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一氧化碳釋放因數 2 可促進肝素或阿加曲班抗凝血漿的凝血並減少纖溶受損

Carbon Monoxide Releasing Molecule-2 Enhances Coagulation and Diminishes Fibrinolytic Vulnerability in Plasma Exposed to Heparin or Argatroban

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背景：最近的研究表明：在正常及血友病人的血漿中加入一氧化碳釋放因數（三羰基二氯代釕(II) 二聚物，CORM-2）能增強凝血作用並降低纖溶損傷。作者驗證了以下假說：經過肝素或者阿加曲班抗凝的血漿在暴露於 CORM-2 後能加強凝血同時減少纖溶蛋白受損。

方法：正常血漿以 0 至 0.1 U/mL 不同劑量的普通肝素或者 0 至 1 μg/mL 不同劑量的阿加曲班抗凝處理。隨後將部分樣本暴露於 0 至 100 μM 濃度的 CORM-2，再予組織因數啓動。另外一些樣本予以同樣的抗凝及 CORM-2 方案處理，相應的以 100 U/mL tPA（組織型纖維蛋白溶酶原啓動劑）啓動，用以評估纖維蛋白的受損性。收集血栓彈性描記器的資料直至血栓的強度趨於穩定或者至血栓溶解。

結果：未加入 tPA 的樣本，CORM-2 顯著增加了血栓形成的速度，其中肝素組 75%，阿加曲班 40%。血栓的強度也明顯增加，肝素組爲 69%，阿加曲班 72%。tPA 處理組，兩種抗凝劑處理的樣本在暴露於 CORM-2 後血栓的形成速度與強度增加更加明顯，達到 94%–731%，同時血栓溶解時間也長達 103%–200%。

結論：肝素或者阿加曲班抗凝的血漿暴露於 CORM-2 能增加血栓的形成速度，血栓強度以及壽命。對於 CORM-2 是否能降低血栓相關的出血併發症，還需要進行其他的臨床前研究。

（鄒巧群　譯　陳傑　校）

BACKGROUND: It has been recently demonstrated that a carbon monoxide releasing molecule (tricarbonyldichlororuthenium [II] dimer; CORM-2) enhances coagulation and attenuates vulnerability to fibrinolysis in normal and hemophiliac human plasma. We tested the hypothesis that plasma anticoagulated with heparin or argatroban would demonstrate improved coagulation and decreased fibrinolytic vulnerability after exposure to CORM-2.

METHODS: Normal plasma was anticoagulated with 0 to 0.1 U/mL unfractionated heparin or 0 to 1 μg/mL argatroban. Samples were subsequently exposed to 0 or 100 μM CORM-2 and activated with tissue factor. Additional samples with the same anticoagulant and CORM-2 exposure schema were incubated with 100 U/mL tissue-type plasminogen activator (tPA) to assess fibrinolytic vulnerability. Thrombelastographic data were collected until either clot strength stabilized or clot lysis occurred as appropriate.

RESULTS: In the absence of tPA, CORM-2 significantly increased the velocity of clot growth in heparin (75%) and argatroban-exposed (40%) samples. Clot strength was also significantly increased in heparin (69%) and argatroban-exposed (72%) samples. In the presence of tPA, CORM-2–treated samples had even greater (94%–731%) increases in velocity of growth and strength after exposure to either anticoagulant and significantly increased clot lysis time (103%–200%).

CONCLUSIONS: CORM-2 exposure resulted in faster-growing, stronger, longer-lived thrombi after anticoagulation with heparin or argatroban. Additional preclinical investigation is warranted to determine whether CORM-2 administration will be useful in attenuating bleeding complications associated with thromboprophylaxis.

液體限制的持續時間對異丙酚誘導期低血壓的影響

The Influence of Duration of Fluid Abstinence on Hypotension During Propofol Induction

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背景：術前長時間禁食可能加重全麻誘導期的低血壓。本文的研究目的是確定是否術前液體限制的持續時間與異丙酚誘導期的動脈血壓變化及藥物用量有關。

方法：選取了 130 名 ASA 分級在 I 到 II、無高血壓、年齡在 18 到 65 歲、擇期行全身麻醉下手術的患者。應用標準的生理監測和腦電圖雙頻指數 (BIS) 監測。靜脈輸注異丙酚開始時 40 mg ·kg^{-1} ·h^{-1} ，當 BIS 降至 50 時減至 8 mg ·kg^{-1} ·h^{-1} 。收集 15 分鐘內的心血管資料。主要觀察指標是從基礎平均動脈壓減少的最大的百分比（max%ΔMAP)。次要指標是 BIS 下降至 50 時的異丙酚用量（PDBIS50）。應用一元線性回歸和多元線性回歸用來分析空腹時間、主次觀察指標的指標之間的潛在關係。

結果：平均液體限制時間是 694 min（範圍：115 至 1263 min）。液體限制（分鐘）對應最大值 %ΔMAP (%)和 PDBIS50 (mg)的非標準化回歸係數（95%可信區間）分別是 0.003%(−0.002%至+ 0.009%) 和 0.021 mg (−0.017 mg 至+ 0.059 mg)。校正多元模型中具有顯著性。其他因素並應用 2 型離差平方和檢驗後，相應回歸係數是 −0.0001%（−0.004%至+ 0.004%，P = 0.94） 和 −0.006 mg (−0.039 mg 至+ 0.026 mg，P = 0.70）。以小時爲單位的液體限制對最大 %ΔMAP 影響是 −0.01%(−0.26%至+ 0.24%) ，對 PDBIS50 的影響是−0.38 mg （−2.34 mg 至+ 1.58 mg）。

結論：小於 65 歲的健康成人異丙酚麻醉誘導迅速靜脈注射時，術前液體限制的持續時間不會影響 MAP 及異丙酚的用量。

（唐穎　譯　陳傑　校）

BACKGROUND: Prolonged preoperative fasting might be expected to exacerbate hypotension during the induction of general anesthesia. We aimed to establish whether

the duration of preoperative abstinence from fluids independently contributed to arterial blood pressure changes and dosage requirements during propofol induction.

METHODS: We prospectively recruited 130 ASA I or II nonhypertensive patients, ages 18 to 65 years scheduled for surgery under general anesthesia. Standard physiological and electroencephalographic bispectral index (BIS) monitoring was applied to each patient. Intravenous propofol infusion was commenced at 40 mg $\cdot kg^{-1} \cdot h^{-1}$ and reduced to 8 mg $\cdot kg^{-1} \cdot h^{-1}$ when the BIS decreased to 50. Frequent cardiovascular data were collected for 15 minutes. The primary endpoint was maximal percentage decrease from baseline mean arterial blood pressure (max%ΔMAP). The secondary endpoint was the propofol dose at which BIS decreased to 50 (PDBIS50). Univariate linear regression and then multivariate linear regression was used to analyze the associations between potential predictors, including fasting time, and these 2 endpoints.

RESULTS: Mean fluid abstinence time was 694 minutes (range: 115 to 1263 minutes). Unstandardized regression coefficients (95% confidence intervals [CIs]) for fluid abstinence (minutes) versus max%ΔMAP (%) and PDBIS50 (mg) were, respectively, 0.003% (−0.002% to + 0.009%) and 0.021 mg (−0.017 mg to + 0.059 mg). On adjusting for other, significant predictors in a multivariate model and applying type II sum of squares tests, the corresponding values were −0.0001% (−0.004% to + 0.004%, P = 0.94) and −0.006 mg (−0.039 mg to + 0.026 mg, P = 0.70). The effect of a 1-hour increase in fluid abstinence on max%ΔMAP was therefore −0.01% (−0.26% to + 0.24%) and on PDBIS50, −0.38 mg (−2.34 mg to + 1.58 mg).

CONCLUSION: When propofol is infused rapidly for induction of anesthesia in healthy adults younger than 65 years, the duration of preoperative fluid abstinence does not appear to affect MAP or propofol dose requirements.

地西泮通過兩種截然不同的機制減少大腦新皮層神經元動作電位的釋放

Diazepam Decreases Action Potential Firing of Neocortical Neurons via Two Distinct Mechanisms

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背景：苯二氮卓類藥物廣泛用於臨床麻醉的術前用藥和全麻誘導。最近的體外研究表明 A 型 γ-氨基丁酸受體除了含有一個經典高親和力結合位點，還擁有另外一個非經典的苯二氮卓類藥物結合位點。目前，這個新的非經典的結合位點對苯二氮卓類藥物在中樞神經系統中的作用還不清楚。

方法：因爲新皮層介導全麻中鎮靜、催眠作用，作者在新皮層培養腦片應用多細胞記錄法定量記錄濃度範圍（從 10 nM 到 100 μM）較寬的地西泮對細胞外自發動作電位的影響。

結果：地西泮濃度從低濃度至 6.25 μM 能降低大腦神經元的活動性，接近於最大值的 20%。這種作用可被苯二氮卓類拮抗劑氟馬西尼逆轉。在地西泮濃度>12.5 μM，誘發另一濃度相關網狀活動抑制。與低濃度作用不同，這種高濃度效應不能被氟馬西尼拮抗。

結論：地西泮誘發大腦新皮層神經元自發性動作電位釋放的雙相抑制作用。低中濃度通過經典的結合位點產生單相的，緩和的抑制作用且能被氟馬西尼拮抗。然而，高濃度地西泮的作用不受氟馬西尼的影響。因此，這些發現支持苯二氮卓類藥物在 A 型 γ-氨基丁酸受體上至少有兩個不同的結合位點。此外，研究結果也支持以下假設：經典的高親和力的結合位點調節低劑量的地西泮的作用，例如遺忘，抗焦慮和鎮靜，而另一非經典獨立的結合部位則與地西泮的催眠肌松等麻醉效應有關。

（陳靈科　譯　陳傑　校）

BACKGROUND: Benzodiazepines are widely used in clinical anesthesia as premedication, but also to induce general anesthesia. Recent in vitro studies suggest that γ-aminobutyric acid type A receptors, harboring a classical high-affinity benzodiazepine binding site, possess another “nonclassical” binding site for benzodiazepines. At present, it is unclear if, and to what extent, this novel nonclassical binding site is of relevance for the actions of benzodiazepines in the central nervous system.

METHODS: Because neocortex is involved in mediating the sedative and hypnotic properties of general anesthetics, we quantified the actions of diazepam over a wide range of concentrations (from 10 nM up to 100 μM) in organotypic slice cultures using extracellular multiunit recordings of spontaneous action potential activity.

RESULTS: Up to a concentration of 6.25 μM, diazepam reduced the activity of neocortical neurons, approaching a maximum of approximately 20%. This action was nullified by the benzodiazepine antagonist flumazenil. At concentrations >12.5 μM, diazepam evoked a second concentration-dependent dampening of network activity. Unlike the low concentration effect, this high concentration component was resistant to flumazenil.

CONCLUSIONS: Diazepam induced a biphasic attenuation of spontaneous action potential firing of neocortical neurons. Low to moderate concentrations caused a monotonic, mild depression that is mediated via the classical binding site as it is antagonized by flumazenil. However, the effects of diazepam observed at high concentrations were not affected by flumazenil. Hence, these findings support the concept of at least 2 different binding sites for benzodiazepines on γ-aminobutyric acid type A receptors. Furthermore, our results are consistent with the hypothesis that the classical high-affinity binding site mediates low-dose diazepam actions, such as amnesia, anxiolysis, and sedation, while a second, nonclassical and independent site contributes to the anesthetic effects of diazepam, such as hypnosis and immobility.

簡報：揮發性麻醉藥調節乳腺和腦腫瘤細胞基因的表達

Brief Report: Volatile Anesthetics Modulate Gene Expression in Breast and Brain Tumor Cells

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基因表達越來越多地應用於臨床實踐中診斷、預後判斷和治療。作者對揮發性麻醉藥可以影響腫瘤細胞的基因表達這一假說進行了測試。將離體的神經細胞系 SH-SY5Y 細胞和乳腺細胞系 MCF-7細胞暴露于安氟醚，異氟醚，地氟醚，氟烷和七氟醚或笑氣中。對此，應用微陣列基因表達譜進行研究。結果顯示在細胞培養中，基因表達水準有顯著性的差異，並且暴露于不同時間長度和不同的揮發性麻醉藥，反應也不同。一些構成乳房癌預測基因指紋法的基因受揮發性麻醉藥的影響。結果表明揮發性麻醉藥在乳腺和腦腫瘤的細胞培養中，可以以一種獨特和時間依賴式的方式對基因的表達進行調製。

（張蕾　譯　陳傑　校）

Gene expression is increasingly used for diagnostic, prognostic, and therapeutic purposes in clinical practice. We tested the hypothesis that volatile anesthetics (VA) affect gene expression of tumor cells. Cells from the neuronal cell line SH-SY5Y and from the breast cell line MCF-7 were exposed ex vivo to enflurane, isoflurane, desflurane, halothane, sevoflurane, or nitrous oxide. Microarray gene expression profiles were studied. We observed significant differences in gene expression levels of cell cultures and response in time when exposed to different VA. Some genes used for predictive genetic fingerprints for breast cancer were affected by VA. Our findings suggest that VA modulate gene expression in breast and brain tumor cell cultures in a unique and time-dependent manner.

剖腹產術後傷口皮下連續灌輸布比卡因相比於生理鹽水可減少切口白細胞介素－10 及增加 P 物質的產生

Continuous Subcutaneous Instillation of Bupivacaine Compared to Saline Reduces Interleukin 10 and Increases Substance P in Surgical Wounds After Cesarean Delivery

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背景：最新的證據表明，局部麻醉藥局部給藥可能對局部組織產生影響，如關節內注射後軟骨組織溶解。炎症反應的改變爲局部麻醉誘導組織毒性的可能機制之一。這項研究中，作者測試了連續局部浸潤麻醉對炎症介質的釋放及對剖宮產術後傷口皮膚的影響。

方法：腰麻下剖宮產術的 38 名健康婦女參與了這項研究，隨機分爲兩組，一組產婦術後 24 小時給予布比卡因 5mg/mL 手術傷口皮下浸潤，另一組給予 2 mL/h 生理鹽水。術後 1, 3, 5, 7 及 24 小時採用皮下傷口引流技術收集傷口滲出液。採用多重 Bio-Plex®（Bio-Rad, Hercules, CA）和酶聯免疫吸附法測定細胞因數，趨化因數，P 物質，前列腺素 E2 和神經生長因數。

結果：手術傷口布比卡因皮下浸潤與生理鹽水浸潤相比，白細胞介素 10 明顯降低，P 物質明顯增加（24 小時濃度時間曲線；P<0.001）。其他細胞因數，神經生長因數，前列腺素 E2 無顯著差異。

結論：這項研究表明，術後傷口連續輸注臨床常用劑量的布比卡因會影響局部炎症介質成份。白細胞介素 10 減少意味著抗炎機制受損。在皮膚癒合過程中這一變化伴隨這促炎介質如 P 物質的釋放是否導致手術傷口炎症反應加劇需更多的研究來證實。

（陳毓雯　譯　陳傑　校）

BACKGROUND: Recent evidence suggests that locally delivered local anesthetics may exert tissue-damaging effects such as chondrolysis after intraarticular injection. Alteration of the inflammatory response is a potential mechanism for local anesthetic-induced tissue toxicity. In this study, we tested the effects of continuous local anesthetic infiltration on the release of inflammatory and nociceptive mediators in skin wounds after cesarean delivery.

METHODS: Thirty-eight healthy women undergoing cesarean delivery with spinal anesthesia were enrolled in this study, and were randomized to receive subcutaneous surgical wound infiltration with bupivacaine 5 mg/mL or saline at 2 mL/h for 24 hours

after cesarean delivery. Wound exudate was sampled at 1, 3, 5, 7, and 24 hours after cesarean delivery using a subcutaneous wound drain technique. Cytokines, chemokines, substance P, prostaglandin E_2, and nerve growth factor were assayed using multiplex Bio-Plex® (Bio-Rad, Hercules, CA) and enzyme-linked immunosorbent assays.

RESULTS: Bupivacaine wound infusion resulted in a significant decrease of interleukin 10 and increase of substance P in wounds compared with saline infusion (area under the 24-hour concentration-time curve; $P < 0.001$). No statistically significant differences were detected for other cytokines, nerve growth factor, and prostaglandin E_2.

CONCLUSIONS: This study demonstrates that the continuous administration of clinically used doses of bupivacaine into wounds affects the local composition of wound mediators. Observed changes in interleukin 10 are compatible with a disruption of antiinflammatory mechanisms. Whether such modulation combined with the release of the proinflammatory mediator substance P results in an overall proinflammatory wound response will require future studies of wound healing

焦點回顧：產科連續脊麻和鎮痛

Focused Review: Continuous Spinal Anesthesia and Analgesia in Obstetrics

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本文重述連續脊麻技術在產科人群中的應用。探討連續脊麻的利弊，目前常用的導管和配件。同時討論了連續脊麻在無痛分娩和手術麻醉中的管理策略。連續脊麻在一些特殊臨床情況下可能有獨特的價值。

（楊秋娟　譯　陳傑　校）

The development of the technique of continuous spinal anesthesia as it relates to the obstetric population is recounted. The advantages and disadvantages of continuous spinal anesthesia are examined, currently available catheters and kits are reviewed, and strategies for the management of continuous spinal techniques for labor analgesia and

surgical anesthesia are discussed. Continuous spinal anesthesia may have particular value over other regional techniques in several specific clinical circumstances.

高熱對血漿谷氨酸濃度的影響

The Effect of Hyperthermia on Blood Glutamate Levels

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前言：谷氨酸的神經毒性是由谷氨酸在腦內合成和過度後釋放入血這兩者之間的平衡所決定。血漿中谷氨酸濃度降低會增加谷氨酸經血腦屏障濾過的量。而有關高熱對於降低血液中谷氨酸濃度的機制及其有效性的報導尚不完全。雖然有假設認爲高熱可能是通過啓動應激機制來降低血漿谷氨酸濃度的，但是阻滯β受體後可以減弱這種應激反應從而防止血漿谷氨酸濃度的降低。此外，處於高熱狀態的同時，谷氨酸也會從肌肉釋放到血液中，這又造成了血液中谷氨酸濃度的增加。因而本研究旨在探討高熱對血液中谷氨酸濃度的影響以及β受體阻滯劑-心得安對應激引起的谷氨酸濃度改變的作用。本試驗同時還研究了血漿谷氨酸清除劑草醯乙酸鹽對於改善高熱引起的谷氨酸濃度增加的有效性。

材料方法：24 只小鼠隨機分成 3 組。小鼠的體溫每隔 40min 升高 1℃，從 37℃一直升高到 42℃。第一組給予 1ml/100g 的等張生理鹽水（對照）。當體溫升至 39℃後，第二組給予 ml/100g 的 1M 草醯乙酸鹽。而第三組在加熱前給予 10mg/kg 的心得安。

結果：將小鼠的體溫從 37℃升高到 39℃，對照組和草醯乙酸鹽治療組的血漿谷氨酸濃度都有顯著下降（分別 P<0.01 vs P<0.0001），但是從 40℃繼續升高到 42℃

則會增加血漿谷氨酸的濃度（分別 P<0.01 vs P<0.0001）。用心得安預處理的小鼠可以防止輕微體溫升高導致的血漿谷氨酸濃度降低，但是心得安不影響 41℃ 和 42℃ 高熱時引起的血漿谷氨酸濃度的增加（P<0.005）。

討論：本研究的結果證明高熱會降低血漿中谷氨酸濃度，其可能的機制是刺激了交感神經系統。之前報導草醯乙酸鹽在 37℃ 時能減少血漿谷氨酸的濃度，但是在 40℃ 是無效的。給予心得安預處理後能減弱血漿谷氨酸濃度減少的程度，而心得安的這種效應在對照組和治療組是沒有差異的。從而可以理解在高熱時血漿中谷氨酸濃度改變的基本機制，以及在治療神經退行性疾病時應激具有重要的臨床意義。（張婷　譯　陳傑　校）

INTRODUCTION: Glutamate neurotoxicity is determined by the balance between glutamate release within the brain and efflux of excess glutamate from the brain. Brain-to-blood efflux of glutamate is increased by decreasing the concentration of glutamate in blood. Little is known about the effect of hyperthermia on blood glutamate concentrations, and the effectiveness of blood glutamate–decreasing mechanisms in these conditions. Although hyperthermia is hypothesized to decrease blood glutamate concentrations by activation of stress mechanisms, blunting the stress response by blocking β-adrenergic receptors should prevent this decrease. Furthermore, during hyperthermia there should be a concurrent process of leakage of glutamate from muscle tissue into blood, resulting in a contradictory increase of blood glutamate concentrations. In this study we investigated the effects of hyperthermia on blood glutamate levels and studied the effects of the β-adrenergic receptor antagonist propranolol on stress-induced changes in glutamate levels. We then studied the effectiveness of the blood glutamate scavenger oxaloacetate on hyperthermia-induced increases of glutamate levels.

MATERIALS AND METHODS: Twenty-four rats were randomly divided into 3 groups. Rats' body temperatures were increased (by 1℃ every 40 minutes) from 37℃ to 42℃. The first group received 1 mL per 100 g of isotonic saline (control). The second group received 1 mL per 100 g of 1M oxaloacetate when the temperature reached 39℃. The third group received 10 mg/kg of propranolol before initiation of the warming.

RESULTS: Warming the rats from 37℃ to 39℃ decreased the blood glutamate levels in the control group ($P < 0.01$) and oxaloacetate treatment group ($P < 0.0001$), whereas further increases in temperature from 40℃ to 42℃ increased the blood glutamate levels ($P < 0.01$ and $P < 0.0001$, respectively). Pretreatment with propranolol prevented the decrease in blood glutamate concentrations seen in mild hyperthermia and did not affect the increase in blood glutamate levels seen at temperatures of 41℃ and 42℃ ($P < 0.005$).

DISCUSSION: The results of this study demonstrated that hyperthermia leads to decreases in glutamate levels in the blood, presumably by activation of the sympathetic nervous system. Oxaloacetate, previously reported to reduce blood glutamate levels at 37℃, was ineffective at temperatures over 40℃. Propranolol pretreatment blunted the initial decrease in blood glutamate, and thereafter had no effect when compared with control and treatment groups. Understanding the mechanisms underlying glutamate regulation in the blood during states of hyperthermia and stress has important clinical implications in treating neurodegenerative conditions.

心臟再同步療法治療心臟衰竭

Cardiac Resynchronization Therapy for Treatment of Heart Failure

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傳導異常，通常可見於收縮期心衰，因爲沒有正常傳導組織的幫助電脈衝傳播緩慢導致心機啓動延遲。心室收縮不同步的結果是機械性效力較少，收縮功能減退和舒張期充盈受損。左右心室同時起搏（即兩心室起搏）可以減少心室不同步收縮，可以克服傳導延遲的這些後果。使用可植入性節律管理裝置進行心臟再同步療法的一個重要作用就是在心力衰竭中使得心室功能最佳化。在病人預後中其遠期好處已被充分確認。隨著使用的增多，對心臟再同步治療裝置及其治療原理的理解對醫生處理圍手術期間和重症監護中的心衰病人很重要。
（周潔 譯 馬皓琳 李士通 校）

Conduction abnormalities, commonly seen in systolic heart failure, lead to delayed activation of the myocardium as the electrical impulse spreads slowly without the aid of healthy conduction tissue. The resulting dyssynchronous ventricular contraction is mechanically less efficient, reducing systolic function and impairing diastolic filling. Simultaneous pacing of the right and left ventricles (i.e., biventricular pacing) reduces ventricular dyssynchronous contraction, overcoming these consequences of conduction delay. An important role for implantable rhythm-management devices providing cardiac

resynchronization therapy has emerged in the optimization of ventricular function in heart failure. Long-term benefits in patient outcomes have been well established. With increasing use, understanding of cardiac resynchronization therapy devices and the principles behind the therapy are important for physicians providing perioperative and intensive care for patients with heart failure.

苯乙胺增加 S(+)氯胺酮對淋巴瘤，神經元和神經膠質細胞的細胞毒性

Benzethonium Increases the Cytotoxicity of S(+)-Ketamine in Lymphoma, Neuronal, and Glial Cells

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背景：已有研究表明氯胺酮在動物及人體有神經毒性。已歸咎於氯胺酮中添加的防腐劑誘導了這種神經毒性。因此，我們體外研究了使用最廣的氯胺酮防腐劑——氯化苄乙銨——是否會增加 S(+)-氯胺酮在淋巴瘤、成神經瘤細胞及初級星形膠質細胞中的毒性
方法：將人外周血白血病 T 淋巴瘤細胞和成神經瘤細胞（SHEP）與通過商業途徑可獲得的含有苯乙胺的 S-氯胺酮、純 S-氯胺酮和純氯化苄乙銨中孵育 24 小時。用流式細胞儀評價早期和晚期細胞凋亡率。第二步，通過對成神經瘤細胞以及小鼠初級星形膠質細胞進行線粒體活性測定（XTT）來研究苯乙胺與氯胺酮的混合毒性。用等輻射分析法來評估毒性的相加效應。
結果：在人外周血白血病 T 淋巴瘤細胞和成神經瘤細胞中，苯乙胺增加了氯胺酮的毒性，細胞壞死率分別從 32%增加到 80%及從 64%增加到 84%。從等效線圖解法中可以看出，成神經瘤細胞和小鼠初級星形膠質細胞中測的的混合毒性在計算得出的純淨物毒性之和的置信區間內。
結論：我們的結論是苯乙胺以相加的方式增加了氯胺酮對造血、神經元和神經膠質起源細胞的局部毒性。因此，建議當使用含有防腐劑的 S-氯胺酮作爲長時程椎管內鎮痛治療的添加藥物時應特別予以謹慎。
（瞿亦楓 譯 馬皓琳 李士通校）

INTRODUCTION: Ketamine has been demonstrated to be neurotoxic in animals as well as in patients. Preservatives added to ketamine have been accused to induce this neurotoxicity. Therefore, we investigated whether the most widely used preservative of ketamine–benzethonium chloride–enhances the toxicity of S(+)-ketamine in vitro in lymphoma, neuroblastoma cells and primary astrocytes.
METHODS: Human Jurkat T-lymphoma- and neuroblastoma cells (SHEP) were incubated for 24 hours with commercially available S-ketamine containing benzethonium, pure S-ketamine and pure benzethonium chloride. The rate of early- and late-apoptotic cells was evaluated by flowcytometry. In a second step the combined toxicity of

benzethonium and ketamine was investigated in neuroblastoma cells and primary rat astrocytes in a mitochondrial activity assay (XTT). The additivity of the toxicities was evaluated by employing isobolographic analysis.
RESULTS: In Jurkat T-lymphoma and neuroblastoma cells benzethonium increased the toxicity of ketamine from 32% to 80% and from 64% to 84% cell deaths, respectively. In neuroblastoma cells as well as in primary rat astrocytes the measured combined toxicity was within the confidence interval of the calculated pure additive toxicity as seen in the isobolograms.
CONCLUSIONS: We conclude that benzethonium increases the local toxicity of ketamine in cells of hematopoetic, neuronal and glial origin in an additive manner. Therefore, caution is recommended especially when using preservative containing S-ketamine as an additive for long-term neuraxial analgesia.

對海地地震的一個有組織的、綜合性的、安全的戰略策略反應：一項由無國際性災難應急預案的一個學術性麻醉系描述的關於前期部署準備和初步經驗的敍述

An Organized, Comprehensive, and Security-Enabled Strategic Response to the Haiti Earthquake: A Description of Pre-Deployment Readiness Preparation and Preliminary Experience from an Academic Anesthesiology Department with No Preexisting International Disaster Response Program

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背景：海地當地時間 2010 年 1 月 12 日（星期二）16 時 53 分發生了裏氏 7.0 級地震。全球人道主義組織對此的反應是迅速的，但是薄弱的基礎設施和對緊急事件的準備限制了很多艱難的嘗試。對於大型災難人員傷亡應急有經驗的組織能快速成功地對緊急事件醫療處理團隊進行部署。有應急意向但無準備的醫療團隊對緊急事件也做出了反應。在本次報導中，我們描述了一個無國際性災難應急預案的學術大學麻醉系在接到海地的一個美國非政府組織尋求醫療支援的電話後所做出的準備和規劃過程。本文的焦點是前期部署準備的過程，而不是描述在海地當地提供醫療處理的後期部署報導。
方法：我們對賓夕法尼亞大學附屬醫院與海地地震有關的通訊和措施進行了一項即時的定性評估和系統回顧。回顧並摘要了直到部署當天的關於計畫、決策支援、設備獲取的團隊會議、電話會議和電子郵件通訊及行動和步驟。我們編譯了重要事件的計時並發展了此過程的反應時間線。我們組織了回國的麻醉成員的訪談。

結果：海地地震後 4 天，一個在海地以馬塞諸塞州波士頓爲基地的名爲“健康”的非營利、非政府組織裏有超過 20 年醫療工作經驗的成員與賓夕法尼亞大學健康機構取得聯繫，要求提供醫療團隊的支持。麻醉科、外科、整形外科和護理部回應了該請求，並進行了志願者的選擇、疫苗接種以及裝備清單的系統化研究。用世界衛生組織和疾病控制中心指南、美國麻醉協會創傷和急症準備委員會、發表的文章及國內的討論交流來指導準備過程。
結論：對於國際性自然災害緊急事件後的醫療需求而做出的有組織的戰略應答可以由一個有醫療系統支援但無先前已建立應急系統的學術性麻醉系在 6 到 12 天內安全有效地完成。在今後的研究中，該應答的價值和時機有待確定。當與災難當地已有支持基礎的醫護機構以有組織的方式互相合作時，在將應急醫療團隊投放到該區域方面經驗有限的機構可迅速地做出該應答。
（毛祖旻 譯 馬皓琳 李士通 校）

BACKGROUND: On Tuesday, January 12, 2010 at 16:53 local time, a magnitude 7.0 M_w earthquake struck Haiti. The global humanitarian attempt to respond was swift, but poor infrastructure and emergency preparedness limited many efforts. Rapid, successful deployment of emergency medical care teams was accomplished by organizations with experience in mass disaster casualty response. Well-intentioned, but unprepared, medical teams also responded. In this report, we describe the preparation and planning process used at an academic university department of anesthesiology with no preexisting international disaster response program, after a call from an American-based nongovernmental organization operating in Haiti requested medical support. The focus of this article is the pre-deployment readiness process, and is not a post-deployment report describing the medical care provided in Haiti.
METHODS: A real-time qualitative assessment and systematic review of the Hospital of the University of Pennsylvania's communications and actions relevant to the Haiti earthquake were performed. Team meetings, conference calls, and electronic mail communication pertaining to planning, decision support, equipment procurement, and actions and steps up to the day of deployment were reviewed and abstracted. Timing of key events was compiled and a response timeline for this process was developed. Interviews with returning anesthesiology members were conducted.
RESULTS: Four days after the Haiti earthquake, Partners in Health, a nonprofit, nongovernmental organization based in Boston, Massachusetts, with >20 years of experience providing medical care in Haiti contacted the University of Pennsylvania Health System to request medical team support. The departments of anesthesiology, surgery, orthopedics, and nursing responded to this request with a volunteer selection process, vaccination program, and systematic development of equipment lists. World Health Organization and Centers for Disease Control guidelines, the American Society of Anesthesiology Committee on Trauma and Emergency Preparedness, published articles, and in-country contacts were used to guide the preparatory process.
CONCLUSION: An organized strategic response to medical needs after an international natural disaster emergency can be accomplished safely and effectively within 6 to 12 days by an academic anesthesiology department, with medical system support, in a center with no previously established response system. The value and timeliness of this response will be determined with further study. Institutions with limited experience in putting an

emergency medical team into the field may be able to quickly do so when such efforts are executed in a systematic manner in coordination with a health care organization that already has support infrastructure at the site of the disaster.

在海地 2010 地震後的麻醉實踐

Anesthetic Practice in Haiti After the 2010 Earthquake

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2010 年 1 月 12 日，裏氏 7 級地震襲擊了海地——一個醫療資源極其匱乏、西半球最貧窮的國家。我們來到了米洛（位於太子港以北）的聖心醫院照顧那些受傷的患者，聖心醫院是海地地震中倖存的一所醫院，擁有 74 張床位和 2 個手術房間。由直升機從受震地區送來的大量病患使該醫院規模擴充到 400 張床位和 6 個手術房間。如同 2005 年喀什米爾和 2008 年中國的地震一樣，大多數受災者都遭受著極度的創傷的折磨，包括擠壓傷、撕裂傷、骨折和截肢，並伴有脫水和貧血。術前評估包括詢問一些基本的問題，如空腹狀態、過敏症及已有疾病情況等，但是由於語言問題的限制而需要一名翻譯。目標包括適當的麻醉深度，但避免窒息/氣道操作。這些目標使我們頻繁地使用了咪達唑侖和氯胺酮或區域麻醉。雖然我們有各種名稱及各種濃度的很多藥物，但是中央供氣的缺乏仍然帶來了不少麻煩。有破傷風、糖尿病酮症酸中毒、吸入性肺炎、由擠壓導致的急性腎衰、嚴重貧血、膿毒血症以及其他一些疾病的患者需要的術後監護，由於麻醉後監護室/重症監護室只有 8 張床位而受到限制。我們這次工作的其他一些重要方面還包括有衛生保健專業人員區分患者治療的輕重緩急，適應有限的實驗室和影像學輔助裝置，並合理安排現場物資供應。雖然各方面都存在很大的挑戰，但是這次的經歷讓我們在感情上充實了自我，並回憶起當初之所以選擇醫學和麻醉作爲專業的最根本原因。
（徐妍君 譯， 馬皓琳、李士通 校）

On January 12, 2010, a 7.0 M_L earthquake devastated Haiti, the most impoverished nation in the Western hemisphere with extremely limited health care resources. We traveled to Milot, Haiti situated north of Port-au-Prince, to care for injured patients at Hôpital Sacré Coeur, an undamaged hospital with 74 beds and 2 operating rooms. The massive influx of patients brought by helicopter from the earthquake zone transformed the hospital to >400 beds and 6 operating rooms. As with the 2005 Kashmir and 2008 China earthquake, most victims suffered from extremity injuries, encompassing crush injuries, lacerations, fractures, and amputations with associated dehydration and anemia. Preoperative evaluation was limited by language issues requiring a translator and included basic questions of fasting status, allergies, and coexisting conditions. Goals included adequate depth of anesthesia, while avoiding apnea/airway manipulation. These goals led to frequent use of midazolam and ketamine or regional anesthesia. Although many medications were present under various names and concentrations, the absence of a central gas supply proved troublesome. Postoperative care was limited to an 8-bed

postanesthesia care unit/intensive care unit caring for patients with tetanus, diabetic ketoacidosis, pulmonary aspiration, acute renal failure due to crush, extreme anemia, sepsis, and other illnesses. Other important aspects of this journey included the professionalism of the health care personnel who prioritized patient care, adaptation to limited laboratory and radiological services, and provision of living arrangements. Although challenging from many perspectives, the experience was emotionally enriching and recalls the fundamental reasons why we selected medicine and anesthesiology as a profession.

硬膜外鎮痛分娩和產婦發熱

Labor Epidural Analgesia and Maternal Fever

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接受硬膜外鎮痛的分娩婦女更有可能經歷高熱和明顯的臨床發熱。在行硬膜外鎮痛分娩的婦女中觀察到的逐步發展的中度發熱在那些選擇行其他鎮痛方法或沒有做鎮痛治療的分娩婦女中都沒有見到。臨床發熱在行硬膜外鎮痛分娩的婦女中也是大大地更多見。觀察到的平均體溫的緩慢上升也許是一小部分最終發熱的婦女和更多的在分娩過程中無發熱的婦女的體溫曲線平均的假像。選擇偏差混淆了硬膜外鎮痛和發熱之間的關係，因爲存在發熱風險（由於經受更長時間的羊膜破裂、更長時間的分娩期、接受更頻繁的子宮頸檢查和更多其他干擾）的婦女也更可能選擇硬膜外鎮痛。然而，即使隨機實驗已經證實接受硬膜外鎮痛的婦女發熱的發生率較高，並提示其因果關係，但硬膜外相關性發熱的機制仍然不能完全被理解。未接受硬膜外鎮痛的婦女體溫調節的改變和阿片類藥物的解熱作用也許能部分解釋這種現象，但更可能的原因是炎症，且最常見於胎盤和羊膜（絨毛膜羊膜炎）。產婦發熱的後果是多種多樣的。產科醫生更可能對發熱的分娩婦女進行手術干預，而新生兒科醫生更可能去評估患有敗血症的發熱婦女的新生兒狀況。更嚴重的是，產婦炎症性高熱與新生兒腦部損傷有關，表現爲腦癱、腦病以及兒童期後期中的學習障礙。現在，還沒有能安全有效抑制硬膜外相關發熱的方法。將來的研究應該確定這種發熱的病因，並且尋找安全有效的干預措施，以預防這種發熱和抑制其對新生兒腦部的潛在危害作用。

(楊秀娟 譯 馬皓琳 李士通 校)

Women in labor who receive epidural analgesia are more likely to experience hyperthermia and overt clinical fever. The gradual development of modest hyperthermia observed in laboring women with epidural analgesia is not seen in those electing other forms of analgesia or unmedicated labor. Clinical fever is also far more likely in women laboring with epidural analgesia. It is possible that the observed slow increase in mean temperature is an artifact of averaging the temperature curves of a small group of women who eventually develop fever with a larger group who remain afebrile throughout labor. Selection bias confounds the association between epidural analgesia and fever, because women at risk for fever—due to longer duration of ruptured membranes, longer labor,

more frequent cervical examinations, and other interventions—are also more likely to select epidural analgesia. However, even randomized trials have confirmed a higher incidence of fever in epidural-exposed women, suggesting a causal relationship. The mechanisms of epidural-associated fever remain incompletely understood. Altered thermoregulation and an antipyretic effect of opioids given to women without epidural analgesia may explain part of the phenomenon, but the most likely etiology is inflammation, most commonly in the placenta and membranes (chorioamnionitis). The consequences of maternal fever are diverse. Obstetricians are more likely to intervene surgically in laboring women with fever, and neonatologists are more likely to evaluate neonates of febrile women for sepsis. More ominously, maternal inflammatory fever is associated with neonatal brain injury, manifest as cerebral palsy, encephalopathy, and learning deficits in later childhood. At present, there are no safe and effective means to inhibit epidural-associated fever. Future research should define the etiology of this fever and search for safe and effective interventions to prevent it and to inhibit its potential adverse effects on the neonatal brain.

接受常規心導管檢查的心臟移植患兒靜脈快速注射右旋美托咪定後的急性血流動力學變化

Acute Hemodynamic Changes After Rapid Intravenous Bolus Dosing of Dexmedetomidine in Pediatric Heart Transplant Patients Undergoing Routine Cardiac Catheterization

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背景：右美托咪定是一種高選擇性的 α_2-腎上腺素能受體激動劑，具有鎮靜、抗焦慮以及鎮痛功能，並且對呼吸動力影響較小。其鎮靜以及降壓作用經由中樞 α_{2A} 以及 1 型咪唑啉受體調節，而外周 α_{2B}-腎上腺素受體活化則導致動脈血壓以及外周血管阻力增加。在這項隨機、前瞻性的臨床研究中，我們嘗試去量化在心臟移植的患兒中快速靜脈注射右美托咪定的短期的血流動力學作用。
方法：12 例年齡小於或者等於十歲，體重小於等於 40kg，在心臟移植術後行左心和右心導管插入術的常規監測的患兒被納入本項研究。在吸入或者靜脈誘導後，進行氣管插管，麻醉維持以 1MAC 的異氟醚混合空氣吸入、芬太尼(1 μg/kg)和羅庫溴銨(1 mg/kg)。在計畫的心導管插入術結束時，吸入 100%純氧。記錄一系列的基礎值包括心率（HR）、收縮壓、舒張壓、中心靜脈壓、肺動脈收縮壓、肺動脈舒張壓、肺毛細血管楔壓以及熱稀釋心輸出量後，在 5 秒內快速靜脈推注右美托咪定 0.25 或 0.5 μg/kg。在 1 分鐘以及 5 分鐘後再次測量血流動力學指標。
結果：每組有六位患者。研究顯示在快速靜脈注射兩種劑量的右美托咪定 1 分鐘後收縮壓、舒張壓、肺動脈收縮壓、肺動脈舒張壓、肺毛細血管楔壓以及全身血管阻

力均增高，並且在 5 分鐘後均顯著下降至接近基礎值。這種壓力的短暫上升在全身系統較肺系更爲明顯。在全身系統中，舒張壓的上升百分率較收縮壓大。心輸出量、中心靜脈壓以及肺血管阻力並無明顯變化。心率在兩組不同劑量推注後 1 分鐘時均下降，並且是 0.5 μg/kg 組中注射後 5 分鐘時唯一一個與基礎值仍然有差異的血流動力學指標。

結論：這組小樣本的已行心臟移植的患兒接受快速靜脈注射右美托咪定在臨床上完全能耐受，雖然其導致了短暫並且顯著的全身和肺動脈壓力升高以及心率的減慢。在全身系統，舒張壓上升比率較舒張壓大，而且這些短暫的壓力升高在全身系統較肺系更爲明顯。

（龔寅 譯 馬皓琳 李士通校）

BACKGROUND: Dexmedetomidine is a highly selective α_2-adrenoceptor agonist with sedative, anxiolytic, and analgesic properties that has minimal effects on respiratory drive. Its sedative and hypotensive effects are mediated via central α_{2A} and imidazoline type 1 receptors while activation of peripheral α_{2B}-adrenoceptors result in an increase in arterial blood pressure and systemic vascular resistance. In this randomized, prospective, clinical study, we attempted to quantify the short-term hemodynamic effects resulting from a rapid IV bolus administration of dexmedetomidine in pediatric cardiac transplant patients.
METHODS: Twelve patients, aged 10 years or younger, weighing ≤40 kg, presenting for routine surveillance of right and left heart cardiac catheterization after cardiac transplantation were enrolled. After an inhaled or IV induction, the tracheas were intubated and anesthesia was maintained with 1 minimum alveolar concentration of isoflurane in room air, fentanyl (1 μg/kg), and rocuronium (1 mg/kg). At the completion of the planned cardiac catheterization, 100% oxygen was administered. After recording a set of baseline values that included heart rate (HR), systolic blood pressure, diastolic blood pressure, central venous pressure, systolic pulmonary artery pressure, diastolic pulmonary artery pressure, pulmonary artery wedge pressure, and thermodilution-based cardiac output, a rapid IV dexmedetomidine bolus of either 0.25 or 0.5 μg/kg was administered over 5 seconds. The hemodynamic measurements were repeated at 1 minute and 5 minutes.
RESULTS: There were 6 patients in each group. Investigation suggested that systolic blood pressure, diastolic blood pressure, systolic pulmonary artery pressure, diastolic pulmonary artery pressure, pulmonary artery wedge pressure, and systemic vascular resistance all increased at 1 minute after rapid IV bolus for both doses and decreased significantly to near baseline for both doses by 5 minutes. The transient increase in pressures was more pronounced in the systemic system than in the pulmonary system. In the systemic system, there was a larger percent increase in the diastolic pressures than the systolic pressures. Cardiac output, central venous pressure, and pulmonary vascular resistance did not change significantly. HR decreased at 1 minute for both doses and was, within the 0.5 μg/kg group, the only hemodynamic variable still changed from baseline at the 5-minute time point.
CONCLUSION: Rapid IV bolus administration of dexmedetomidine in this small sample of children having undergone heart transplants was clinically well tolerated, although it resulted in a transient but significant increase in systemic and pulmonary pressure and a decrease in HR. In the systemic system, there is a larger percent increase

in the diastolic pressures than the systolic pressures and, furthermore, these transient increases in pressures were more pronounced in the systemic system than in the pulmonary system.

既往有椎管狹窄、腰椎間盤疾病或脊柱手術史的病人行椎管內阻滯：效能和神經系統併發症

Neuraxial Blockade in Patients with Preexisting Spinal Stenosis, Lumbar Disk Disease, or Prior Spine Surgery: Efficacy and Neurologic Complications

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背景：有椎管病變（包括椎管狹窄和腰椎間盤病變）的病人，通常避免行椎管內阻滯，因爲有已存在的神經系統病變惡化或發展爲新的神經系統功能障礙的風險。相反，既往脊柱手術史被認爲會增加椎管內阻滯的困難和失敗率。我們在本回顧性研究中調查了既往存在椎管病變伴或無脊柱手術史的病人行椎管內麻醉後的神經系統併發症的風險和阻滯效能。
方法：以 15 年內具有椎管狹窄或腰神經根疾病史且行椎管阻滯的所有病例爲研究物件。記錄病人人口統計、術前神經病學診斷和手術／椎管內阻滯時的神經系統發現、椎管內阻滯細節（腰麻比硬膜外麻醉，單次注射比.持續輸注）、注射劑、操作併發症（誘發感覺異常、進針或置管見血、無法置管、硬膜意外穿破）和阻滯成功率。識別新的或進行性的神經系統病變。所有病人隨訪至症狀緩解或評估的最後日期。
結果：共納入 937 位病例，其中 207 例(22%)曾接受脊柱手術。椎管狹窄病人 187 例（20%），腰神經根病變者 570 例（61%），周圍神經病變 210 例（22%）；180 位病人（19%）存在多種神經病學診斷。大多數病人手術時有活動的但穩定的神經系統症狀。總阻滯成功率爲 92.7%。脊柱手術史不影響阻滯成功率和穿刺操作併發症的發生率。10 位病人(1.1%; 95% 可信區間[CI] 0.5%–2.0%)出現新的神經系統缺陷或現有症狀惡化。3 例（1.4%）併發症發生於有脊柱手術史的病人，餘下 7 例（1.0%）併發症發生於無手術減壓或固定術史的病人(P = NS)。儘管畸形矯正術不是一個危險因數，6 位單側下肢手術的病人中有 5 位術後併發症出現於手術側。同樣地，由於發展爲雙側缺陷而行雙側畸形矯正的病人，在先前受累一側的預後較差。假定 10 位病人中 4 位（40%）併發症的主要原因是手術操作。餘下 6 位病人（60%）併發症的主要原因經鑒定爲非手術原因（包括麻醉相關因素）。術前存在壓迫性神經根病變的診斷(P = 0.0495)或具有多種神經病學診斷(P = 0.005)增加了術後神經系統併發症的風險。
結論：我們推斷，既往存在椎管病變的病人比不存在這些基礎病變的病人在椎管阻滯後神經系統併發症發生率較高(1.1%; 95% CI 0.5%–2.0%)。然而，在缺乏具有相似解剖病變並行全身麻醉的手術病人作爲對照組的情況下，我們無法確定這種較

高的神經系統損傷發生率是否是繼發於手術操作、麻醉方法、脊柱病變的自然病程或者是這些相關因素的複合效應以及每個因素的相對作用。
(江繼宏 譯 馬皓琳 李士通 校）

BACKGROUND: Patients with spinal canal pathology, including spinal stenosis and lumbar disk disease, are often not considered candidates for neuraxial blockade because of the risk of exacerbating preexisting neurologic deficits or developing new neurologic dysfunction. In contrast, a history of spine surgery is thought to increase the likelihood of difficult or unsuccessful block. In this retrospective study we investigated the risk of neurologic complications and block efficacy in patients with preexisting spinal canal pathology, with or without a history of spine surgery, after neuraxial anesthesia.
METHODS: During the 15-year study period, all patients with a history of spinal stenosis or lumbar radiculopathy undergoing a neuraxial technique were studied. Patient demographics, preoperative neurologic diagnoses and neurologic findings at the time of surgery/neuraxial block, details of the neuraxial block including technique (spinal vs. epidural, single injection vs. continuous), injectate, technical complications (paresthesia elicitation, bloody needle/catheter placement, inability to advance catheter, accidental dural puncture), and block success were noted. New or progressive neurologic deficits were identified. All patients were followed until resolution or last date of evaluation.
RESULTS: There were 937 patients included, 207 (22%) of whom had undergone spinal surgery. A history of spinal stenosis was present in 187 (20%), lumbar radiculopathy in 570 (61%), and peripheral neuropathy in 210 (22%) patients; 180 patients (19%) had multiple neurologic diagnoses. A majority of patients had active but stable neurologic symptoms at the time of surgery. Overall block success was 97.2%. A history of spine surgery did not affect the success rate or frequency of technical complications. Ten (1.1%; 95% confidence interval [CI] 0.5%–2.0%) patients experienced new deficits or worsening of existing symptoms. Three (1.4%) complications occurred in patients with a history of spinal surgery, and the remaining 7 (1.0%) in patients without prior surgical decompression or stabilization (P = NS). Although an orthopedic procedure was not a risk factor, in 5 of the 6 patients in which the surgery was a unilateral lower extremity procedure, the postoperative deficit involved the operative side. Likewise, in both patients undergoing bilateral orthopedic procedures who developed bilateral deficits, the outcome was worse on the previously affected side. A surgical cause was presumed to be the primary etiology in 4 (40%) of 10 patients. The primary etiology of the remaining 6 (60%) complications was judged to be nonsurgical (including anesthetic-related factors). The presence of a preoperative diagnosis of compressive radiculopathy (P = 0.0495) or multiple neurologic diagnoses (P = 0.005) increased the risk of neurologic complications postoperatively.
CONCLUSIONS: We conclude that patients with preexisting spinal canal pathology have a higher incidence of neurologic complications after neuraxial blockade (1.1%; 95% CI 0.5%–2.0%) than that previously reported for patients without such underlying pathology. However, in the absence of a control group of surgical patients with similar anatomic pathology undergoing general anesthesia, we cannot determine whether the higher incidence of neurologic injury is secondary to the surgical procedure, the anesthetic technique, the natural history of spinal pathology, or a combination of factors and the relative contributions of each.

人血中右旋布洛芬、布洛芬及氟比洛芬抗血小板差異的體外研究

Differences in the in vitro antiplatelet effect of dexibuprofen, Ibuprofen, and flurbiprofen in human blood.

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背景：此項研究旨在通過比較體外環境下右旋布洛芬、布洛芬及氟比洛芬的藥效學特性，明確其各自抗血小板效應間的相互差異。
方法：此項研究的全血標本來源於健康志願者。此項研究須在乙醯水楊酸、右旋布洛芬、布洛芬及氟比洛芬濃度增加的前後，分別檢測全血標本中二磷酸腺苷、膠原及花生四烯酸介導的血小板聚集功能，血小板血栓烷 B（2）（TxB（2））、脂多糖介導的前列腺素 E（2）、白細胞 6-酮-前列腺素 F（1a）（PGF（1a））及構成和誘導兩種途徑介導的一氧化氮的生成。每抑制（IC（50））或增加 50%時的藥物濃度則被要求計算。
結果：所有這 3 種藥物均劑量依賴性地抑制血小板聚集、抑制 TxB（2）、前列腺素 E（2）及 6-酮-PGF（1a）合成，增加鈣離子介導的一氧化氮生成。與布洛芬和氟比洛芬相比，右旋布洛芬展現出更強的抗血小板效應，其藥效學特徵與阿司匹林更爲近似。例如，右旋布洛芬對於花生四烯酸介導的血小板聚集功能的 IC（50）值爲 0.85 ± 0.06 uM，布洛芬爲 14.76 ± 1.22 uM，氟比洛芬爲 6.39 ± 0.51 uM，阿司匹林爲 0.38 ± 0.03 uM。所有這 3 種藥物均可抑制血栓烷與前列腺素合成，但右旋布洛芬其抗- TxB（2）的 IC（50）值與其抗-6-酮-PGF（1a）的 IC（50）值之比爲 0.21 ± 0.03，布洛芬爲 1.05 ± 0.08，氟比洛芬爲 0.79 ± 0.11，阿司匹林爲 0.46 ± 0.06。所有這 3 種藥物均可增加鈣離子依賴的一氧化氮生成。
結論：芳香基丙酸衍生物右旋布洛芬是最強效的抗血小板藥物，其藥理學特性與阿司匹林相似。
（范羽譯 薛張綱校）

BACKGROUND: In this study, we compared the in vitro pharmacodynamic profile of dexibuprofen, ibuprofen, and flurbiprofen to identify possible differences in antiplatelet activity.
METHODS: In whole blood samples from healthy volunteers, we measured platelet aggregation induced by adenosine diphosphate, collagen and arachidonic acid, platelet thromboxane B(2) (TxB(2)), lipopolysaccharide-induced prostaglandin E(2), leukocyte 6-keto-prostaglandin F(1a) (PGF(1a)), and nitric oxide induced by both constitutive and inducible pathways before and after incubation with increasing concentrations of acetylsalicylic acid, dexibuprofen, ibuprofen, or flurbiprofen. The concentration that inhibited (IC(50)) or increased each variable by 50% was calculated.
RESULTS: All 3 drugs inhibited platelet aggregation in a dose-dependent manner, TxB(2), prostaglandin E(2), and 6-keto-PGF(1a), and increased calcium-induced nitric oxide production. Dexibuprofen showed greater antiplatelet potency than ibuprofen and

flurbiprofen, and its profile was similar to that of aspirin. For example, IC(50) values for arachidonic acid-induced platelet aggregation were 0.85 ± 0.06 uM for dexibuprofen, 14.76 ± 1.22 uM for ibuprofen, 6.39 ± 0.51 uM for flurbiprofen, and 0.38 ± 0.03 uM for aspirin. All drugs inhibited both thromboxane and prostacyclin synthesis, but the IC(50) anti-TxB(2)/IC(50) anti-6-keto-PGF(1a) ratio was 0.21 ± 0.03 for dexibuprofen, 1.05 ± 0.08 for ibuprofen, 0.79 ± 0.11 for flurbiprofen, and 0.46 ± 0.06 for aspirin. All drugs increased calcium-dependent nitric oxide production.
CONCLUSIONS: The aryl propionic acid derivative dexibuprofen was the most potent antiplatelet drug, and its pharmacodynamic profile is similar to aspirin.

門診手術麻醉學會制定了糖尿病患者門診手術圍手術期血糖管理的共識聲明

Special article: society for ambulatory anesthesia consensus statement on perioperative blood glucose management in diabetic patients undergoing ambulatory surgery.

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關於門診手術患者的圍手術期血糖控制的最佳方案仍然存在著爭議。因此，門診手術麻醉學會制定了一個門診手術患者圍手術期血糖管理的共識聲明，並且根據Cochrane協作網推薦的方法對文獻進行了系統性回顧。共識小組對於這些建議分別給予了推薦等級、評估、發展和評價分級系統。據透露，並沒有足夠的證據顯示這能夠爲臨床問題提出有力的建議。由於缺乏高品質的證據，提出這些建議是基於糖尿病患者血糖控制的一般原則、藥物的藥理學、住院手術病人的人群資料以及臨床經驗和判斷。此外，也確定了在這一區域未來需要更進一步的研究。
（黃劍譯 薛張綱校）

Optimal evidence-based perioperative blood glucose control in patients undergoing ambulatory surgical procedures remains controversial. Therefore, the Society for Ambulatory Anesthesia has developed a consensus statement on perioperative glycemic management in patients undergoing ambulatory surgery. A systematic review of the literature was conducted according the protocol recommended by the Cochrane Collaboration. The consensus panel used the Grading of Recommendations, Assessment, Development, and Evaluation (GRADE) system for providing suggestions. It was revealed that there is insufficient evidence to provide strong recommendations for the

posed clinical questions. In the absence of high-quality evidence, recommendations were based on general principles of blood glucose control in diabetics, drug pharmacology, and data from inpatient surgical population, as well as clinical experience and judgment. In addition, areas of further research were also identified.

丹曲林的細胞保護機制：一種Ryanodine受體拮抗劑

The Cytoprotective Effects of Dantrolene:A Ryanodine Receptor Antagonist

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鈣，作爲一個第二信使，在眾多細胞功能中有很重要的作用。然而，細胞內鈣失衡會引起細胞毒性甚至細胞死亡。細胞內儲存的鈣離子過度釋放，鈣離子通道ryanodine受體啓動都會促成細胞損傷。鈣失衡引起的機能障礙已在組織培養和缺血、缺氧、損傷、麻醉及神經變性疾病動物模型中得以確定。丹曲林，作爲治療惡性高熱的首選藥物，是ryanodine受體拮抗劑。丹曲林抑制細胞內主要的鈣庫-肌漿網內鈣離子的異常釋放。丹曲林在不同的組織培養和動物疾病模型中對抗由細胞內鈣失衡介導的細胞毒性 引起的細胞損傷可能的細胞保護機制已被廣泛研究。在這篇綜述中，我們總結了細胞內鈣失衡在細胞死亡中的作用，丹曲林的藥理學和藥代動力學，以及丹曲林在眾多應激及疾病模型中抑制細胞損傷的細胞保護機制及潛在的應用價值。
（毛慧譯 薛張剛校）
Calcium, as a second messenger, has an important role in a variety of cellular functions.However, disruption of intracellular calcium homeostasis leads to cytotoxicity and cell death.Excessive calcium release from intracellular stores, via the calcium channel ryanodine receptor, contributes to cell damage. Dysfunction of calcium homeostasis is established in tissue culture and animal models of ischemia, hypoxia, seizure, trauma, anesthesia, and neurodegenerative diseases. Dantrolene, the primary drug to treat malignant hyperthermia, is a ryanodine receptor antagonist. Dantrolene inhibits abnormal calcium release from the sarco-endoplasmic reticulum, which is the primary intracellular calcium store. Dantrolene has been investigated widely for its possible cytoprotective effects against cell damage in different tissue culture or animal models of diseases involving cytotoxicity induced by disruption of intracellular calcium homeostasis in pathogenesis. In this review, we summarize the role of the disruption of intracellular calcium homeostasis on cell death, the pharmacologic and pharmacokinetic features of dantrolene, and the cytoprotective effects and potential application of dantrolene for the inhibition of cell damage in a wide variety of models of stress and disease. (Anesth Analg 2010;111:1400–10)

在受試者血液稀釋過程中使用脈搏碳氧血氧儀持續無創監測總血紅蛋白含量的準確性

The Accuracy of Noninvasive and Continuous Total Hemoglobin Measurement by Pulse CO-Oximetry in Human Subjects Undergoing Hemodilution

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背景：總血紅蛋白含量測定(tHb)是最常用的實驗室檢查之一。脈搏碳氧血氧儀可通過多波長分光光度計測量方法持續無創監測血紅蛋白含量(SpHb)。本研究中，我們通過測量血液稀釋的 20 例健康志願者總血紅蛋白含量，並與實驗室檢查對照，評估了 SpHb 的準確性。
方法：受試者入選試驗後，約 500ml 血液被經靜脈或動脈導管導出。隨後他們都接受了靜脈快速輸晶體液以代償減少的血容量，這同時降低了血紅蛋白濃度。補液量最多爲 30ml/kg。過程中持續監測記錄 SpHb，並採集了一系列的動脈血樣。把動脈血樣測得的 tHb 與 SpHb 配對並進行分析以檢測的 SpHb 準確性。同時計算了偏倚、精確度和平均標準誤。
結果：實驗共收集了 165 個 tHb 結果。在失血和血液稀釋後 tHb 平均減少了(2.4±0.8)g/dL (均值±標準差)。在 335 對測量資料中，SpHb 與 tHb 的平均差異是-0.15g/dL，差異的 1 個單位標準差是 0.92g/dL，平均均方根差異是 0.94g/dL。對於 97%的測量資料，SpHb 與 tHb 的差異小於 2.0 g/dL。在 tHb 小於 10 g/dL 的情況下，對於 97%的測量資料，SpHb 與 tHb 的差異小於 1.5g/dL。
結論：在受試者血液稀釋過程中，以脈搏碳氧血氧儀爲基礎的 SpHb 測量與實驗室碳氧血氧儀測量值相差在 1 g/dL (1 個標準差)內。
（任雲譯 薛張剛校）
BACKGROUND: Total hemoglobin (tHb) is one the most frequently ordered laboratory measurements. Pulse CO-Oximetry™ (Masimo Corp., Irvine, CA) is a multi-wavelength spectrophotometric method for noninvasive and continuous hemoglobin monitoring (SpHb). In this study, we evaluated the accuracy of SpHb compared with laboratory CO-Oximeter measurement of tHb from arterial blood samples in 20 healthy volunteer subjects undergoing hemodilution.
METHODS: After enrollment, approximately 500 mL of blood was drawn from subjects through an arterial or venous catheter. Each subject then rapidly received crystalloid IV fluid to compensate for the decrease in intravascular volume and reduce the hemoglobin concentration. Subjects received a maximum of 30 mL/kg IV fluid. SpHb was continuously monitored and recorded, and serial arterial blood samples were taken during the procedure. SpHb accuracy was analyzed by pairing SpHb and tHb measurements after the arterial blood draw with the resulting tHb test result. Bias, precision, and the average root-mean-square error were calculated.
RESULTS: One hundred sixty-five tHb measurements were collected. The average decrease in tHb during the blood removal and hemodilution procedure was 2.4 ± 0.8 g/dL (mean ± SD). The average difference between 335 paired measurements of SpHb and tHb was -0.15 g/dL, 1 SD of the difference was 0.92 g/dL, and the average root-mean-square difference was 0.94 g/dL. The difference between SpHb and tHb was <2.0 g/dL for 97% of the measurements. The difference was <1.5 g/dL for 97% of the measurements when tHb was <10 g/dL.

CONCLUSIONS: Pulse CO-Oximetry-based SpHb measurement is accurate within 1.0 g/dL (1 SD) compared with laboratory CO-Oximeter tHb measurement in subjects undergoing hemodilution.

一項隨機雙盲多對照實驗：剖宮產術後子宮收縮乏力的病人單次給予 5 個單位劑量的催產素

Five Unit Bolus Oxytocin at Cesarean Delivery in Women at Risk of Atony: A Randomized, Double-Blind, Controlled Trial

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背景：靜脈內常規使用催產素可以預防剖宮產術後出血。它會引起血流動力學的不良反應也是人們所熟知的，所以導致了最近的劑量從 10 個單位減少到了 5 個單位。是否這 5 個單位的催產素比持續輸注更有優勢，我們不是很清楚。所以我們提出了我們的假設：是否剖宮產後子宮收縮乏力的病人在給予催產素持續輸注前單次給予 5 個單位的催產素能減少產後 24 小時內對其他子宮收縮藥物的需求量，將這組病人與持續輸注催產素組相比。
方法：一項隨機雙盲對照實驗在 143 名剖宮產後有子宮收縮乏力的病人中進行。病人在臍帶血流夾閉 30 秒後靜脈內分別給予 5 個單位的催產素或者生理鹽水。之後所有的病人給予 40 個單位的催產素加入 500 毫升的生理鹽水持續輸注 30 分鐘，然後給予 20 個單位的催產素加入 1 升的生理鹽水持續輸注 8 個小時。我們主要目的是比較患者在產後 24 小時內對其他子宮收縮藥物的需求量。同時還要觀察子宮的收縮力（由婦產科醫生來評估，0 分=鬆軟，4 分=像石頭一樣堅硬），失血量，靜脈一次注射後的不良反應，靜脈一次輸注後到胎盤娩出所需的時間。
結果：兩組病人在產後 24 小時內對其他子宮收縮藥物的需求量是沒有差異的。但兩組在胎盤娩出後子宮收縮力有顯著性差異（P＜0.01 ，催產素組是 2.8，95%可信區間 2.6-3.0；生理鹽水組是 2.2，95%可信區間 1.8-2.5），5 分鐘後這種差異消失。兩組之間能觀察到的和被報導的不良反應沒有差異。
結論：我們發現在給予持續輸注催產素前靜脈內先單次給予 5 個單位的催產輸並不能治療和預防剖宮產術後病人子宮收縮乏力，減少對其他子宮收縮藥物的需求量，但是催產素組可引起子宮在最初時更強烈的收縮。我們的研究中兩組的不良反應沒有顯著差異。這個研究可能意味著持續輸注催產素的量已經很充足，沒有必要單次注射，即使是高危病人。
（翁梅琳譯 薛張剛校）

Background: IV bolus oxytocin is used routinely during cesarean delivery to prevent postpartum hemorrhage. Its adverse hemodynamic effects are well known, resulting in a recent change in dose from 10 IU to 5. Whether a 5 IU bolus has any advantages over

infusion alone is unclear. We tested the hypothesis that a 5 IU IV bolus of oxytocin before the initiation of a continuous infusion decreases the need for additional uterotonic drugs in the first 24 hours after delivery in women with risk factors for uterine atony undergoing cesarean delivery, compared with infusion alone.

Methods: A prospective, randomized, double-blind, controlled trial was conducted in 143 subjects undergoing cesarean delivery with at least 1 risk factor for uterine atony. Subjects received 5 IU bolus of oxytocin or normal saline IV over 30 seconds after umbilical cord clamping. All subjects received an infusion of 40 IU oxytocin in 500 mL normal saline over 30 minutes, followed by 20 IU in 1 L over 8 hours. The primary outcome was the need for additional uterotonics in the first 24 hours after delivery. Secondary outcomes included uterine tone as assessed by the surgeon (5-point Likert scale: 0 = "floppy," 4 = "rock hard"), estimated blood loss, side effects of bolus administration, and the oxytocin bolus–placental delivery interval.

Results: There was no difference in the need for additional uterotonic drugs in the first 24 hours between groups. There was a significant difference in uterine tone immediately after placental delivery ($P < 0.01$) (2.8 in the oxytocin group [95% confidence interval 2.6–3.0] vs 2.2 in the saline group [95% confidence interval 1.8–2.5]), which disappeared after 5 minutes. There were no differences in observed or reported side effects between groups.

Conclusions: We found that a 5 IU IV bolus of oxytocin added to an infusion did not alter the need for additional uterotonic drugs to prevent or treat postpartum hemorrhage in the first 24 hours in women undergoing cesarean delivery with risk factors for uterine atony, despite causing an initial stronger uterine contraction. Our study was not powered to find a difference in side effects between groups. These results suggest that an oxytocin infusion may be adequate without the need for a bolus, even in high-risk patients.

2009 年產科麻醉進展----分娩患者安全的知識更新

What's New in Obstetric Anesthesia in 2009? An Update on Maternal Patient Safety

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每年,產科麻醉學會和圍生醫學會都會推薦一個人對前一年的文獻進行調查找出對產科麻醉科學和臨床實踐最重要的文章.本文回顧了 2009 年針對分娩病人安全的文獻,並且主張產科麻醉醫師不但是圍術期而且是圍產期的內科醫師.回顧了三個特別的話題:產科麻醉的併發症,全身性產科併發症和圍產期質控和安全.

(姚敏敏譯 薛張綱校)

Every year, the Society of Obstetric Anesthesia and Perinatology nominates 1 individual to survey the prior year's literature and to identify the most notable articles for the science and practice of obstetric anesthesiology. This article reviews the 2009 literature, focusing on the theme of maternal patient safety, and advancing the notion of the obstetric anesthesiologist as both a perioperative and a peripartum physician. Three specific topics are reviewed: complications of obstetric anesthesia, general obstetric complications, and quality and safety in peripartum care.

顱腦外傷中肌酐清除率增長

Augmented Creatinine Clearance in Traumatic Brain Injury

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背景：輸注高張生理鹽水和/或去甲腎上腺素在處理顱腦外傷時被常規用來獲得理想的腦灌注壓。我們假定肌酐清除率在這種情況下會顯著增高。
方法：這是個對 16 歲以上的擁有正常血肌酐並需要維持腦灌注壓的顱腦外傷病人進行的佇列觀察研究。在積極處理開始和結束時收集 8 小時尿肌酐清除率。在研究過程中記錄人口資料、血管活性藥的使用情況、液體平衡、營養治療和血流動力學變化。肌酐清除率升高定義爲女性>150 mL/min/1.73 m(2)，男性>160 mL/min/1.73 m(2)。
結果：20 名患者入組，17 名發現肌酐清除率升高（85%）。接受腦灌注壓治療的患者平均最高肌酐清除率爲 179 mL/min/1.73 m(2)（95%置信區間 159-198），而該數據在離開重症監護室後則回到了 111 mL/min/1.73 m(2) (95% CI, 91-131; P < 0.001)。沒有接受腦灌注壓治療的患者平均最高肌酐清除率爲 150 mL/min/1.73 m(2)（95%置信區間 134-167; P = 0.03）。接受積極治療的患者肌酐清除率達到峰值的平均時間是 4.7 天(95% CI, 3.0-6.4)。在一個多元分析中，去甲腎上腺素的應用、鹽水負荷、平均動脈壓和中心靜脈壓與肌酐清除率升高相關。
結論：顱腦外傷病人接受積極地腦灌注壓治療後肌酐清除率通常升高並持續至治療結束。進一步的工作需要展開來明晰這種情況下該治療對腎臟清除藥物的影響。
（張玥琪譯，薛張綱校）

Background: Hypertonic saline and/or norepinephrine infusion are routinely used to achieve a desired cerebral perfusion pressure (CPP) in the management of traumatic brain injury (TBI). We hypothesized that creatinine clearances (CrCls) would be significantly augmented in this setting.
Methods: This was an observational cohort study in TBI patients older than 16 years with normal serum creatinine concentrations, requiring maintenance of CPP. Eight-hour urinary CrCl collections were performed while on and off active management. Demographic data, use of vasoactive medications, fluid balance, feeding regimen, and hemodynamic variables were recorded throughout the study period. Augmented CrCl was defined as >150 mL/min/1.73 m(2) in women and >160 mL/min/1.73 m(2) in men.
Results: Twenty patients were enrolled, and augmented clearances were demonstrated in 17 (85%). The mean maximum CrCl was 179 mL/min/1.73 m(2) while receiving CPP therapy (95% confidence interval [CI], 159-198), returning to a mean of 111 mL/min/1.73 m(2) (95% CI, 91-131; P < 0.001) when measured after discharge from the intensive care unit. The mean CrCl in the intensive care unit while not receiving CPP

therapy was 150 mL/min/1.73 m(2) (95% CI, 134-167; P = 0.03). The mean time to reach peak CrCl while receiving active treatment was 4.7 days (95% CI, 3.0-6.4). In a multivariate analysis, norepinephrine use, saline loading, mean arterial blood pressure, and central venous pressure were associated with augmented CrCl on the day of measurement.

Conclusions: Augmented CrCls are common in TBI patients receiving active management of CPP and persist even after discontinuation of such therapy. Further work is needed to clarify the impact of such clearances on renally excreted drugs in this setting.