受體是否可翻轉記憶缺失，我們對經異氟醚處理後 23.5 及小時或行爲訓練前 30 分鐘用 L-655，708 對小鼠進行預處理。
結果：野生型小鼠在異氟醚處理後 24 小時，其解析度下降，證明其短時記憶功能修復。相反地，工作記憶在異氟醚處理後不能修復。短時記憶在經 L-655，708 處理後可完全被翻轉。72 小時候後，短時記憶缺失可自行修復。Gabra5-/-小鼠在異氟醚處理後不表現出短時記憶缺損。七氟醚麻醉後 24 小時也可導致記憶缺損，表現爲解析度下降。
結論：吸入麻醉藥會導致順時認知記憶的缺失，通過這種“證明-概念式”的研究發現 α5 亞型 GABA 受體對麻醉後認知記憶缺失的形成是必須的，同時，這些受體也是氣體消除後恢復認知記憶的作用靶點。
(李麗紅譯 薛張綱校)

BACKGROUND: General anesthetics cause cognitive deficits that persist much longer than would be expected on the basis of their pharmacokinetics. The cellular mechanisms underlying these postanesthetic cognitive deficits remain unknown. γ-Aminobutyric acid type A (GABA(A)) receptors are principal targets for most anesthetics. In particular, theC receptor subtype has been implicated in acute memory blockade during anesthesia and memory deficits in the early postoperative period. We first sought to determine whether working memory and short-term recognition memory are impairedafter isoflurane anesthesia. The second aim of the study was to determine whether memory deficits after isoflurane can be reversed by inhibiting

α5GABA(A) receptors. We also sought to determine whether the expression of α5GABA(A) receptorsis necessary for the development of memory dysfunction after isoflurane. Lastly, the effect of sevoflurane on memory was studied.
METHODS: Wild-type and α5GABA(A) receptor null-mutant (Gabra5-/-) mice were treated with isoflurane (1.3%; 1 minimum alveolar concentration [MAC]) or sevoflurane (2.3%; 1 MAC) or vehicle gas for 1 hour. Memory performance was assessed with a novel object recognition task. Mice were trained on the recognition task either 24 hours or 72 hours after isofluraneanesthesia. Working memory and short-term memory were tested 1 minute and 1 hour after training, respectively. To determine whether inhibition of α5GABA(A) receptors reverses memory deficits, we treated a subset of mice with L-655,708 (0.35 mg/kg or 0.7 mg/kg) 23.5 hours after isoflurane and 30 minutes before behavioral training.
RESULTS: Short-term memory was impaired in wild-type mice 24 hours after isoflurane as evidenced by a decrease in the discrimination ratio (control 0.66 ± 0.03 vs isoflurane 0.51 ± 0.03, P = 0.0005). In contrast, working memory was not impaired by isoflurane (control 0.68 ± 0.05 vs isoflurane 0.67 ± 0.04, P = 0.979). The deficit in short-term memory was fully reversed by L-655,708 (effect of isoflurane × L-655,708, F(2,102) = 3.59, P = 0.032; isoflurane 0.51 ± 0.03 vs isoflurane + L-655,708 at 0.35 mg/kg 0.67 ± 0.03, P < 0.05). By 72 hours, the deficits in short-term memory resolved spontaneously (control 0.65 ± 0.05 vs isoflurane 0.60 ± 0.04, P = 0.441). Gabra5-/- mice showed no short-term memory deficits 24 hours after isoflurane (effect of isoflurane F(1,47) = 0.375, P = 0.544). Sevoflurane also caused memory deficits 24 hours after anesthesia, as evidence by a reduction in the discrimination ratio (control 0.63 ± 0.02 vs sevoflurane 0.53 ± 0.03, P = 0.039).
CONCLUSIONS: Inhalational anesthetics cause deficits in anterograde recognition memory. This proof-of-concept study shows that α5GABA(A) receptors are necessary for the development of postanesthetic deficits in recognition memory and that these receptors can be targeted to restore memory even after the anesthetic has been eliminated.

沃克 256 細胞癌時針對足三裡穴位的電針刺療法對癌症疼痛的作用和瞬態感受器陽離子電壓通道的解釋

The Effects of Electroacupuncture at the ST36 (Zusanli) Acupoint on Cancer Pain and Transient Receptor Potential Vanilloid Subfamily 1 Expression in Walker 256 Tumor-Bearing Rats.

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背景：一些研究已經闡明了瞬態感受器陽離子通道的表達在形成癌症疼痛起了重要的作用。電針刺療法是一種有效的減弱不同種類疼痛的方法，比如炎症性疼痛，神經性疼痛和腫瘤疼痛。這些研究中，我們調查了電針刺療法對因注射了癌症細胞而患有癌症疼痛的 256 只老鼠的作用和腫瘤通過瞬態感受器陽離子電壓通道達到多節段的背根神經節。

方法：老鼠被隨機的分爲 4 組：接種非腫瘤細胞組（對照組，n=8）；接種沃克 256 癌細胞組（腫瘤組，n=8）；接種沃克 256 細胞癌假電刺治療組（假針刺治療組，n=8）；接種沃克 256 細胞癌予以電針刺療法組（電針刺治療組，n=8）。定義了預熱的時間、機械靈敏度和自發的疼痛反應的行爲。另外，在多節段的背根神經節中瞬態感受器陽離子電壓通道的解釋已經被免疫印跡和相當數量的即時的聚合酶鏈反應印證了。
結果：注射腫瘤細胞的老鼠減少了爪子退縮的閾值，增加了自發的疼痛反應的行爲，誘發了嚴重的溫度痛覺過敏，通過對足三裡的電針刺療法使之減少（2 赫茲，0.3ms，<=1 毫安培）。在癌症疼痛模型中瞬態感受器陽離子電壓通道的 mRNA 和多節段的背根神經節中的蛋白表達升高，對足三裡學位的電針刺療法中和了相應多節段的背根神經節中的瞬態感受器陽離子電壓通道的表達。
結論：對足三裡的電針刺療法能夠減弱腫瘤引起的疼痛，至少在一部分，通過抑制多節段的背根神經節中瞬態感受器陽離子電壓通道中的 mRNA 和蛋白的上調。
（孫莉萍譯 薛張綱校）

BACKGROUND: Several studies have addressed the expression of transient receptor potential vanilloid subfamily 1(TRPV1) playing an important role in the generation of cancer pain. Electroacupuncture (EA) is an effective method of acupuncture shown to attenuate different kinds of pain such as inflammatory, neuropathic, and cancer. In this study, we investigated the effect of EA on cancer pain caused by intraplantar injection of Walker 256 carcinoma cells and cancer-driven TRPV1 expression in the dorsal root ganglions (DRGs).
METHODS: Rats were randomly divided into 4 groups: the nontumor cell inoculation group (normal control, n = 8); Walker 256 carcinoma cell inoculation group (tumor control, n = 8); sham point electrical stimulation treatment with Walker 256 carcinoma cell inoculation group (SES, n = 8); EA treatment with Walker 256 carcinoma cell inoculation group (EA, n = 8). The time courses of thermal, mechanical sensitivity, and spontaneous nocifensive behavior were determined. In addition, TRPV1 expression in DRGs was observed by quantitative real-time polymerase chain reaction and Western blotting.
RESULTS: Injection of cancer cells decreased the paw withdrawal threshold, increased spontaneous nocifensive behavior, and induced significant thermal hyperalgesia that was attenuated by EA at the ST36 acupoint (2 Hz, 0.3 ms, ≤1 mA). TRPV1 mRNA and protein in DRGs were upregulated in the cancer pain model, and EA at ST36 acupoint counteracted the cancer-driven upregulation of TRPV1 expression in the corresponding DRGs.
CONCLUSIONS: EA at ST36 could attenuate cancer-induced pain, at least in part, through suppressing TRPV1 mRNA and protein upregulation in the DRGs.

靜脈注射脂肪乳只是最低限度的影響血漿中布比卡因和甲呱卡因的分佈，並不會提高豬中毒的復蘇

Intravenous lipid emulsion only minimally influences bupivacaine and mepivacaine distribution in plasma and does not enhance recovery from intoxication in pigs.

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背景：目前報導的成功使用脂肪乳劑治療局麻藥中毒被認爲是由於局麻藥的脂質封存。然而由於缺乏療效對比試驗尚有其他可能的機制未被發現。我們研究脂質輸注對 2 種不同脂溶性局麻藥—布比卡因和甲帕卡因的血漿濃度和心血管效應。

方法：分佈比卡因組（n=20）和甲呱卡因組（n=20），從已麻醉的豬（1%異氟醚，吸入氧濃度 21%）中心靜脈分別給予布比卡因或甲呱卡因（直至平均動脈壓下降至基線的 50%.停止異氟醚，調節吸入氧濃度至 100%。兩組各有 10 只豬分別注射 20%脂質溶劑 (ClinOleic®)和林格氏溶液，最初 29 分鐘注速爲 0.25ml/kg/min，最後 1 分鐘調至 1.5ml/kg/min。五隻額外的豬被注入布比卡因和脂肪乳® 。反復血液樣本測量局麻藥總濃度和不含脂局麻藥濃度。

結果：脂質組和林格氏液組二者無論血漿總的局麻藥濃度或不含脂局麻藥濃度均沒有顯著差異。然而，脂質組總布比卡因濃度在 20 分鐘和 30 分鐘時分別增加 21%和 23%（P=0.016，無 Bonferroni 校正）。脂質組和林格氏液組血流動力學的恢復速率和心電圖變數也無明顯差異。脂質組中布比卡因中毒組的平均動脈壓分別在注射後 10 分鐘和 15 分鐘比林格氏液組高出 16 mmHg 和 15mmHg（P=0.021，無 Bonferroni 校正）脂肪乳針對布比卡因總濃度和不含脂濃度對於復蘇無明顯增強作用。

結論：脂溶劑既不可測量局麻藥清除效果，也不會增加恢復速率（如以血流動力學參數做參考）

（楊琰譯 薛張綱校）

BACKGROUND: The reported successful use of IV lipid emulsions in local anesthetic intoxications is thought to be due to lipid sequestration of local anesthetics. However, controlled efficacy studies were lacking, and other mechanisms of action have also been suggested. We investigated the effect of lipid infusion on plasma concentrations and cardiovascular effects of 2 local anesthetics differing in lipophilicity, bupivacaine, and mepivacaine.

METHODS: Bupivacaine (n = 20) or mepivacaine (n = 20) was infused into a central vein of anesthetized (isoflurane 1%, Fio(2) 0.21) pigs until mean arterial blood pressure decreased to 50% from baseline. Isoflurane was discontinued and Fio(2) was increased to 1.0. Ten pigs in each local anesthetic group were treated with 20% lipid emulsion (ClinOleic®), and 10 pigs with Ringer's solution: 1.5 mL/kg in 1 minute followed by an infusion of 0.25 mL ·kg(-1) ·min(-1) for 29 minutes. Five additional pigs were infused bupivacaine and Intralipid® . Total and nonlipid-bound local anesthetic concentrations were determined from repeated blood samples.

RESULTS: There were no overall differences in total or nonlipid-bound plasma local anesthetic concentrations between the lipid and Ringer's groups. However, plasma median total bupivacaine concentration was 21% and 23% higher at 20 and 30 minutes, respectively, in the lipid group (P = 0.016 without Holm-Bonferroni correction). There was also no overall difference between lipid and Ringer's groups in the rate of recovery of hemodynamic and electrocardiographic variables. Median mean arterial blood pressure in the lipid group with bupivacaine intoxication was 16 mm Hg and 15 mm Hg higher than in the corresponding Ringer's group at 10 and 15 minutes, respectively (P = 0.016 and P = 0.021, respectively, without Holm-Bonferroni correction). Intralipid® also caused no difference between total plasma and nonlipid-bound concentrations of bupivacaine with no apparent enhancement of recovery.

CONCLUSIONS: Lipid emulsion neither had any measurable effect on the disposition of the studied local anesthetics in plasma, nor did it improve the rate of recovery from intoxication by either local anesthetic as measured by hemodynamic variables.

血栓彈性描記器和血栓彈性檢測器全血纖維蛋白凝塊試驗的比較

Comparison of Whole Blood Fibrin-Based Clot Tests in Thrombelastography and Thromboelastometry

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背景：基於纖維蛋白的血凝塊硬度測量，功能性纖維蛋白原（FF）血栓彈性描記法測量的是最大振幅（MA），FIBTEM 血栓彈性檢測法測量的是凝塊的最大硬度（MCF）。方法儀器的不同可能有臨床意義。我們的目的是通過標準（血栓彈性描記器[TEG®]測量 FF; 血栓彈性檢測器[ROTEM®]測量 FIBTEM) 和交叉 (ROTEM® 測量 FF; TEG® 測量 FIBTEM)比較凝塊硬度參數。

方法：對採集健康志願者的全血樣本進行血栓彈性描記器和血栓彈力檢測儀的分析。分別檢測不經過處理的樣本，以及使用氯化鈉溶液逐步稀釋過的樣本（稀釋 20%、40%和 60%）。並且還評估了在體外加入藥物（肝素、魚精蛋白、氨甲環酸）後和使用羥乙基澱粉、明膠、氯化鈉和白蛋白稀釋 50%後的樣本。

結果：不考慮儀器的不同，FF 比 FIBTEM 測量值高；不考慮方法的不同，TEG® 測量值比 ROTEM® 高。加了除了肝素 400U/公斤體重以外的所有藥物後，FF MA 都明顯比 FIBTEM MCF 高（$P<0.05$），FIBTEM MCF 很大程度上沒有變化。FF MA 在加入大劑量的肝素後明顯減小（$P = 0.04$），加入魚精蛋白後有部分恢復。採用羥乙基澱粉、白蛋白和明膠稀釋 50%後，FIBTEM MCF 和 FF MA 都下降了超過 50%。

結論：這些結果顯示了在使用 TEG® and ROTEM® 通過 FF 和 FIBTEM 法測量纖維蛋白爲基礎的凝血情況的不同。針對糾正凝血狀態所做的及時處理可能會被檢測方法和儀器所影響。缺少對於 FF 分析法的資料。

（安光惠 譯 馬皓琳 李士通 校）

BACKGROUND: Fibrin-based clot firmness is measured as maximum amplitude (MA) in the functional fibrinogen (FF) thrombelastographic assay and maximum clot firmness (MCF) in the FIBTEM thromboelastometric assay. Differences between the assays/devices may be clinically significant. Our objective was to compare clot firmness parameters through standard (FF on a thrombelastography device [TEG®]; FIBTEM on a thromboelastometry device [ROTEM®]) and crossover (FF on ROTEM® ; FIBTEM on TEG®) analyses.

METHODS: Whole-blood samples from healthy volunteers were subjected to thrombelastography and thromboelastometry analyses. Samples were investigated native and following stepwise dilution with sodium chloride solution (20%, 40%, and 60% dilution). Samples were also assessed after in vitro addition of medications (heparin, protamine, tranexamic acid) and 50% dilution with hydroxyethyl starch, gelatin, sodium chloride, and albumin.
RESULTS: FF produced higher values than FIBTEM, regardless of the device, and TEG® produced higher values than ROTEM® , regardless of the assay. With all added medications except heparin 400 U/kg bodyweight, FF MA remained significantly higher ($P < 0.05$) than FIBTEM MCF, which was largely unchanged. FF MA was significantly reduced ($P = 0.04$) by high-dose heparin and partially restored with protamine. Fifty percent dilution with hydroxyethyl starch, albumin, and gelatin decreased FIBTEM MCF and FF MA by >50%.
CONCLUSIONS: These results demonstrate differences when measuring fibrin-based clotting via the FF and FIBTEM assays on the TEG® and ROTEM® devices. Point-of-care targeted correction of fibrin-based clotting may be influenced by the assay and device used. For the FF assay, data are lacking.

對兒科患者應用生物電阻抗心動描計儀[PhysioFlow®]進行無創性心排量監測與核磁共振顯像相比精確性較差

Poor Accuracy of Noninvasive Cardiac Output Monitoring Using Bioimpedance Cardiography [PhysioFlow®] Compared to Magnetic Resonance Imaging in Pediatric Patients

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背景：在麻醉中確認低心排出量（CO）狀態很重要，因爲術前優化血流動力學可能改善手術預後。精確的心排量即時測量會對優化“目標-嚮導”治療有意義。我們試圖對合併或不合併心臟疾病的兒科患者在行麻醉下磁共振成像（MRI）時評估應用生物電阻抗心動描記儀(PhysioFlow® , NeuMeDx, Bristol, PA)進行 CO 檢測的可靠性和準確性。
方法：所有獲得允許的進行麻醉下心臟 MRI 的病人均入組。在麻醉平衡≥10 分鐘後，6 個 PhysioFlow 電極置於患者的胸部進行 10 分鐘連續即時監測。資料記錄 15 秒時間然後離線計算均值得到 CO。相位差 MRI 對上腔靜脈流量和升、降主動脈流量的測量由一個在右肺動脈水準通過所有 3 條血管的單一成像平面做出。兩種 CO 測量均引入體表面積。用於兩種測量的麻醉技術相同。用 Bland-Altman 分析評估一致性。
結果：31 名患者入組，其中 23 例獲得分析。年齡中位數爲 2.8 歲（範圍：0.02–8.02 歲），體表面積中位數爲 0.54 m^2 (範圍：0.21–1.00 m^2)。23 例中有 11 例（48%）爲男性。患者分爲單心室 6/23 例（26%）；雙心室伴分流 3/23 例（13%）；雙心室不伴分流 10/23（43%）；非器質性心臟病 4/23 例（17%）。平均偏差爲−0.34 ± 1.50 L/min/m^2 ($P = 0.29$)。一致性的 95%可信區間爲−3.21 to +2.69 L/min/ m^2。只有 8/23 例結果（35%）在 20%以內，14/23 例結果（61%）在 30%之內。

結論：PhysioFlow 的性能在此人群中不夠精確。在此設備被推薦給兒科患者常規臨床使用之前，尚需矯正演算法和進一步的檢驗。
（瞿亦楓 譯 馬皓琳 李士通 校）
BACKGROUND: Identification of low cardiac output (CO) states in anesthesia is important because preoperative hemodynamic optimization may improve outcome in surgery. Accurate real-time CO measurement would be useful in optimizing "goal-directed" therapy. We sought to evaluate the reliability and accuracy of CO measurement using bioimpedance cardiography (PhysioFlow® , NeuMeDx, Bristol, PA) in pediatric patients with and without cardiac disease undergoing anesthesia for magnetic resonance imaging (MRI).
METHODS: All consenting patients undergoing anesthesia for cardiac MRI were enrolled. After equilibration of anesthesia for ≥10 minutes, 6 PhysioFlow electrodes were applied to the patient's chest for continuous real-time monitoring for 10 minutes. Data were stored in 15-second epochs and later averaged offline to obtain CO. Phase contrast MRI measurements of flow volumes in the superior vena cava and ascending and descending aorta were made from a single imaging plane through all 3 vessels at the level of the right pulmonary artery. Both CO measurements were indexed to body surface area. The anesthetic technique was the same for both measurements. Agreement was assessed using Bland-Altman analysis.
RESULTS: Thirty-one patients were enrolled and 23 were analyzed. The median age at study was 2.8 years (range, 0.02–8.02 years) and median body surface area was 0.54 m^2 (range, 0.21–1.00 m^2). Eleven of the 23 patients (48%) were males. Patients were grouped into those with univentricular physiology, 6 of 23 (26%); biventricular physiology with shunt, 3 of 23 (13%); biventricular without shunt, 10 of 23 (43%); and no structural heart disease, 4 of 23 (17%). The mean bias was −0.34 ± 1.50 L/min/m^2 (P = 0.29). The 95% limits of agreement were −3.21 to +2.69 L/min/m^2. Only 8 of 23 measurements (35%) were within 20% and 14 of 23 measurements (61%) were within 30% of each other.
CONCLUSION: PhysioFlow performance was not sufficiently accurate in this population. Modifications of the algorithm and further testing are required before this device can be recommended for routine clinical use in pediatric patients.

麻醉患者安全基金 25 歲：在安全性上的開創性成功，25 周年引發的思考和期待

The Anesthesia Patient Safety Foundation at 25: A Pioneering Success in Safety, 25th Anniversary Provokes Reflection, Anticipation

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麻醉患者安全基金（APSF）創立於 1985 年。它的創始人創造"患者安全"這個詞意在現代公共使用並且創立了第一個患者安全性群組織，引發了一場現在在所有醫療機構普遍存在的運動。"沒有患者應當受到麻醉的損害"作爲他們的美好願景，APSF 組織不知疲倦地在超過四分之一世紀的時間裡工作，通過它的廣泛時事通訊、其節目及其介紹推動安全教育和交流。APSF 的廣泛研究資助項目已支持了許多項目，這些項目導致重要的安全進步，尤其是在發展高保真模特模擬研究和教學工具。憑藉其開創性的合作，APSF 在納入各類麻醉專家、安全方面的科學家、製藥和設備製造商、監管機構、責任保險公司及外科醫生的人才和資源上非常特別。具體的警示、活動、討論和專案多年來都已針對于許多安全問題和危險，從在 1986 年的最小的術中監測，一路到沙灘椅位的腦灌注壓、手術室用

藥錯誤和在 2010 年非常流行的手術室消防安全 DVD；名單非常長和廣泛。APSF 作爲一種後續的患者安全性群組織的模範和啓示，並且廣泛認識到對麻醉管理的安全性有顯著的積極影響。認識到這項工作還沒有結束，該系統、組織和設備仍會有時失敗，基本可以預防的人爲錯誤仍然有時發生，以及在麻醉實踐中的"產量壓力"威脅過往的安全收益，APSF 堅定地獻身於並繼續努力工作於已確立的原則和新的病人安全原則。
（劉朝輝譯，馬皓琳，李士通校）

The Anesthesia Patient Safety Foundation (APSF) was created in 1985. Its founders coined the term "patient safety" in its modern public usage and created the very first patient safety organization, igniting a movement that is now universal in all of health care. Driven by the vision "that no patient shall be harmed by anesthesia," the APSF has worked tirelessly for more than a quarter century to promote safety education and communication through its widely read *Newsletter*, its programs, and its presentations. The APSF's extensive research grant program has supported a great many projects leading to key safety improvements and, in particular, was central in the development of high-fidelity mannequin simulation as a research and teaching tool. With its pioneering collaboration, the APSF is unique in incorporating the talents and resources of anesthesia professionals of all types, safety scientists, pharmaceutical and equipment manufacturers, regulators, liability insurance companies, and also surgeons. Specific alerts, campaigns, discussions, and projects have targeted a host of safety issues and dangers over the years, starting with minimal intraoperative monitoring in 1986 and all the way up to beach-chair position cerebral perfusion pressure, operating room medication errors, and the extremely popular DVD on operating room fire safety in 2010; the list is long and expansive. The APSF has served as a model and inspiration for subsequent patient safety organizations and has been recognized nationally as having a dramatic positive impact on the safety of anesthesia care. Recognizing that the work is not over, that systems, organizations, and equipment still at times fail, that basic preventable human errors still do sometimes occur, and that "production pressure" in anesthesia practice threatens past safety gains, the APSF is firmly committed and continues to work hard both on established tenets and new patient safety principles.

對小兒心臟驟停豬模型的無創自動調節監測

Noninvasive Autoregulation Monitoring in a Swine Model of Pediatric Cardiac Arrest

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背景：復蘇後腦血管自動調節在小兒心跳驟停實驗模型中尚未被充分研究。此外，正在研究發展中的利用近紅外光譜(NIRS)進行自動調節監控的無創檢測方法將在小兒心跳驟停後神經保護作用的血流動力學管理方面有指導性的臨床意義。筆者驗證了自動調節的低限

（LLA）將在心跳驟停復蘇後的第一天和第二天之間將導致更高的動脈血壓，並且 LLA 可以通過 NIRS 推導出的小兒心跳驟停豬模型的自動調節指數進行檢測的假設。筆者同時驗證過心跳驟停後高血壓時的自動調節功能將會受損的假設。

方法： 通過經低氧窒息心跳驟停並且復蘇 1 天（n=8）或 2 天（n=8）或者經假手術恢復 2 天（n=8）的新生小豬獲得 LLA 的資料。通過一個單獨的經過低氧缺血心跳驟停（n=5）或者假手術恢復 2 天（n=5）的小豬佇列研究高血壓的自動調節。恢復期後，小豬被再次麻醉，通過標準鐳射多普勒血流儀和由 NIRS 推導出的自動調節指數（腦血氧飽和度[COx]和血紅蛋白含量[HVx]指數）檢測自動調節。通過給放置在下腔靜脈的球囊導管充氣以降低血壓來測定 LLA。通過給主動脈球囊導管充氣來測定高血壓過程中的自動調節。

結果：假手術組和心跳驟停後恢復 1 或 2 天組小豬的 LLA 近似。 近紅外光譜派生指數能夠精確檢測出由鐳射多普勒血流儀測定出的 LLA。腦血氧飽和度指數的接收操作特徵型曲線的曲線下面積在心跳驟停後第一天和第二天分別爲 0.91 和 0.92。血紅蛋白容量指數曲線下面積在分別的時間點爲 0.92 和 0.89。在誘導的高血壓中，靜態自動調節率，定義爲腦血管阻力的變化百分率除以腦灌注壓的變化百分比，在心跳驟停組和假手術組無統計學差異。心跳驟停恢復後的 2 天內，小豬表現出了神經行爲缺陷和組織學神經元損傷。

結論： 在有明確腦損傷的小兒缺血缺氧性心跳驟停小豬模型中，LLA 在復蘇後第一天和第二天無差異。近紅外光譜派生指數和鐳射多普勒血流儀相比，在相應時間點能精確地檢測 LLA。在高血壓時自動調節仍發揮功能。

（許辛 譯 馬皓琳 李士通校）

BACKGROUND: Cerebrovascular autoregulation after resuscitation has not been well studied in an experimental model of pediatric cardiac arrest. Furthermore, developing noninvasive methods of monitoring autoregulation using near-infrared spectroscopy (NIRS) would be clinically useful in guiding neuroprotective hemodynamic management after pediatric cardiac arrest. We tested the hypotheses that the lower limit of autoregulation (LLA) would shift to a higher arterial blood pressure between 1 and 2 days of recovery after cardiac arrest and that the LLA would be detected by NIRS-derived indices of autoregulation in a swine model of pediatric cardiac arrest. We also tested the hypothesis that autoregulation with hypertension would be impaired after cardiac arrest.

METHODS: Data on LLA were obtained from neonatal piglets that had undergone hypoxic–asphyxic cardiac arrest and recovery for 1 day (n = 8) or 2 days (n = 8), or that had undergone sham surgery with 2 days of recovery (n = 8). Autoregulation with hypertension was examined in a separate cohort of piglets that underwent hypoxic–asphyxic cardiac arrest (n = 5) or sham surgery (n = 5) with 2 days of recovery. After the recovery period, piglets were reanesthetized, and autoregulation was monitored by standard laser-Doppler flowmetry and autoregulation indices derived from NIRS (the cerebral oximetry [COx] and hemoglobin volume [HVx] indices). The LLA was determined by decreasing blood pressure through inflation of a balloon catheter in the inferior vena cava. Autoregulation during hypertension was evaluated by inflation of an aortic balloon catheter.

RESULTS: The LLAs were similar between sham-operated piglets and piglets that recovered for 1 or 2 days after arrest. The NIRS-derived indices accurately detected the LLA determined by laser-Doppler flowmetry. The area under the curve of the receiver operator characteristic curve for cerebral oximetry index was 0.91 at 1 day and 0.92 at 2 days after arrest. The area under the curve for hemoglobin volume index was 0.92 and 0.89 at the respective time points. During

induced hypertension, the static rate of autoregulation, defined as the percentage change in cerebrovascular resistance divided by the percentage change in cerebral perfusion pressure, was not different between postarrest and sham-operated piglets. At 2 days recovery from arrest, piglets exhibited neurobehavioral deficits and histologic neuronal injury.
CONCLUSIONS: In a swine model of pediatric hypoxic–asphyxic cardiac arrest with confirmed brain damage, the LLA did not differ 1 and 2 days after resuscitation. The NIRS-derived indices accurately detected the LLA in comparison with laser-Doppler flow measurements at those time points. Autoregulation remained functional during hypertension.

利多卡因延期治療減少脂多糖和干擾素γ刺激後引起的小鼠微神經膠質細胞損傷和細胞因數的產生

Delayed Treatment with Lidocaine Reduces Mouse Microglial Cell Injury and Cytokine Production After Stimulation with Lipopolysaccharide and Interferon γ

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背景：神經炎症反應對於幾乎所有的獲得性神經性疾病都是一個重要的病理過程。微神經膠質細胞在神經性炎症中扮演著重要角色。利多卡因是一種有抗炎作用的局麻藥，我們測定了其是否有保護微神經膠質細胞並減少啓動的微神經膠質細胞產生細胞因數的作用。
方法：小鼠微神經膠質培養基中加入或不加入 1 μg/mL 脂多糖和 10 U/mL 干擾素 γ (IFNγ) 培養 24 小時，在脂多糖和干擾素刺激開始後 2、3 或 4 小時時開始加入或不加入利多卡因作用一小時。細胞接受脂多糖和干擾素 γ 刺激 24 小時後檢測乳酸脫氫酶的釋放和細胞因數的產生。
結果：利多卡因呈劑量依賴性地減少脂多糖和干擾素 γ 介導的微神經膠質細胞損傷可以通過乳酸脫氫酶的釋放來衡量。這一結果在利多卡因爲 2 μg/mL 時較明顯（單獨刺激和存在利多卡因下刺激時分別爲 30.3% ± 5.8%和 23.1% ± 9.7%，n = 18, P = 0.025）。在脂多糖和干擾素 γ 刺激開始後 2、3 或 4 小時應用利多卡因減少了細胞損傷。利多卡因這一效應不受線粒體 K_{ATP} 通道抑制劑 5-羥癸酸鹽的影響。QX314 是一種永久性帶電荷的利多卡因類似物，通常不能通過質膜，與利多卡因一樣可以減少脂多糖和干擾素 γ 介導的微神經膠質細胞損傷。QX314 同時可以減少刺激引起的白介素-1 β 的產生。
結論：利多卡因的延期治療保護微神經膠質細胞並減少這些細胞產生的細胞因數。這種效應可能與細胞表面的作用位點有關。
（張怡 譯 馬皓琳 李士通校）

BACKGROUND: Neuroinflammation is an important pathological process for almost all acquired neurological diseases. Microglial cells play a critical role in neuroinflammation. We determined whether lidocaine, a local anesthetic with anti-inflammatory property, protected microglial cells and attenuated cytokine production from activated microglial cells.
METHODS: Mouse microglial cultures were incubated with or without 1 μg/mL lipopolysaccharide and 10 U/mL interferon γ (IFNγ) for 24 hours in the presence or absence of lidocaine for 1 hour started at 2, 3, or 4 hours after the onset of lipopolysaccharide and IFNγ stimulation. Lactate dehydrogenase release and cytokine production were determined after the cells were stimulated by lipopolysaccharide and IFNγ for 24 hours.

RESULTS: Lidocaine dose-dependently reduced lipopolysaccharide and IFNγ-induced microglial cell injury as measured by lactate dehydrogenase release. This effect was apparent with lidocaine at 2 μg/mL (30.3% ± 5.8% and 23.1% ± 9.7%, respectively, for stimulation alone and the stimulation in the presence of lidocaine, $n = 18$, $P = 0.025$). Lidocaine applied at 2, 3, or 4 hours after the onset of lipopolysaccharide and IFNγ stimulation reduced the cell injury. This lidocaine effect was not affected by the mitochondrial K_{ATP} channel inhibitor 5-hydroxydecanoate. Similar to lidocaine, QX314, a permanently charged lidocaine analog that usually does not permeate through the plasma membrane, reduced lipopolysaccharide and IFNγ-induced microglial cell injury. QX314 also attenuated the stimulation-induced interleukin-1β production.
CONCLUSIONS: Delayed treatment with lidocaine protects microglial cells and reduces cytokine production from these cells. These effects may involve action site(s) on the cell surface.

脂質、腎上腺素及其聯合應用在離體大鼠心臟中逆轉布比卡因引起的心臟停搏作用的比較

The Comparative Effects of Lipid, Epinephrine, and Their Combination in the Reversal of Bupivacaine-Induced Asystole in the Isolated Rat Heart

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背景：治療布比卡因引起的心臟毒性作用時，脂質與腎上腺素合用的效果究竟優於還是劣于其中任一藥物單獨應用，至今仍不清楚。我們比較了脂質、腎上腺素以及兩種藥物聯合應用在離體大鼠心臟模型中逆轉布比卡因引起的心臟停搏的作用。並且測定了這三種治療方案對心肌組織中布比卡因含量的影響。
方法：切取雄性 S-D 大鼠的心臟，並在非再迴圈的 Langendorff 灌流裝置中逆向灌流。灌注 100μmol/L 布比卡因直到心臟停搏後 3 分鐘。隨後，脂質組灌注 2%脂質和 30μmol/L 布比卡因混合物；腎上腺素組灌注 0.15μg/mL 腎上腺素和 30μmol/L 布比卡因混合物；聯合組灌注 2%脂質、0.15μg/mL 腎上腺素和 30μmol/L 布比卡因混合物；對照組單獨灌注 30μmol/L 布比卡因。心搏恢復定義爲形成自主規律的節律，且心率血壓乘積（RPP）大於基礎值的 10%，持續一分鐘以上。我們比較各組從 100μmol/L 布比卡因灌注結束到心搏恢復的時間（$T_{recovery}$）。記錄心搏恢復後 40 分鐘內的心功能相關指標。實驗結束後，取每個心臟標本的心尖，通過液相色譜法-串聯質譜法測定布比卡因含量。
結果：脂質組和聯合組的心搏恢復的時間（$T_{recovery}$）明顯短於腎上腺素組和對照組($P < 0.001$)，且腎上腺素組 $T_{recovery}$ 短於對照組($P < 0.05$)。心搏恢復後 40 分鐘內平均 RPP 由高到低依次爲：聯合組＞脂質組和腎上腺素組＞對照組($P < 0.01$)。恢復期間 RPP 最大值($RPP_{maximum}$)和 $RPP_{maximum}$ 與基礎值之比($RPP_{maximum}/RPP_{baseline}$)由高到低依次爲：聯合組＞脂質組和腎上腺素組＞對照組($P < 0.01$)。RPP、$RPP_{maximum}$ 和 $RPP_{maximum}/RPP_{baseline}$ 在脂質

組和腎上腺素組之間均無顯著差異。腎上腺素組和對照組的心肌組織布比卡因含量高於脂質組和聯合組($P < 0.001$)。
結論：在離體大鼠心臟模型中逆轉布比卡因引起的心臟停搏的作用，脂質與腎上腺素聯合應用對心功能的恢復作用優於其中任一藥物單獨應用。
（陳彬彬譯 馬皓琳 李士通校）
BACKGROUND: It remains unclear whether lipid combined with epinephrine is superior or inferior to either drug alone in treating bupivacaine cardiotoxicity. We compared the effects of lipid, epinephrine, and the combination of the two in reversing bupivacaine-induced asystole in the isolated rat heart model. We also measured the effects of lipid, epinephrine, and the combination of the two on bupivacaine content in cardiac tissue.
METHODS: Hearts from male Sprague–Dawley rats were excised and retrograde-perfused in a nonrecirculating Langendorff preparation. Bupivacaine 100 μmol/L was perfused until 3 minutes after asystole. Two percent lipid and 30 μmol/L bupivacaine mixture was then perfused in the lipid group; 0.15 μg/mL epinephrine and 30 μmol/L bupivacaine mixture in the epinephrine group; 2% lipid combined with 0.15 μg/mL epinephrine and 30 μmol/L bupivacaine in the combination group; and 30 μmol/L bupivacaine alone in the control group. Recovery of heartbeat was defined as unassisted regular rhythm with a rate-pressure product (RPP) >10% of baseline for >1 minute. We compared the time from the end of 100 μmol/L bupivacaine infusion to recovery of heartbeat ($T_{recovery}$) for each group. The variables of cardiac function were recorded for 40 minutes after recovery of heartbeat. The cardiac apex of each heart was taken for measurement of the bupivacaine content by liquid chromatography–tandem mass spectrometry at the end of the experiment.
RESULTS: Time to recovery ($T_{recovery}$) in the lipid and combination groups was significantly shorter than that in the epinephrine and control groups ($P < 0.001$), and $T_{recovery}$ in the epinephrine group was shorter than that in the control group ($P < 0.05$). The rank order of the mean RPP during the 40 minutes after recovery of heartbeat from highest to lowest was the combination group > the lipid and epinephrine groups > the control group ($P < 0.01$). The rank order of the highest RPP value during recovery ($RPP_{maximum}$) and the ratio of $RPP_{maximum}$ to baseline value ($RPP_{maximum}/RPP_{baseline}$) from highest to lowest was the combination group > the lipid and epinephrine groups > the control group ($P < 0.01$). There was no significant difference between the lipid and epinephrine groups for RPP, $RPP_{maximum}$, and $RPP_{maximum}/RPP_{baseline}$. Cardiac tissue bupivacaine content in the epinephrine and control groups was higher than that in the lipid and combination groups ($P < 0.001$).
CONCLUSIONS: Lipid combined with epinephrine resulted in better recovery of cardiac function than either drug alone in reversal of bupivacaine-induced asystole in the isolated rat heart model.