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October 2009

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Anesth Analg 2009 109: 1135-1146.

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Anesth Analg 2009 109: 1202-1208.

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Anesth Analg 2009 109: 1219-1224.

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www.bjpa.org

Qc TG

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Anesth Analg 2009 109: 1284-1286.

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Anesth Analg 2009 109: 1287-1296.

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Laszlo Mecs, Gabor Tuboly, Endre Nagy, Gyorgy Benedek, and Gyongyi Horvath

Anesth Analg 2009 109: 1297-1304.

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Duchenne muscular dystrophy (DMD) and Becker dystrophy (BD) represent nearly all the anesthesia-related reports.

RESULTS: Anesthetic complications in patients with DMD and BD include intraoperative heart failure, inhaled anesthetic-related rhabdomyolysis (absence of succinylcholine), and succinylcholine-induced rhabdomyolysis and hyperkalemia.

CONCLUSION: We did not find an increased risk of malignant hyperthermia susceptibility in patients with DMD or BD compared with the general population. However, dystrophic patients who are exposed to inhaled anesthetics may develop disease-related cardiac complications, or rarely, a malignant hyperthermia-like syndrome characterized by rhabdomyolysis. This latter complication may also occur postoperatively. Succinylcholine administration is associated with life-threatening hyperkalemia and should be avoided in patients with DMD and BD.

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The Relationship Between Exertional Heat Illness, Exertional Rhabdomyolysis, and Malignant Hyperthermia

John F. Capacchione, MD, and Sheila M. Muldoon, MD

From the Department of Anesthesiology, Uniformed Services University of the Health Sciences, Bethesda, Maryland.

Anesth Analg 2009 109: 1065-1069.

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Exertional heat illness, exertional rhabdomyolysis, and malignant hyperthermia (MH) are complex syndromes with similar pathophysiology. All three are hypermetabolic states that include high demand for adenosine triphosphate, accelerated oxidative, chemical, and mechanical stress of muscle, and uncontrolled increase in intracellular calcium. Although there are no controlled clinical studies to support a relationship, there is evidence to suggest an association between unexpected heat/exercise intolerance and MH susceptibility. There are multiple case reports and a small number of clinical studies that have used *in vitro* muscle contracture testing and/or genetic testing to make the association. However, such methodology is problematic in that these tests are validated for clinical MH in association with anesthesia, and not for exertional heat illness or exertional rhabdomyolysis. Nevertheless, these relationships may have implications for some MH-susceptible patients and their capacity to exercise, as well as for clinicians treating and anesthetizing patients with histories of unexplained exertional heat and exercise illnesses.

different at the 30 min period compared with the 120 min period (1.8 ± 0.1 atm vs 1.8 ± 0.2 atm).

CONCLUSIONS: Destroying brainstem noradrenergic neurons or prolonged exposure to N_2O removes its analgesic effects, but does not change MAC. The immobilizing mechanism of N_2O is independent from its analgesic effects.

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Monitoring with Head-Mounted Displays: Performance and Safety in a Full-Scale Simulator and Part-Task Trainer

David Liu, BE (Hons)*, Simon A. Jenkins, BMBS, FANZCA, Penelope M. Sanderson, PhD, FASSA*||, Marcus O. Watson, PhD||, Terrence Leane, RN, **GDPH**, GDNursSci¶, Amanda Krays, MBBS, and W. John Russell, MBBS, DPhil, DIC, FANZCA, FRCA
From the *School of Information Technology and Electrical Engineering, The University of Queensland, Brisbane; Department of Anaesthesia, Pain Medicine and Hyperbaric Medicine, Royal Adelaide Hospital; Centre for Sleep Research, University of South Australia, Adelaide; School of Psychology, The University of Queensland; ||School of Medicine, The University of Queensland, Brisbane; and ¶Nursing and Patient Care Services, Royal Adelaide Hospital, Adelaide, Australia.
Anesth Analg 2009 109: 1135-1146.

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HMD

BACKGROUND: Head-mounted displays (HMDs) can help anesthesiologists with intraoperative monitoring by keeping patients' vital signs within view at all times, even while the anesthesiologist is busy performing procedures or unable to see the monitor. The anesthesia literature suggests that there are advantages of HMD use, but research into head-up displays in the cockpit suggests that HMDs may exacerbate inattentive blindness (a tendency for users to miss unexpected but salient events in the field of view) and may introduce perceptual issues relating to focal depth. We investigated these issues in two simulator-based experiments.

METHODS: Experiment 1 investigated whether wearing a HMD would affect how quickly anesthesiologists detect events, and whether the focus setting of the HMD (near or far) makes any difference. Twelve anesthesiologists provided anesthesia in three naturalistic scenarios within a simulated operating theater environment. There were 24 different events that occurred either on the patient monitor or in the operating room. Experiment 2 investigated whether anesthesiologists physically constrained by performing a procedure would detect patient-related events faster with a HMD than without. Twelve anesthesiologists performed a complex simulated clinical task on a part-task endoscopic dexterity trainer while monitoring the simulated patient's vital signs. All participants experienced four different events within each of two scenarios.

RESULTS: Experiment 1 showed that neither wearing the HMD nor adjusting the focus setting reduced participants' ability to detect events (the number of events detected and time to detect events). In general, participants spent more time looking toward the patient and less time toward the anesthesia machine when they wore the HMD than when they used standard monitoring alone. Participants reported that they preferred the near focus setting. Experiment 2 showed that participants detected two of four events faster with the HMD, but one event more slowly with the HMD. Participants turned to look toward the anesthesia machine significantly less often when using the HMD. When using the HMD, participants reported that they were less busy, monitoring was easier, and they believed they were faster at detecting abnormal changes.

CONCLUSIONS: The HMD helped anesthesiologists detect events when physically constrained, but not when physically unconstrained. Although there was no conclusive evidence of worsened inattentive blindness, found in aviation, the perceptual properties of the HMD display appear to influence whether events are detected. Anesthesiologists wearing HMDs should self-adjust the focus to minimize eyestrain and should be aware that some changes may not attract their attention. Future areas of research include developing principles for the design of HMDs, evaluating other types of HMDs, and evaluating the HMD in clinical contexts.

2001–2005 ON

Prevalence of Malignant Hyperthermia Due to Anesthesia in New York State, 2001–2005

Joanne E. Brady, SM*, Lena S. Sun, MD*, Henry Rosenberg, MD, and Guohua Li, MD, DrPH*||

From the *Department of Anesthesiology; Department of Pediatrics, College of Physicians and Surgeons Columbia University, New York, New York; Department of Medical Education and Clinical Research, Saint Barnabas Medical Center, Livingston, New Jersey; Malignant Hyperthermia Association of the United States, Sherburne, New York; and ||Department of Epidemiology, Mailman School of Public Health, Columbia University, New York, New York.

Anesth Analg 2009 109: 1162-1166.

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BACKGROUND: Malignant hyperthermia (MH) is a pharmacogenetic syndrome that variably expresses itself on exposure to triggering agents. MH prevalence in the United States is not well documented. In this study, we assessed the prevalence of MH in New York State hospitals.

METHODS: Using New York hospital discharge data for the years 2001 through 2005, we identified all patients with a diagnosis of MH due to anesthesia using International Classification of Diseases, Ninth Revision, Clinical Modification code 995.86. MH prevalence was evaluated by demographic and clinical characteristics.

RESULTS: Of the 12,749,125 discharges from New York hospitals during the study period, 73 patients had a recorded diagnosis of MH due to anesthesia. Nearly three quarters of the MH patients were male and 71% were patients from emergency/urgent admissions. The estimated prevalence rate of MH was 0.96 (95% confidence interval [CI] 0.67–1.24) per 100,000 surgical discharges and 1.08 (95% CI 0.75–1.41) per 100,000 discharges in which there was any indication of exposure to anesthesia. The estimated prevalence of MH for males was 2.5 to 4.5 times the rate for females.

CONCLUSION: The prevalence of MH due to anesthesia in surgical patients treated in New York State hospitals is approximately 1 per 100,000. MH risk in males is significantly higher than in females.

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Obstructive Sleep Apnea Is Not a Risk Factor for Difficult Intubation in Morbidly Obese Patients

Patrick J. Neligan, MA, MB, FFARSCI*, Steven Porter, MD, Bryan Max, MD, Guarav Malhotra, MD, Eric P. Greenblatt, MD*, and E. Andrew Ochroch, MD, MSCE*

intubation attempts, a difficult intubation rate of 3.3%. There was an 8.3% incidence of difficult laryngoscopy, defined as a Cormack and Lehane Grade 3 or 4 view. There was no relationship between NC and difficult intubation (odds ratio 1.02, 95% confidence interval 0.93-1.1), between the diagnosis of OSA and difficult intubation ($P = 0.09$), or between BMI and difficult intubation (odds ratio 0.99, 95% confidence interval 0.92-1.06, $P = 0.8$). There was no relationship between number of intubation attempts and BMI ($P = 0.8$), AHI ($P = 0.82$), or NC ($P = 0.3$). Mallampati Grade III or more predicted difficult intubation ($P = 0.02$), as did male gender ($P = 0.02$). Finally, there was no relationship between Cormack and Lehane grade and BMI ($P = 0.88$), AHI ($P = 0.93$), or OSA ($P = 0.6$). Increasing NC was associated with difficult laryngoscopy but not difficult intubation ($P = 0.02$).

CONCLUSIONS: In MO patients undergoing bariatric surgery in the "ramped position," there was no relationship between the presence and severity of OSA, BMI, or NC and difficulty of intubation or laryngoscopy grade. Only a Mallampati score of 3 or 4 or male gender predicted difficult intubation.

PEEP IMPROVES

CPR USING

Positive End-Expiratory Pressure Improves Survival in a Rodent Model of Cardiopulmonary Resuscitation Using High-Dose Epinephrine

Conán McCaul, MB^{*} ||, Alik Kornecki, MD^{*¶}, Doreen Engelberts, AHT^{*}, Patrick McNamara, MB^{*||#}, and Brian P. Kavanagh, MB^{* ||**}

From the *Program in Physiology and Experimental Medicine, Departments of Critical Care Medicine, and Anesthesia, Hospital for Sick Children; Departments of Anesthesia, and ||Pediatrics, University of Toronto, Toronto; ¶Department of Paediatrics, Critical Care Unit, Children's Hospital of Western Ontario, University of Western Ontario, London; #Department of Pediatrics, Hospital for Sick Children; and **Department of Physiology, and Interdepartmental Division of Critical Care Medicine, University of Toronto, Toronto, Ontario, Canada.

Anesth Analg 2009 109: 1202-1208.

Survival during CPR using high-dose epinephrine

with PEEP in a rodent model

CPR O₂ 14%

with PEEP 8% RVDIKD%t{h6R} f

CPR m4 PEEP <R

1 Sprague - Dawley UUUJ 1 S% [OUR=upgHL,]

1.0 Fio₂ 2 1.0 IB;OBH 30µg/kg O₂ 1 10µg/kg

2 4L9T8JQVhO 1UU CPR KhTBDC

8:uP9m@w9 5cm H₂O PEEP 0 PEEP=Rls8<HcūP

60min C 120min O 2

O, O₂ (c98p=O 1S 0 PEEP Rm w9 5 cm H₂O PEEP

(Fio₂ 1.0 and 0.21) R79 and 6/6 vs 0/9, P < 0.01 and <0.001 K4

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BACKGROUND: Multiple interventions have been tested in models of cardiopulmonary resuscitation (CPR) to optimize drug use, chest compressions, and ventilation. None has studied the effects of positive end-expiratory pressure (PEEP) on outcome. We hypothesized that because PEEP can reverse pulmonary atelectasis, lower pulmonary vascular resistance, and potentially improve cardiac output, its use during CPR would increase survival.

METHODS: Anesthetized Sprague-Dawley rats were exposed to 1 min of asphyxial cardiac arrest. Resuscitation was standardized and consisted of chest compressions, oxygen (Fio₂ 1.0), and IV epinephrine 30 μg/kg (Series 1) and 10 μg/kg (Series 2). Left ventricular function was assessed by echocardiography (Series 1), and animals were randomized to receive either 5 cm H₂O PEEP or zero PEEP at commencement of CPR and throughout resuscitation. Survival was defined as the presence of a spontaneous circulation 60 or 120 min (Series 2) after initial resuscitation.

RESULTS: There were no baseline differences between the groups. In Series 1, administration of 5 cm H₂O PEEP (Fio₂ 1.0 and 0.21) was associated with improved survival compared with zero PEEP (7/9 and 6/6 vs 0/9, *P* < 0.01 and <0.001, respectively). Application of 5 cm H₂O PEEP (Fio₂ 1.0) increased left ventricular end-diastolic area, systemic oxygenation, and functional residual capacity. Use of PEEP during CPR did not adversely affect left ventricular systolic function or arterial blood pressure. The outcome differences were not due to increased oxygenation because the rank order of survival was 5 cm H₂O PEEP (Fio₂ 1.0) > 5 cm H₂O PEEP (Fio₂ 0.21) > zero PEEP (Fio₂ 1.0), whereas the rank order of Pao₂ was 5 cm H₂O PEEP (Fio₂ 1.0) > 5 cm H₂O PEEP (Fio₂ 0.21) > zero PEEP (Fio₂ 1.0). In an additional series in which epinephrine 10 μg/kg was used (Series 2), the survival was 100% with no beneficial effects of PEEP.

CONCLUSION: In asphyxial cardiac arrest in a small rodent model, continuous application of PEEP (5 cm H₂O) during and after CPR had beneficial effects on survival that were independent of oxygenation and without adverse cardiovascular effects.

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HTQ

The Effect of Obesity on Neuraxial Technique Difficulty in Pregnant Patients: A Prospective, Observational Study

Elizabeth H. Ellinas, MD*, Daniel C. Eastwood, MS, Smita N. Patel, MD*, Anita M. Maitra-D’Cruze, MD*, and Thomas J. Ebert, MD, PhD*

From the *Department of Anesthesiology, Medical College of Wisconsin; Department of Obstetric Anesthesiology, Wheaton Franciscan Healthcare–St. Joseph; Division of Biostatistics, Biostatistics Consulting Service, Medical College of Wisconsin; and

Department of Anesthesiology, Clement J. Zablocki VA Medical Center, Milwaukee, Wisconsin.
Anesth Analg 2009 109: 1225-1231.

8XHCDDsE8tSd[CXO(tM-E8 ITU"TB
O8Q79S%e@gtMII+TPH=CQPQ3:T
GbTLSd[CXO(tM-E8 ITU IH
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BACKGROUND: Practitioners often presuppose that obesity will increase neuraxial technique difficulty in pregnant patients, but few investigators have systematically examined this population for risk factors associated with difficult epidural or spinal needle placement. We designed this study to prospectively identify factors that predict neuraxial technique difficulty in pregnant patients.

METHODS: Using a prospective, observational format, pregnant patients were examined for multiple potential risk factors for neuraxial technique difficulty, including current body mass index, ability to palpate spinous processes, maximum back flexion, scoliosis, and experience of the practitioner. Neuraxial technique difficulty was then assessed using two measures: 1) the number of needle passes needed to reach the desired space, and 2) the placement time from skin infiltration to either spinal injection or epidural catheter threading. Predictors of total needle passes were determined by fitting the data to a generalized linear model with negative binomial error. Predictors of neuraxial anesthetic time were determined by fitting a linear model to the log of neuraxial anesthetic placement time. A survival model was used to account for bias introduced when attending physicians intervened in resident physician procedures.

RESULTS: Neuraxial procedures in 427 pregnant patients were studied. For both the number of needle passes and the neuraxial anesthetic placement time, the significant predictors of difficulty were the practitioner's ability to palpate the patient's bony landmarks and the patient's ability to flex her back. Obesity, as measured by body mass index, was not an independent predictor of either end point. Obesity did, however, strongly predict both the ability to palpate landmarks and flex the back.

CONCLUSIONS: Despite concerns that obesity may cause difficulty with neuraxial technique, some obese patients have surprisingly easy neuraxial block placements. When approaching any neuraxial anesthetic in a pregnant patient, and especially in the obese parturient, back flexion and landmark palpation predict neuraxial technique difficulty.

OCISW;8I

BBSSpRm

A Comparison of Intravenous Oxycodone and Intravenous Morphine in Patient-Controlled Postoperative Analgesia After Laparoscopic Hysterectomy

Harald Lenz, MD*, **Leiv Sandvik, MSc, PhD***, **Erik Qvigstad, MD, PhD***, **Carl Eivind Bjerkelund, MD**, and **Johan Raeder, MD, PhD***

From the *University of Oslo, Faculty Division Ullevaal University Hospital; and Department of Anesthesia, Center for Clinical Research, and Department of Gynecology and Obstetrics, Oslo University Hospital, Ullevaal, Oslo, Norway.
Anesth Analg 2009 109: 1279-1283.

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fOIE-4E

#E 91 **ieSW;8IM[O8O,w9O[IB;E**

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INTRODUCTION: In this study, we investigated the dose requirements, pain relief, and side effects of oxycodone versus morphine after surgery with visceral pain.

METHODS: Ninety-one women received IV oxycodone or morphine before the end of laparoscopic hysterectomy and then continued with patient-controlled analgesia for 24 h postoperatively.

RESULTS: The accumulated oxycodone consumption was less (13.3 ± 10.4 mg vs 22.0 ± 13.1 mg, $P = 0.001$) than morphine. With oxycodone, the visual analog scale scores were significantly lower in the first hour postoperatively and sedation was less during the 24-h postoperative period, $P = 0.006$.

CONCLUSIONS: Oxycodone was more potent than morphine for visceral pain relief but not for sedation.

8UO -1 **IEUIPSA1LHnK**

The Peripheral Antinociceptive Effects of Endomorphin-1 and Kynurenic Acid in the Rat Inflamed Joint Model

Laszlo Meacs, MD*, **Gabor Tuboly, MD**, **Endre Nagy, MD**, **Gyorgy Benedek, MD, DSc**, and **Gyongyi Horvath, MD, DSc**

From the *Department of Orthopedics, University of Szeged, Department of Physiology, University of Szeged, and Department of Radiology, Faculty of Medicine, University of Szeged, Szeged, Hungary.

Anesth Analg 2009 109: 1297-1304.

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 160-251| 330µg[CI 280-407]=EM1ED30 ED50 9H 112µg[CI80-146]
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BACKGROUND: Several data suggest that both opioid and *N*-methyl-d-aspartate (NMDA) receptors are localized at the peripheral level, and drugs acting on these receptors may produce antinociception after topical administration; however, the antinociceptive effect of endogenous ligands at these receptors is poorly clarified. Our goal in this study was to determine the antinociceptive potency of the endogenous opioid peptide, endomorphin-1 (EM1), and the endogenous NMDA receptor antagonist, kynurenic acid (KYNA), and their interaction at the peripheral level in the rat inflamed joint model.

METHODS: Mechanical hypersensitivity was produced by injection of carrageenan (300 µg/20 µL) into the tibiotarsal joint of the right hind leg. The mechanical pain threshold was assessed by von Frey filaments (0.064-110 g). EM1 (30, 100, and 200 µg), KYNA (30, 100, 200, and 400 µg), and their combinations in a fixed-dose ratio (1:1) were injected into the inflamed joint, and the pain threshold was determined repeatedly for 75 min after the drug administrations.

RESULTS: Neither EM1 nor KYNA administered to the inflamed joint influenced the pain threshold at the noninflamed side. Both ligands produced dose-dependent antihyperalgesia, and the highest doses caused a prolonged effect. EM1 had higher potency (30% effective dose [ED₃₀] and 50% effective dose [ED₅₀] values were 112 µg [confidence interval {CI}: 80-146] and 167 µg [CI: 135-220], respectively) compared

with KYNA (ED₃₀ and ED₅₀ values were 204 μg [CI: 160-251] and 330 μg [CI: 280-407], respectively). The antinociceptive effect of EM1 was prevented by subcutaneous naltrexone pretreatment. The coadministration of EM1 with KYNA caused an enhanced and/or prolonged antinociceptive effect. The ED₃₀ and ED₅₀ values of the combination were 141 μg [CI: 83-182] and 231 μg [CI: 190-293], respectively, which did not differ significantly from the theoretically additive values (ED₃₀ and ED₅₀ values were 145 μg [CI: 68-237] and 220 μg [CI: 144-230], respectively), thus the interaction between these ligands is additive. None of the treatments caused any sign of side effects.

CONCLUSION: Peripherally administered endogenous opioid agonist and NMDA receptor antagonist ligands might be beneficial in inflammatory pain. Because both drugs barely cross the blood-brain barrier, their local administration causes no central side effects.

UU9W __ □ Licking

Licking Decreases Phosphorylation of Extracellular Signal-Regulated Kinase in the Dorsal Horn of the Spinal Cord After a Formalin Test

Taeko Fukuda, MD*, Setsuji Hisano, PhD, and Makoto Tanaka, MD*

From the *Department of Anesthesiology, Institute of Clinical Medicine, and Laboratory of Neuroendocrinology, Institute of Basic Medical Sciences, Graduate School of Comprehensive Human Sciences, Tsukuba University, Tsukuba-city, Ibaraki, Japan. Anesth Analg 2009 109: 1318-1322.

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BACKGROUND: Nociceptive behaviors might attenuate pain sensation.

Phosphorylation of extracellular signal-regulated kinase (pERK) was recently reported to be induced by noxious stimuli in dorsal horn neurons. We investigated, in a formalin test, whether pERK of the dorsal horn is affected by licking.

METHODS: Twenty-four adult male rats were divided into four groups: control, formalin test, restricted control, and restricted formalin test. Ten percent formalin was

injected subcutaneously into the left rear paw of the formalin test and restricted formalin test groups. The control and formalin test group rats were kept in a clear plastic chamber, whereas the restricted control and restricted formalin test group rats were kept in a modified-restraint, pipe-shaped chamber. All rats were killed after 25 min. Twelve sections of the lumbar spinal cord were processed for p-ERK immunohistochemistry using the avidin-biotin peroxidase method.

RESULTS: The number of p-ERK positive cells in the restricted formalin test group was significantly higher than in the other three groups in the ipsilateral-side superficial dorsal horn ($P < 0.05$). However, there was no significant difference between the formalin test group and the two control groups in pERK expression.

CONCLUSION: Licking decreased pERK of the spinal cord of the formalin test group. The findings suggested that licking attenuated the pain of the formalin test.

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Journal of Clinical Pharmacy and Therapeutics

The Median Effective Dose of Bupivacaine, Levobupivacaine, and Ropivacaine After Intrathecal Injection in Lower Limb Surgery

Ying Y. Lee, MBChB, FFARCSI, MScPainM^{*}, Warwick D. Ngan Kee, MBChB, MD, FANZCA^{*}, Siu Y. Fong, MBBS, FHKCA, John T. C. Liu, MBBS, FFARCSI, FHKCA, and Tony Gin, MBChB, MD, FANZCA, FRCA^{*}

From the ^{*}Department of Anaesthesia and Intensive Care, The Chinese University of Hong Kong, Shatin; and Department of Anaesthesiology and Operating Theatre Services, Kwong Wah Hospital, Kowloon, Hong Kong SAR, China.

Anesth Analg 2009 109: 1331-1334.

Correspondence: Ying Y. Lee, MD, FANZCA, FRCA, Department of Anaesthesia and Intensive Care, The Chinese University of Hong Kong, Shatin, New Territories, Hong Kong SAR, China.

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6.10mg □ ED₅₀ H 5.68mg 95% □ CI 92-6.44mg □ O-:á □

□ ED₅₀ H 8.41mg 95% □ CI 15-9.67mg □ S □

8 □ 0.97 (95% □ CI: □ 0.81-1.17) O-:á S □

□ 0.65 (95% □ CI 0.54-0.80) O-:á S □

□ 0.68 (95% □ CI 0.55-0.84) □

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BACKGROUND: Thrombelastography has received renewed interest in the perioperative setting. The main determinants of thrombelastographic results are coagulation factor concentrations (various zymogens and fibrinogen) and platelet count; thus, platelet inhibition renders these assays mainly coagulation factor dependent. Assays with and without platelet inhibition are thus increasingly used to trigger and monitor replacement therapy with blood products. In this study, we evaluated the effect of factor XIII inhibition and additional glycoprotein (GP) IIB/IIIa blockade on (platelet-inhibited) whole blood thrombelastography and whether a modified routine assay (using factor XIII antibody) can be used to detect factor XIII deficiency.

METHODS: Normal whole blood was incubated with increasing amounts of a nonspecific antibody, an anti-GPIIb/IIIa antibody, or a neutralizing anti-factor XIII antibody; samples were analyzed with a tissue factor-activated and platelet-inhibited whole blood thrombelastographic assay. Clotting time, clot formation time, maximum clot firmness, and clot lysis at 60 min were evaluated in triplicate. Also, 25 whole blood routine samples were evaluated for factor XIII deficiency using a new thrombelastographic assay incorporating a factor XIII antibody and using a standard factor XIII assay for comparison.

RESULTS: Although GPIIb/IIIa inhibition did not alter the results of the platelet-inhibited whole blood thrombelastography, factor XIII inhibition significantly reduced maximum clot firmness (P = 0.020) and increased clot formation time (P = 0.025) and clot lysis (P = 0.007), leaving clotting time unchanged; a ceiling effect seemed to be present with increasing antibody concentrations in whole blood (but not plasma). The thrombelastographic assay for factor XIII deficiency (<70% activity) had a 90% sensitivity and negative predictive value (area under receiver operating characteristic curve 0.803, P = 0.0015); for a deficiency <60%, sensitivity and negative predictive value were 100% (area under receiver operating characteristic curve 0.84, P = 0.0037).

CONCLUSION: Factor XIII has significant impact on platelet-inhibited activated whole blood thrombelastography. This phenomenon should be considered when interpreting thrombelastographic results in the bleeding patient, especially when the results trigger procoagulant therapy. Antibody-mediated factor XIII inhibition can be used to establish thrombelastography-based assays to detect factor XIII deficiency.

Ü>4IKMM4□□

The Myotonias and Susceptibility to Malignant Hyperthermia

Jerome Parness, MD, PhD*, Oliver Bandschapp, MD, and Thierry Girard, MD

From the Departments of *Anesthesiology, Pharmacology, and Chemical Biology, Children's Hospital of Pittsburgh/University of Pittsburgh Medical Center, University of Pittsburgh, School of Medicine, Pittsburgh, Pennsylvania; and Department of Anaesthesia, University Hospital of Basel, Basel, Switzerland.

Anesth Analg 2009 109: 1054-1064

KMM4"±#PÜRHI5CPF6iÜLê8U

Ca²⁺ IR 8IX

IQÜ>4BOÜ±H±Ü>4"±□

DNA gh□

7JS±ÜI%:OO¶III±□□

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Q6H¶Ü>4IBG□□vHKMM4ITTP±pmzHêIÜ

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KMM4II@±zHSII@O,QLISDLZ□□

Ü[CMKMM4I7SO3±7J9¶SD LZMIBI□

vHKMM4I98%zHQ6H:;BII"zHxIhQV□

IvKMM4ITTP±

TG"p"pQc 9KO±

Malignant hyperthermia (MH) is a pharmacogenetic disorder of skeletal muscle in which volatile anesthetics trigger a sustained increase in intramyoplasmic Ca²⁺ via release from sarcoplasmic reticulum and, possibly, entry from the extracellular milieu that leads to hypermetabolism, muscle rigidity, rhabdomyolysis, and death. Myotonias are a class of myopathies that result from gene mutations in various channels involved in skeletal muscle excitation-contraction coupling and sarcolemmal excitability, and unusual DNA sequence repeats that result in the inability of many proteins, including skeletal muscle channels that affect excitability, to undergo proper splicing. The suggestion has often been made that myotonic patients have an increased risk of developing MH. In this article, we review the physiology of muscle excitability and excitation-contraction coupling, the pathophysiology of MH and the myotonias, and review the clinical literature upon which the claims of MH susceptibility are based. We conclude that patients with these myopathies have a risk of developing MH that is equivalent to that of the general population with one potential exception, hypokalemic periodic paralysis. Despite the fact that there are no clinical reports of MH developing in patients with hypokalemic periodic paralysis, for theoretical reasons we cannot be as certain in estimating their risk of developing MH, even though we believe it is low.

Prolonged propofol anesthesia is not associated with an increase in blood lactate.

Irene Rozet, Nuj Tontisirin, Monica S. Vavilala, Miriam M. Treggiari, Lorri A. Lee, and Arthur M. Lam.

Departments of Anesthesiology, Pediatrics and Neurological Surgery, Harborview Medical Center, University of Washington, Seattle, Washington.

irozet@u.washington.edu.

Anesth Analg. 2009 Oct; 109(4):1105-10.

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S9T4#ISKI5(PFMA \pm pH 7.39
T8SI 8 [9IKZi*8[CI(QB:/8Xm>78S3Q
O8Q3GdQ#(9TAT \pm 30min \pm IBL \pm 500ml
12 I#W9#1(CSw9 VA (I[CR#
O, T 246 [Cme<e7 50 W9#(C
AT=10 \pm 2hBL=1955 \pm 1409ml \pm S 100 W9 VA (I[CAT=10 \pm 1h
BL=1801 \pm 1543 \pm m9 40 72 [CKMYJeIOS 8
9@K05#1O[CW9#1AIRH
8.8 \pm 2mg/kg/hVA O[CMUJ#1O[C9H 58 \pm 12 JS 51 \pm 15
JP=0.002 \pm 9C9ASA O8mL8fGdL,I%8PF
#A8p#8 [9S#1OMA O[C8;Rm
OU#SO#1O#H 0.48 \pm 0.72mmol/LMA O#
H 1.2 \pm 1.2mmol/LP=0.001
O,QI8SI 8 [9I*8mSR}#W9 VA (I
U#E#O,QLI%#IH8<}R#B

#Qc 9KO#

BACKGROUND: Lactic acidosis is considered an early sign of propofol infusion syndrome. In this study, we investigated the changes in lactate and pH with propofol versus volatile anesthesia (VA) of long duration.

METHODS: Demographic and intraoperative data were recorded retrospectively from the anesthesia records of patients who underwent elective spine surgery longer than 8 h. Propofol patients were matched 1:2 to VA patients, based on anesthesia time (AT) (\pm 30 min) and blood loss (BL) (\pm 500 mL).

RESULTS: Of 246 patients identified, 50 received propofol (AT = 10 \pm 2 h, BL = 1955 \pm 1409 mL) and were matched to 100 VA cases (AT = 10 \pm 1 h, BL = 1801 \pm 1543 mL), and of those, 40 and 72 patients, respectively, had complete lactate data at baseline and at 8 h after anesthesia and were included in the main analysis. The propofol group received 8.8 \pm 2 mg \cdot kg⁻¹ \cdot h⁻¹ of propofol. The VA group age was older than the propofol group (58 \pm 12 vs 51 \pm 15 yr, respectively, P = 0.002), but there was no difference between the groups in gender, ASA grade, intraoperative hemodynamic variables, and use of vasopressors. After 8 h, the VA group had a larger increase in arterial lactate from baseline compared with the propofol group (change from baseline: propofol, 0.48 \pm 0.72 mmol/L; VA, 1.2 \pm 1.2 mmol/L, P = 0.001).

CONCLUSIONS: During prolonged spine surgery >8 h, VA was associated with higher serum lactate, when compared with propofol infusion. Prospective studies are needed to elucidate the exact mechanisms and clinical implications of this finding.

G"GESH-SY5Y O12KYO,hBQhD
D%

Isoflurane inhibits cyclic adenosine monophosphate response element-binding protein phosphorylation and calmodulin translocation to the nucleus of SH-SY5Y cells.

Zhang J, Sutachan JJ, Montoya-Gacharna J, Xu CF, Xu F, Neubert TA, Recio-Pinto E, Blanck TJ.

Department of Anesthesiology, NYU School of Medicine, 550 1st Ave., New York, NY 10016, USA.

Anesth Analg. 2009 Oct;109(4):1127-34.

S CaM(SQh) δS"WRt08X(CREB(:~□
KMO,}h) nP-CREB(□-KMO,}h) □□
#9"9DO(9IURSSZlú4"lú9H"QBK;n
mzDIUR=G"GQBK;ZHLQg@"8&CaM
iXO(OO□□=P-CREB□□
#□ 8X3XO(O"SH-SY5Y OKCl #@-#□
<éAO"lúQgU:DOGdI#LPLCREB□□
O(9I#LPLCaM 8XQlll@f#CaM 9}O □
GdI#stq<éIK}st#KCl pI9m#O□
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O,OO(KCl P-CREB□@fú}MKhB0□
8I#9"l#□ EEH9"KIG"GII"l□
IRfR#O(L S"WR8IRBay K 8644 T8EG"G
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CaMlIG"G=#BAY K 8644 T8EReG"G8CaM □
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O,QIzHI@KBG"G#CaM lI9IP-CREB lhn□
h"7JG"G#IL S"WR8S"WI□
#BITQc 9KO#

BACKGROUND: Calmodulin (CaM) activation by Ca(2+), its translocation to the nucleus, and stimulation of phosphorylation of cyclic adenosine monophosphate response element-binding protein (CREB) (P-CREB) are necessary for new gene expression and have been linked to long-term potentiation, a process important in memory formation. Because isoflurane affects memory, we tested whether isoflurane interfered with the translocation of CaM to the neuronal cell nucleus and attenuated the formation P-CREB.
METHODS: SH-SY5Y cells, a human neuroblastoma cell line, were cultured. Cells were depolarized with KCl and the phosphorylation of CREB examined by Western blotting, enzyme-linked immunosorbant assay, and immunocytochemistry. The translocation of CaM from the cytosol to the nucleus was also examined after

depolarization. Cells were depolarized and lysed and fractionated by centrifugation to determine the amount of CaM translocated to the nucleus. CaM was localized by immunocytochemistry and quantitated by Western blotting and imaging. Before and during KCl depolarization, cells were exposed to isoflurane, isoflurane plus Bay K 8644, nitrendipine, and Ω -conotoxin GVIA, respectively.

RESULTS: P-CREB increased after KCl depolarization. The increase of P-CREB peaked at depolarization duration of 30 s. The increase in P-CREB formation was inhibited by nitrendipine, but not omega-conotoxin, and by isoflurane in a concentration-dependent fashion. Pretreatment with the L-type Ca(2+) channel agonist, Bay K 8644, attenuated the inhibition of P-CREB formation by isoflurane. CaM presence in the nucleus occurred after KCl depolarization. CaM translocation was inhibited by nitrendipine and attenuated by isoflurane. Bay K 8644 pretreatment decreased the isoflurane inhibition of CaM translocation to the nucleus.

CONCLUSIONS: Our data demonstrate that isoflurane inhibits CaM translocation and P-CREB formation. This most likely occurs through isoflurane inhibition of Ca(2+) entry through L-type Ca(2+) channels.

9[4mB;5O88PF"R} □

8[Gd(f9)QP5IRfIR—

An Analysis of Drug Delivery Dynamics via a Pediatric Central Venous Infusion System: Quantification of Delays in Achieving Intended Doses

Karsten Bartels, David R. Moss, and Robert A. Peterfreund

Department of Anesthesia and Critical Care, Massachusetts General Hospital, Jackson 439, 55 Fruit St., Boston, MA 02114, USA.

Anesth Analg 2009 109: 1156-1161.

8[4[CMKI8Q(D9mB;J%R-OfPF

"998:L, I[4mB;R} O8 U:SQPQ3O2PF=

8 QVL, I[4 8 O4- f 8gUJ%O2PF=8:tW998:KI

R-wJ%1Ht3MHLRg=9KIItu#8:wQ#28:

tW 0.5ml/h I8,s15SPF"H8PrS%IR} fR

sfzH 2ml/h 12ml/h IKGLfS:D 3kg J4IO2PF94S

PFR%GbS:DPF"tutRS:D#8:IR} =PF

"WPhIdMofQ38Rh9IQ@

O, 9;8#8:IPF"R} S9J%#PF"9)QgU:QP5L I9T

99)BL I#e19TGbL f#PF"R} 9;89KGLf9(t(50) = 23.5 +/-

2.1 min)RIKGLf9(t(50) = 15.7 +/- 2.9 min) =Tc4H8PrRR} □

%KO9)BL I9T(low flow t(50) = 12.7 +/- 0.6 min, high flow t(50)

= 5.2 +/- 0.8 min) =PF"W4IRgU98gUJ%#PF"tI9

T(t(50) = 3 +/- 0.5 min) I4RgU9(t(50) = 11.6 +/- 0.8 min)KO

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BACKGROUND: Pediatric patients frequently receive continuous infusions of drugs via central venous catheters in the intensive care unit and the operating room. This study characterized drug delivery profiles in a quantitative laboratory model of a standard pediatric central venous infusion system.

METHODS: We evaluated drug delivery via a standard pediatric 8-cm, 4-F double-lumen catheter. One syringe pump infused normal saline as the carrier fluid through a limb of a Y-piece connected to the catheter's 22-gauge distal lumen. Through the other limb of the Y-piece, a second syringe pump infused methylene blue, the model drug, at a constant rate of 0.5 mL/h. The volume delivered was collected every minute for quantitative analysis. We compared 2 mL/h and 12 mL/h total flow rates to mimic volume delivery to a 3-kg infant, and priming of the Y-piece with the model drug, to mimic resumption of a stopped drug infusion, versus no priming, to mimic a new infusion. Drug pump system start-up performance was measured to estimate this factor's contribution to infusion onset profiles.

RESULTS: When initiating a new infusion of the model drug, the time to steady-state delivery at the catheter's end varied significantly among the studied scenarios as measured by the time to reach half of the targeted dose ($t(50)$). Onset of delivery with a low total flow was much slower ($t(50) = 23.5 \pm 2.1$ min) than with the high flow rate ($t(50) = 15.7 \pm 2.9$ min). Priming the drug limb of the connecting Y-piece with methylene blue substantially shortened the time to steady state (low flow $t(50) = 12.7 \pm 0.6$ min, high flow $t(50) = 5.2 \pm 0.8$ min). Time to cessation of drug delivery to the end of the catheter after stopping the drug pump was substantially shorter using the high carrier flow rate ($t(50) = 3 \pm 0.5$ min) compared with the low carrier flow rate ($t(50) = 11.6 \pm 0.8$ min). Drug pump system start-up performance contributed to onset delay.

CONCLUSIONS: Current infusion techniques in the pediatric care setting can result in significant, unrecognized, and potentially hazardous delays in achieving delivery of intended drug doses to the patient. Total flow rate, priming of the infusion system, the dead volume of the fluid path, and the start-up performance of the infusion pump system contribute to delays in achieving targeted rates of drug delivery.

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Epidemiology of Anesthesia-Related Complications in Labor and Delivery, New York State, 2002-2005

Khadeen Cheesman, BS, Joanne E. Brady, SM, Pamela Flood, MD, and Guohua Li, MD, DrPH

From the Department of Anesthesiology, Columbia University, New York City, New York.

Address correspondence and reprint requests to Guohua Li, MD, DrPH, Department of Anesthesiology, Columbia University, 622 West 168th St., PH5-505, New York, NY 10032. Address e-mail to GL2240@columbia.edu.

Anesth Analg 2009 109: 1174-1181

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8b(OR 1.33, 95% CI 1.21-1.46), Charlson-Deyo u@□ 1 (OR 1.47, 95% CI
1.28-1.69), 1B□ (OR 1.37, 95% CI 1.24-1.52), #KZ5□ (OR 1.10, 95% CI
1.03-1.18)=00SA@G#B189 ± 3.69 [A@# L,□
□ vs 2.92 ± 2.38 9m9(00P < 0.0001# G□

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BACKGROUND: Epidemiologic data on anesthesia-related complications occurring during labor and delivery are essential for measuring and evaluating the safety and quality of obstetric anesthesia care but are lacking. We aimed to fill this research gap by exploring the epidemiologic patterns and risk factors of anesthesia-related complications in a large sample of women giving birth in New York hospitals.

METHODS: Using the Healthcare Cost and Utilization Project State Inpatient Databases files, we identified all discharge records for labor and delivery from New York hospitals between 2002 and 2005. We then identified women who experienced any recorded anesthesia-related complication during labor and delivery as determined by International Classification of Diseases, Ninth Revision, Clinical Modification codes. The incidence of anesthesia-related complications was calculated by demographic and clinical characteristics. Multivariate logistic regression was performed to assess risk factors of anesthesia-related complications.

RESULTS: Of the 957,471 deliveries studied, 4438 (0.46%) had at least one anesthesia-related complication. The majority (55%) of anesthesia-related events occurring during labor and delivery were spinal complications, followed by systemic complications (43%) and overdose or adverse effects (2%). Multivariate logistic regression revealed five risk factors of anesthesia-related complications: cesarean delivery (odds ratio [OR] 2.51, 95% confidence interval [CI] 2.36-2.68), rural area (OR 1.33, 95% CI 1.21-1.46), Charlson-Deyo Comorbidity Index 1 (OR 1.47, 95% CI 1.28-1.69), Caucasian race (OR 1.37, 95% CI 1.24-1.52), and scheduled admission (OR 1.10, 95% CI 1.03-1.18). Anesthesia-related complications were associated with about a one-day increase in the average length of stay (3.89 ± 3.69 [mean \pm sd] days vs 2.92 ± 2.38 days for deliveries without anesthesia-related complications, $P < 0.0001$) and a 22-fold increased risk of maternal mortality (OR 22.26, 95% CI 11.20-44.24).

CONCLUSION: The incidence of anesthesia-related complications during labor and delivery seems to be low but remains a cause of concern, particularly in women

undergoing cesarean delivery, living in rural areas, or having preexisting medical conditions.

DISCUSSION

A Prediction Model for Out-of-Hospital Cardiopulmonary Resuscitation

Iris R. Pircher, MD*, Karl-Heinz Stadlbauer, MD*, Anette C. Severing, MD*, Viktoria D. Mayr, MD*, Hannes G. Lienhart, MD*, Beate Jahn, PhD, Karl H. Lindner, MD*, and Volker Wenzel, MD, MSc*

From the Departments of *Anesthesiology and Critical Care Medicine, and Medical Statistics, Informatics and Health Economics, Innsbruck Medical University, Innsbruck, Austria.

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0898-2643/09/109-1196-05\$5.00

786 (67.4%) died at the scene and 380 (32.6%) were brought to the hospital.

22.7% died in the hospital. One hundred fifteen (9.8%) were discharged from the hospital.

and 92 of the 115 patients (80%) could be followed-up.

Good cerebral performance was regained by 54% of discharged patients (50 of 92 patients).

In 46% of patients (42/92), unconsciousness or severe disability remained.

Ventricular fibrillation was more likely to have occurred in patients with good neurological recovery (42/50 = 84.0%), whereas asystole was more likely in patients with

poor neurological recovery (42/50 = 84.0%).

Conclusions: A prediction model for out-of-hospital cardiopulmonary resuscitation (CPR) attempts as a decision tool to omit futile CPR attempts and to save resources.

Keywords: cardiac arrest, CPR, prediction model, resuscitation, survival.

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0898-2643/09/109-1196-05\$5.00

DOI: 10.1097/ALN.0b013e3181818181

BACKGROUND: We created a prediction model to be used in cardiopulmonary resuscitation (CPR) attempts as a decision tool to omit futile CPR attempts and to save resources.

METHODS: In this post hoc analysis, we assessed predictive parameters for neurological recovery after successful CPR. The original study was designed as a blinded, randomized, prospective, controlled, multicenter clinical trial.

RESULTS: We identified 1166 prehospital cardiac arrest patients being treated with advanced cardiac life support. Seven hundred eighty-six of 1166 patients (67.4%) died at the scene and 380 of 1166 (32.6%) were brought to the hospital. Two hundred sixty-five of 1166 patients (22.7%) died in the hospital. One hundred fifteen of 1166 (9.8%) were discharged from the hospital and 92 of the 115 patients (80%) could be followed-up. Good cerebral performance was regained by 54% of discharged patients (50 of 92 patients). In 46% of patients (42/92), unconsciousness or severe disability remained. Ventricular fibrillation was more likely to have occurred in patients with good neurological recovery (42/50 = 84.0%), whereas asystole was more likely in patients with

poor neurological recovery (9/42 = 21.4%). A score was developed to predict the probability of death using logistic regression analysis. Predicting death in the hospital revealed a sensitivity of 99.8% (953/955), but only a specificity of 2.9% (3/104; threshold 0.5). Predicting survival until discharge from the hospital revealed a sensitivity of 99% (103/104), but only a specificity of 8% (72/955; threshold 0.99). A receiver operating characteristic curve yielded an area under the curve of 0.795 (0.751-0.839) at a confidence interval of 95%.

CONCLUSION: For out-of-hospital patients with cardiac arrest, parameters documented in the field did not allow accurate prediction of hospital survival.

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U:

A Randomized Trial Comparing Colloid Preload to Coload During Spinal Anesthesia for Elective Cesarean Delivery

Sahar M. Siddik-Sayyid, MD, FRCA, Viviane G. Nasr, MD, Samar K. Taha, MD, Reine A. Zbeide, MD, Jules-Marie A. Shehade, MD, Ashir A. Al Alami, MD, Farah H. Mokadem, MD, Faraj W. Abdallah, MD, Anis S. Baraka, MD, FRCA, and Marie T. Aouad, MD

From the Department of Anesthesiology, American University of Beirut Medical Center, Beirut, Lebanon.

Anesth Analg 2009 109: 1219-1224

SB;H8m8<DQ=ciKBu94R□

k8Utu9vP#DQITc5RK8UfP#□

T8#HK=TC4<h>SmffIEG#zH□

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n=88#=#8O8@JIC#8□

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8@KMIwK9O2f%8H#BAOBH#□

O, #I358<H#8:IRf%8H14#□

O, #I39OmT9#K8b9Tc#O□

68%u9<□

O□ 75%9CpT8b9□

95%#8bTH6%

20%; P = 0.28

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BACKGROUND: Hypotension after spinal anesthesia for cesarean delivery is common. Previous studies have demonstrated that a crystalloid fluid "coload" (rapid administration of a fluid bolus starting at the time of intrathecal injection) is superior to the conventional crystalloid preload (fluid

administered before the intrathecal injection) for preventing hypotension. Colloid preload provides a sustained increase in central blood volume. We hypothesized that, in contrast to crystalloid, a colloid preload may be more effective than colloid coload for reducing the incidence of spinal anesthesia-induced hypotension.

METHODS: In this double-blind study, 178 patients were randomly assigned to receive a preload of 500 mL of hydroxyethyl starch over a period of 15–20 min before initiation of spinal anesthesia (n = 90) or an identical fluid bolus of hydroxyethyl starch starting at the time of identification of cerebrospinal fluid (n = 88). Vasopressors (ephedrine or phenylephrine) were administered if systolic arterial blood pressure decreased less than 80% of the baseline pressure and <100 mm Hg, or with smaller decreases in blood pressure if accompanied by nausea, vomiting, or dizziness. The primary outcome was the incidence of hypotension (defined as the administration of at least one dose of vasopressor).

RESULTS: There was no significant difference between the groups in the incidence of hypotension (68% in preload group and 75% in coload group, 95% confidence interval of difference –6%–20%; P = 0.28), doses of ephedrine and phenylephrine, and number of vasopressor unit doses. The incidence of severe hypotension (systolic blood pressure <80 mm Hg) was 16% in the preload group and 22% in the coload group (P = 0.30). There were no differences in the incidence of nausea and/or vomiting, or neonatal outcome between the groups.

CONCLUSION: There was no difference in the incidence of hypotension in women who received colloid administration before the initiation of spinal anesthesia compared with at the time of initiation of anesthesia. Both modalities are inefficient as single interventions to prevent hypotension.

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iRfRKH

Sevoflurane Preconditioning Induces Rapid Ischemic Tolerance Against Spinal Cord Ischemia/Reperfusion Through Activation of Extracellular Signal-Regulated Kinase in Rabbits

Qian Ding, Qiang Wang, Jiao Deng, Qiuhan Gu, Sheng Hu, Yang Li, Bin Xiao Su, Yi Zeng, and Lize Xiong

Anesth Analg 2009 109: 1263-1272.

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BACKGROUND: The protective effect of sevoflurane preconditioning against spinal cord ischemia/reperfusion (I/R) is unclear. We designed this study to investigate whether sevoflurane preconditioning could induce rapid ischemic tolerance to the spinal cord in a rabbit model of transient spinal cord ischemia and how the role of extracellular signal-regulated kinase (ERK) is involved.

METHODS: To test whether preconditioning with sevoflurane induces rapid ischemic tolerance, New Zealand White male rabbits were randomly assigned to three groups. Animals in the Sev group received preconditioning with 3.7% sevoflurane (1.0 minimum alveolar anesthetic concentration) in 96% oxygen for 30 min, whereas animals in the O₂ group serving as controls inhaled only 96% oxygen for 30 min. The Sham group received the same anesthesia and surgical preparation but no preconditioning or spinal cord I/R. To evaluate the role of ERK activation in sevoflurane preconditioning, rabbits were randomly assigned to four groups. U0126, an ERK inhibitor, was administered IV 20 min before the beginning of preconditioning in the U0126 + O₂ and U0126 + Sev groups. Dimethylsulfoxide was administered IV at the same time in the vehicle + O₂ and vehicle + Sev groups. At 1 h after preconditioning, the animals were subjected to spinal cord I/R induced by infrarenal aorta occlusion. All animals were assessed at 48 h after reperfusion with modified Tarlov criteria, and the spinal cord segments (L5) were harvested for histopathological examination, TUNEL staining, and Western blot of phosphor-ERK1/2.

RESULTS: The animals in the Sev group had higher neurological scores and more normal motor neurons than those in the O₂ group (*P* < 0.01 for each comparison). Compared with vehicle + Sev group, the U0126 + Sev group had worse neurological outcomes, fewer viable neurons, more apoptotic neurons, and significantly decreased ERK1/2 phosphorylation (*P* = 0.01 for each comparison). There were no significant differences in the outcomes among vehicle + O₂, U0126 + O₂, and U0126 + Sev groups.

CONCLUSIONS: This study demonstrates that sevoflurane preconditioning induces rapid tolerance to spinal cord I/R in rabbits, and the tolerance is possibly mediated through the activation of ERK. These data suggest that sevoflurane preconditioning might provide a new practical method for protecting perioperative spinal cord I/R.

O - M d i R Q m i T M i h T M @ B S □

Complete Freund's adjuvant-induced intervertebral discitis as an animal model for discogenic low back pain.

Lee M, Kim BJ, Lim EJ, Back SK, Lee JH, Yu SW, Hong SH, Kim JH, Lee SH, Jung WW, Sul D, Na HS.

Department of Biomedical Science, Korea University Graduate School, Seoul, Korea. *Anesth Analg*. 2009 Oct;109(4):1287-96.

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Q#lé8S%IO,QL#D#IJ%4O-Md
IR(CFA)t5UUi*S:D3;hTMl8"St@
:{UU L5-6 nTM8U 10 CFA K4éHGd,OO GdI3"
Gd#hTMIII9#hOO GdIQhTMé
#K4#OPCR :9K}ASKLsSHIO (CGRP)
hH E(PGE)IQ#Ü#A(iNOS) HIBI#hT
M8UCGRP }RHXO(8U3SITwQS
O, 8 7 L CFA OmQ)OFIK#OO GdIMhTM
II@LJO,NO(IE#hTM8U CFA CGRP-)
IM8gHáInTM}K'9#L 4 LKO(PDRG#m
CGRPmRNA d9#L PGE iNOS mRNA 1d9HK#CGRP
DIUU8Ud9K#DUU8U1d9
O,QIhTM8U CFA tQJhTMIIIDhS
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BACKGROUND: Although numerous animal models for low back pain associated with intervertebral disk (IVD) degeneration have been proposed, insufficient data have been provided to make any conclusions regarding pain. Our aim in this study was to determine the reliability of complete Freund's adjuvant (CFA) injection into the rat spine as an animal model representing human discogenic pain.

METHODS: We studied IVD degenerative changes with pain development after a 10-microL CFA injection into the L5-6 IVD of adult rats using behavioral, histologic, and biochemical studies. Serial histologic changes were analyzed to detect degenerative changes. Expression of calcitonin gene-related peptide (CGRP), prostaglandin E (PGE), and inducible nitric oxide synthase (iNOS) were determined using immunohistochemistry or real-time polymerase chain reaction as support data for pain development. In addition, CGRP immunoreactivity (ir) at the IVD was considered indirect evidence of neural ingrowth into the IVD.

RESULTS: There was a significant increase of the hindpaw withdrawal response in the CFA group until 7 wk postoperatively ($P < 0.05$). Histologic analyses revealed progressive degenerative changes of the disks without any damage in adjacent structures, including nerve roots. In the CGRP-ir staining study, the bilateral dorsal horns and IVD had positive ir after intradiscal CFA injection. CGRP mRNA expression was increased in the dorsal root ganglion (DRG) at 2 and 4 wk, whereas PGE and iNOS mRNAs were markedly increased at 2 wk. The increment of CGRP expression was higher in allodynic rats compared with nonallodynic rats.

CONCLUSION: Intradiscal CFA injection led to chronic disk degeneration with allodynia, which was suggested by pain behavior and expression of pain-related mediators. The increment of CGRP, PGE, and iNOS also suggest pain-related signal processing between the IVD and the neural pathway in this animal model. This animal model may be useful for future research related to the pathophysiology and development of novel treatment for spine-related pain.

UUK8UtiSUOYI8tK□

The Synergistic Interaction Between Morphine and Maprotiline After Intrathecal Injection in Rats

Vera L. A. Pettersen, MD*, Gisele Zapata-Sudo, MD, PhD , Juliana M. Raimundo, PhD , Margarete M. Trachez, MD, PhD , and Roberto T. Sudo, MD, PhD

Address correspondence and reprint requests to Roberto Takashi Sudo, MD, PhD, Centro de Ciências da Saúde, Bloco J, Sala 14, Universidade Federal do Rio de Janeiro, Avenida Carlos Chagas 373, Cidade Universitária, Rio de Janeiro 21941-590, Brazil. Address e-mail to rtsudo@farmaco.ufrj.br .

Anesth Analg 2009 109: 1312-1317.

S□ :|PF"□#E8OBHI/ □ 5-O|K7I4DSs8U

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UOY|PFgd4E

O,□ 9#k8Uti□ 2 µgsM□ 125 µgO-H□ 144 µgUOY□

1.25 µg 9HB□ 51.6% ± 8.9%, 10.3% ± 3.2%, 33.8% ± 5.2%□48.5% ± 9.2% □

MPE-E|SsM|49=Hm9Kú>□ 91.3% ± 4.6% MPESU□

OY|4ú>□ 86.9% ± 9.2% MPESO-H|48pú>(40.6% ±

4.6% MPE)=SUOYO□;49|I=Hm99Tú7 120 □

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BACKGROUND: Antidepressant drugs act as potent inhibitors of norepinephrine and/or serotonin reuptake and are widely used with opioids for the treatment of chronic pain. The mechanism of this increased analgesic action is unclear. We compared the anti nociceptive effects of the intrathecal administration of morphine with that of a nonselective (amitriptyline) or selective (maprotiline or citalopram) antidepressant drug

using the thermal withdrawal test in rats. We also investigated the possible mechanisms involved in the interactions of these drugs.

METHODS: Male Wistar rats were anesthetized with sevoflurane and administered morphine and antidepressant drugs, or saline, through intrathecal injection. The antinociceptive effect was evaluated using the thermal withdrawal test before and after drug administration. The time for the withdrawal reaction was expressed as percentage of maximum possible effect (MPE). Animals were also pretreated with yohimbine (a nonselective α_2 -adrenergic antagonist) and naloxone (a nonselective opioid antagonist) for mechanism of action studies. Pharmacologic interaction was evaluated using isobolographic analysis of simultaneous administration of fixed proportions of maprotiline and morphine.

RESULTS: Single intrathecal administration of morphine (2 μ g), amitriptyline (125 μ g), citalopram (144 μ g), and maprotiline (1.25 μ g) produced $51.6\% \pm 8.9\%$, $10.3\% \pm 3.2\%$, $33.8\% \pm 5.2\%$, and $48.5\% \pm 9.2\%$ MPE, respectively. The antinociceptive effect of morphine was increased when combined with amitriptyline ($91.3\% \pm 4.6\%$ MPE) and maprotiline ($86.9\% \pm 9.2\%$ MPE) but not with citalopram ($40.6\% \pm 4.6\%$ MPE). Coadministration of maprotiline increased the antinociceptive duration of morphine by 4-fold (from 120 to 480 min), which was reversed by pretreatment with the α_2 -adrenoceptor inhibitor, yohimbine, and the μ -type opioid receptor antagonist, naloxone. Isobolographic analysis demonstrated a synergistic interaction between morphine and maprotiline.

CONCLUSIONS: Selective norepinephrine reuptake inhibitors can significantly increase the intensity and duration of morphine antinociceptive activity via both α_2 -adrenergic and opioid receptors. This interaction was not observed with the selective serotonin inhibitor, citalopram.

58JRB;8bMIS

The Analgesic Effect of Paracetamol When Added to Lidocaine for Intravenous Regional Anesthesia

Huseyin Sen, MD*, Yalcin Kulahci, MD, Enis Bicerer, MD*, Sezai Ozkan, MD*, Guner Dagl, MD*, and Alparslan Turan, MD

From the Departments of *Anesthesiology and Reanimation, and Plastic and Reconstructive Surgery, Gülhane Military Medical Academy Haydarpa a Training Hospital Üsküdar, Istanbul, Turkey; Department of Anesthesiology and Perioperative Medicine, University of Louisville, Louisville, Kentucky; and Department of Outcomes Research, The Cleveland Clinic, Cleveland, Ohio.

Anesth Analg 2009 109: 1327-1330

SQ ZHQV

58JR#4JB;8b

tM9Q-R8fM19TKIS

60 |e8IB@BH

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AO2f 3 mg/kg 4BMR

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MO 2 O2f

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300mg=9QV 8ZTQR8fM

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 H 78 ± 12 58 ± 14 78 ± 11 µg(44YLB@f9H
 13 3B 3I9 3O 2 3@KRm(P < 0.05)#O 2 84YI9TT6
 RmS9H 15 ± 6, 25 ± 5, 15 ± 4 S%#(P < 0.05)#O 2 8(IQfRm(P <
 0.05)#8 VAS QVIK#4SPF"19TzOmO 2 48g(Y
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 (IQ#8SPF"4If
 TG"p"pQc 9KO

BACKGROUND: In this study, we evaluated the effect of paracetamol on sensory and motor block onset time, tourniquet pain, and postoperative analgesia, when added to lidocaine in IV regional anesthesia (IVRA).

METHODS: Sixty patients undergoing hand surgery were randomly and blindly divided into three groups. All groups received IVRA lidocaine (3 mg/kg) diluted with saline to a total volume of 40 mL. Group 1 received IVRA lidocaine plus IV saline, Group 2 received IVRA lidocaine and paracetamol (300 mg) admixture plus IV saline, and Group 3 received IVRA lidocaine plus IV paracetamol (300 mg). Sensory and motor block onset time, tourniquet pain, and analgesic use were assessed during operation. After tourniquet deflation, visual analog scale (VAS) scores at 1, 2, 4, 6, 12, and 24 h, the time to first analgesic requirement, total analgesic consumption in first 24 h, and side effects were noted.

RESULTS: Onset of motor block was shorter and recovery of motor and sensory block was significantly longer in Group 2 ($P < 0.05$). Intraoperative VAS scores at intraoperative 20, 30, and 40 min were significantly lower in Group 2 ($P < 0.05$). Intraoperative fentanyl consumption (78 ± 12 , 58 ± 14 , 78 ± 11 µg, respectively) and the number of patients who required fentanyl for tourniquet pain (13 patients, 3 patients, 9 patients, respectively) were significantly less in Group 2 ($P < 0.05$). Time to postoperative fentanyl administration was also prolonged (15 ± 6 , 25 ± 5 , 15 ± 4 min, respectively) in Group 2 ($P < 0.05$). The quality of surgical anesthesia was better in Group 2 ($P < 0.05$). Postoperative VAS scores and time of initial analgesic requirement were similar among groups; however, the total amount of diclofenac use was less in Group 2 ($P < 0.05$).

CONCLUSION: The addition of paracetamol during IVRA with lidocaine decreased tourniquet pain, increased anesthesia quality, and decreased postoperative analgesic consumption.

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The Effects of Cardiopulmonary Bypass on the Number of Cerebral Microemboli and the Incidence of Cognitive Dysfunction After Coronary Artery Bypass Graft Surgery

Ying-Hua Liu, MD*, Dong-Xin Wang, MD, PhD*, Li-Huan Li, MD, PhD, Xin-Min Wu, MD*, Guo-Jin Shan, MD*, Yu Su, MD*, Jun Li, MD*, Qin-Jun Yu, MD, Chun-Xia Shi, MD, Yi-Ning Huang, MD, and Wei Sun, MD

From the *Department of Anesthesiology, Peking University First Hospital, Beijing, China; Department of Anesthesiology, Cardiovascular Institute and Fuwai Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College, Beijing, China; and Department of Neurology, Peking University First Hospital, Beijing, China. *Anesth Analg* 2009; 109:1013-1022

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BACKGROUND: Postoperative cognitive dysfunction (POCD) can be a debilitating complication after coronary artery bypass graft (CABG) surgery. Cerebral microemboli during cardiopulmonary bypass (CPB) are believed to be an important etiologic factor of POCD. In this study, we examined whether avoidance of CPB with "off-pump" surgery reduces the number of cerebral microemboli and the incidence of POCD after CABG surgery in Chinese population.

METHODS: Two hundred twenty-seven patients were enrolled in this prospective cohort study. Fifty-nine patients underwent CABG surgery with CPB and 168 underwent off-pump surgery. Cerebral microemboli were measured continuously with bilateral transcranial Doppler ultrasonography of the middle cerebral arteries. A neuropsychological test battery that included seven tests with nine subscales was administered at baseline, as well as at 1 wk and 3 mo after surgery. POCD was defined using the international study of POCD1 definition.

RESULTS: The median total number of cerebral microemboli for the case was 430 (range: 155–2088) in patients undergoing surgery with CPB and 2 (0–66) in the off-pump patients (*P* < 0.001). There were no differences in the incidence of POCD between the patients having surgery with or without CPB either at 1 wk (55.2% or 32 of 58 patients [95% confidence interval: 41.5%–68.3%] vs 47.0% or 78 of 166 patients [39.2%–54.9%], *P* = 0.283) or 3 mo (6.4% or 3 of 47 patients [1.3%–17.5%] vs 13.1% or 16 of 122 of

patients [7.7%–20.4%], $P = 0.214$) after surgery. Increasing age and shorter duration of postoperative hospital stay were independently associated with cognitive dysfunction at 1 wk after surgery. Increasing age and a history of diabetes mellitus were independently associated with cognitive dysfunction 3 mo after surgery. CPB or cerebral microemboli were not significantly related to the occurrence of POCD.

CONCLUSIONS: In Chinese population, avoidance of CPB during CABG surgery significantly decreased the number of cerebral microemboli, but it did not decrease the incidence of POCD at either 1 wk or 3 mo after CABG. Neither CPB nor cerebral microemboli was independently associated with the risk of POCD.

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Malignant Hyperthermia, Coexisting Disorders, and Enzymopathies: Risks and Management Options

Joan Benca, MD, and Kirk Hogan, MD, JD

From the Department of Anesthesiology, University of Wisconsin School of Medicine and Public Health, Madison, Wisconsin.

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Clinical episodes and abnormal laboratory tests compatible with a diagnosis of malignant hyperthermia have been observed in patients with a diversity of syndromes, enzymopathies, and coexisting disorders thereby raising the likelihood of causal associations and heightened perioperative risk in others carrying a shared diagnosis. In the present review, we survey available published series, case reports, and the results of contracture testing in patients identified by others to be potentially predisposed to malignant hyperthermia. For most conditions, evidence for a causal relationship with malignant hyperthermia susceptibility is weak. The review concludes with suggestions for clinical management when evidence for or against an association is uncertain.

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Neuromuscular Block Differentially Affects Immobility and Cortical Activation at Near–Minimum Alveolar Concentration Anesthesia

Anthony G. Doufas, MD, PhD^{*||}, Ryu Komatsu, MD , Mukadder Orhan-Sungur, MD , Papiya Sengupta, MD , Anupama Wadhwa, MD , Edward Mascha, PhD , Steven L. Shafer, MD , and Daniel I. Sessler, MD^{||}

From the *Department of Anesthesia, Stanford University School of Medicine, Stanford, California; Outcomes Research Institute, and Department of Anesthesiology and

Perioperative Medicine, University of Louisville, Louisville, Kentucky; Departments of Quantitative Health Sciences and Outcomes Research, The Cleveland Clinic, Cleveland, Ohio; Department of Anesthesiology, Columbia University, New York, New York; ||Department of Outcomes Research, The Cleveland Clinic, Cleveland, Ohio; and ¶Outcomes Research Consortium, Cleveland, Ohio.
 Anesth Analg 2009; 109:1097-1104

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BACKGROUND: Anesthesia-induced immobility and cortical suppression are governed by anatomically separate, but interacting, areas of the central nervous system. Consequently, larger volatile anesthetic concentrations are required to suppress cortical activation than to abolish movement in response to noxious stimulation. We examined the effect of decreased afferent input, as produced by neuromuscular block (NMB), on immobility and cortical activation, as measured by Bispectral index (BIS) of the electrocardiogram, in the presence of noxious stimulation during approximately minimum alveolar concentrations (MACs) of desflurane anesthesia.

METHODS: The effect of NMB on the median effective end-tidal concentration of desflurane (EtDes₅₀, or MAC_{tetanus}) for immobility was estimated using the up-and-down method and isolated forearm technique in 24 healthy volunteers. Each volunteer sequentially received saline, mivacurium, and succinylcholine in a randomized order, while EtDes concentration during each of the treatments was determined based on the

movement response of the previous volunteer on the same treatment. Nonlinear mixed-effects modeling was used to evaluate the effect of NMB on BIS versus EtDes concentration relationship at baseline and after noxious stimulation, while the frontal electromyogram (EMG_{BIS}) effect on BIS was also modeled as a covariate. Cardiovascular responses to noxious stimulation were compared across treatments.

RESULTS: Succinylcholine and mivacurium significantly reduced MAC_{tetanus} (95% confidence interval) from 5.00% (4.85%–5.13%), during saline, to 4.05% (3.81%–4.29%) and 3.84% (3.60%–4.08%), respectively. Noxious stimulation significantly, although minimally, increased BIS response during all treatments. Succinylcholine increased BIS independently of an effect on EMG_{BIS}. Succinylcholine administration increased cardiovascular activity. Interestingly, although cardiovascular reaction to the noxious event was ablated by mivacurium, cortical response, as determined by BIS, was retained.

CONCLUSIONS: Both succinylcholine and mivacurium enhanced immobility during near-MAC anesthesia. All treatments were associated with a small, although significant, BIS increase in response to noxious stimulation, whereas succinylcholine increased BIS independently of noxious stimulation or EMG_{BIS}. Mivacurium suppressed autonomic response to a noxious event.

Stem Cell-Like Human Endothelial Progenitors Show Enhanced Colony-Forming Capacity After Brief Sevoflurane Exposure: Preconditioning of Angiogenic Cells by Volatile Anesthetics

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Stem Cell-Like Human Endothelial Progenitors Show Enhanced Colony-Forming Capacity After Brief Sevoflurane Exposure: Preconditioning of Angiogenic Cells by Volatile Anesthetics

Eliana Lucchinetti, PhD*, Steffen M. Zeisberger, PhD, Isabella Baruscotti, MS, Johannes Wacker, MD, Jianhua Feng, MD, PhD*, Kathrin Zaugg, MD, PhD^{||}, Raghvendra Dubey, PhD, Andreas H. Zisch, PhD, and Michael Zaugg, MD^{†#}

From the *Department of Anesthesiology and Pain Medicine, University of Alberta, Edmonton, Alberta, Canada; Department of Obstetrics and Gynecology, University Hospital Zurich; Department of Obstetrics and Gynecology, Clinics for Reproductive Endocrinology, University Hospital Zurich; Institute of Anesthesiology, University Hospital Zurich; ^{||}Department of Radiation Oncology, University Hospital Zurich, Zurich, Switzerland; [†]Department of Anesthesiology and Pain Medicine, University of Alberta; and [#]Perioperative Translational Medicine, Mazankowski Alberta Heart Institute, Edmonton, Alberta, Canada.

Anesth Analg 2009; 109:1117-1126

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<1 vol% sevoflurane (end-tidal concentration) would mobilize endothelial progenitor cells from the bone marrow niche into the circulation using flow cytometry of peripheral blood samples. VEGF and G-CSF plasma levels were also measured.

RESULTS: *In vitro* sevoflurane exposure of mononuclear cells enhanced colony-forming capacity and increased VEGF mRNA levels in CD133+/CD34+ cord blood cells ($P = 0.017$). Sevoflurane inhalation in healthy volunteers did not alter the number of CD133+/CD34+ or KDR+/CD34+ endothelial progenitors in the circulation, but increased the number of colony-forming units ($P = 0.034$), whereas VEGF and G-CSF plasma levels remained unchanged.

CONCLUSIONS: Sevoflurane preconditioning promotes growth and proliferation of stem cell-like human endothelial progenitors. Hence, it may be used to promote perioperative vascular healing and to support cell replacement therapies.

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Impact of Multiaccess Infusion Devices on *In Vitro* Drug Delivery During Multi-Infusion Therapy

Bertrand Décaudin, PhD^{*}, Sophie Dewulf, PharmD, Damien Lannoy, MSc^{*}, Nicolas Simon, MSc^{*}, Alexandre Secq, MSc, Christine Barthélémy, PhD^{*}, Bertrand Debaene, PhD, and Pascal Odou, PhD^{*}

From the ^{*}Department of Biopharmacy, Galenic and Hospital Pharmacy EA 4034—IFR114, Lille 2 University, Lille, France; Department of Pharmacy, Lille University Hospital, Lille, France; Department of Pharmacy, Dunkerque General Hospital,

Dunkerque, France; and Anesthesia and Intensive Care Department, University Hospital, INSERM, Poitiers, France.
 Anesth Analg 2009; 109:1147-1155

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BACKGROUND: Multiaccess infusion sets allow multiple simultaneous infusions but may induce interference in drug delivery resulting from large variations in the delivery rate of potent drugs. In this study, we sought to understand the influence of multiaccess infusion device properties (dead space volume and antireflux valve [ARV]) on drug delivery during multi-infusion therapy.

METHODS: Infusion sets differing in length, dead space volume, and presence of an ARV were assessed. Three drugs were infused simultaneously through different access points, and their concentrations were obtained using UV spectrophotometric analysis of the effluent. Different infusion configurations were compared by assessing (1) the amount of drug delivered to the patient per unit of time, (2) the mean amount of drug delivered to the patient per unit of time during the steady-state infusion (mass flow rate plateau), and (3) flow change efficiency calculated from the ratio of the area under the experimental instant mass flow rate curve to the area corresponding to theoretical instant mass flow rate curve.

RESULTS: Infusion sets with lower dead space volumes offered significantly higher flow change efficiency (53.0% ± 15.4% with a dead space volume equal to 0.046 mL 5 min after the start of infusion) than infusion sets with higher dead space volume (5.6% ± 8.2% with a dead space volume equal to 6.16 mL), whatever the flow rate changes. Even in case of large dead space volumes, the presence of an ARV significantly increased the mass flow rate plateau (from 92.4% to 99.3% of the theoretical plateau without and with the presence of an ARV, respectively).

CONCLUSIONS: Multi-infusion therapy induces perturbation in drug delivery. These perturbations (lag time, backflow, and bolus) could be reduced by using infusion sets including very low dead space volume and an ARV.

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Core Myopathies and Risk of Malignant Hyperthermia

Werner Klingler, MD^{*}, Henrik Rueffert, MD, Frank Lehmann-Horn, MD, Thierry Girard, MD, and Philip M. Hopkins, MD^{||}

From the Departments of ^{*}Anaesthesiology, and Applied Physiology, University of Ulm, Ulm, Germany; Department of Anaesthesiology and Intensive Care Medicine, University of Leipzig, Leipzig, Germany; Department of Anaesthesia, University Hospital of Basel, Basel, Switzerland; and ^{||}Academic Unit of Anaesthesia, St. James's University Hospital, Leeds, UK.

Anesth Analg 2009; 109:1167-1173

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In this article, we analyze myopathies with cores, for which an association to malignant hyperthermia (MH) has been suggested. We discuss the clinical features, the underlying genetic defects, subsequent effects on cellular calcium metabolism, and *in vitro* muscle responses to MH triggers. We describe in detail central core disease, multiminicore disease, and nemaline rod myopathy. We categorize the diseases according to the affected proteins and discuss the risk for MH, which is high or theoretically possible when the calcium-conducting proteins are affected.

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The Relative Exposure of the Operating Room Staff to Sevoflurane During Intracerebral Surgery

Béla Tankó, MD, Csilla Molnár, MD, PhD, Tímea B di, MD, Csaba Pet, MSc, László Novák, MD, PhD, and Béla Fülesdi, MD, PhD, DSci

From the Department of Anesthesiology and Intensive Care, Health and Medical Science Centre, University of Debrecen, Debrecen, Hungary.

Anesth Analg 2009; 109:1187-1192

背景: 这个研究中, 我们最初的目的是探讨开颅肿瘤切除术中, 对于从手术部位散发的挥发性麻醉药七氟醚的接触量, 神经外科医生是否大于麻醉医生。

方法: 首先, 我们测定了 35 例颅内肿瘤切除术从硬脑膜打开到关闭, 从手术部位散发的七氟醚量。挥发性麻醉药吸收剂被放置在三个测定部位: 1)外科医生呼吸区域、2)麻醉医生呼吸区域、以及 3)离手术部位最远的手术室角落。在第二个采样系列中(包括 16 名病人), 第一个采样系列中被放置在手术室最远角落的吸收剂这次

被放置在病人的嘴边 (5cm 以内)。被吸收剂吸附的七氟醚用色谱法由一名独立的药剂师进行定量测定。

结果: 外科医生呼吸区域吸收剂吸附的七氟醚量(0.24 ± 0.04 ppm)明显低于麻醉医生呼吸区域(1.40 ± 0.37 ppm), 且与手术室最远角落吸收剂吸附的七氟醚量(0.25 ± 0.07 ppm)相当。吸收剂吸附的七氟醚量与手术切口大小没有相关性, 即使调整了手术时间这一变量, 两者也没有相关性。在第二个采样系列中, 病人嘴边的吸收剂吸附了最高量的七氟醚(1.54 ± 0.55 ppm), 其次是麻醉医生呼吸区域(1.14 ± 0.43 ppm)和外科医生呼吸区域(0.15 ± 0.05 ppm)。

结论: 外科医生呼吸区域最接近的手术部位并不是增加七氟醚暴露的来源。我们观察到麻醉医生在手术室环境中暴露于七氟醚更多, 这有必要深入研究。

(张莹译 马皓琳 李士通校)

BACKGROUND: Our primary aim in this study was to investigate whether escape of the volatile anesthetic sevoflurane from the surgical site during craniotomy for tumor resection increases the exposure of the neurosurgeon to the anesthetic when compared with the anesthesiologist.

METHODS: Initially, the release of sevoflurane from the surgical site was measured during 35 tumorectomies starting from opening to closure of the dura. Volatile anesthetic absorbers were placed at three detection sites: 1) the surgeon's breathing zone, 2) the anesthesiologist's breathing zone, and 3) the farthest corner of the operation room. In the second sampling series that included 16 patients, the detector that had been in the corner of the operating room in the first series was now placed in the vicinity of the patient's mouth (within 5 cm). Sevoflurane captured by the absorbers was quantified by an independent chemist using chromatography.

RESULTS: Absorbers in the surgeon's breathing zone (0.24 ± 0.04 ppm) captured a significantly lower amount of sevoflurane compared with absorbers in the anesthesiologist's breathing zone (1.40 ± 0.37 ppm) and comparable with that in the farthest corner of the operation room (0.25 ± 0.07 ppm). There was no correlation between the amount of absorbed sevoflurane and the size of craniotomy window, even when adjusting for the variation in duration of surgery. In the second series of sampling, absorbers in the proximity of the patient's mouth captured the highest amount of sevoflurane (1.54 ± 0.55 ppm), followed by the anesthesiologist's (1.14 ± 0.43 ppm) and the surgeon's (0.15 ± 0.05 ppm) breathing zones.

CONCLUSIONS: The close proximity of the surgeon's breathing zone to the craniotomy window does not appear to be a source of increased exposure to sevoflurane. The observed higher exposure of the anesthesiologist to sevoflurane in the operating room environment warrants further exploration.

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The Effect of Gender on Compensatory Neuromuscular Response to Upper Airway Obstruction in Normal Subjects Under Midazolam General Anesthesia

Takao Ayuse, DDS, PhD*, Yuko Hoshino, DDS, PhD*, Shinji Kurata, DDS, PhD*, Terumi Ayuse, DDS, Hartmut Schneider, MD, PhD, Jason P. Kirkness, PhD, Susheel P. Patil, MD, PhD, Alan R. Schwartz, MD, and Kumiko Oi, DDS, PhD*

From the *Department of Clinical Physiology, Nagasaki University Graduate School of Biomedical Science; Department of Special Care Dentistry, Nagasaki University Hospital of Medicine and Dentistry, Nagasaki, Japan; and Division of Pulmonary and Critical Care Medicine, The Johns Hopkins School of Medicine, Johns Hopkins Sleep Disorders Center, Baltimore, Maryland.
Anesth Analg 2009; 109:1209-1218

P_{CRIT} and R_{US} were measured during midazolam sedation. In this study, we compared the effects of gender on compensatory neuromuscular responses to upper airway obstruction during midazolam general anesthesia. **METHOD:** Thirty-two subjects (14 men and 18 women) were studied. We constructed pressure-flow relationships to evaluate P_{CRIT} and R_{US} during midazolam anesthesia. The midazolam anesthesia was induced with an initial dose of midazolam (0.07–0.08 mg/kg bolus) and maintained by midazolam infusion (0.3–0.4 $\mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$), and the level of anesthesia was assessed by Ramsay score (Level 5) and Observer’s Assessment of Alertness/Sedation score (Level 2). Polysomnographic and hemodynamic variables were monitored while nasal pressure (via mask), inspiratory air flow (via pneumotachograph), and genioglossal electromyography (EMG_{GG}) were recorded. P_{CRIT} was obtained in both

BACKGROUND: Upper airway patency may be compromised during sleep and anesthesia by either anatomical alterations (mechanical properties) or disturbances in the neural control (compensatory neuromuscular responses). The pathophysiology of upper airway obstruction during anesthesia may differ between men and women. Recently, we reported that the upper airway mechanical properties were comparable with those found during natural nonrapid eye movement sleep, as evaluated by measurements of passive critical closing pressure (P_{CRIT}) and upstream resistance (R_{US}) during midazolam sedation. In this study, we compared the effects of gender on compensatory neuromuscular responses to upper airway obstruction during midazolam general anesthesia.

METHOD: Thirty-two subjects (14 men and 18 women) were studied. We constructed pressure-flow relationships to evaluate P_{CRIT} and R_{US} during midazolam anesthesia. The midazolam anesthesia was induced with an initial dose of midazolam (0.07–0.08 mg/kg bolus) and maintained by midazolam infusion (0.3–0.4 $\mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$), and the level of anesthesia was assessed by Ramsay score (Level 5) and Observer’s Assessment of Alertness/Sedation score (Level 2). Polysomnographic and hemodynamic variables were monitored while nasal pressure (via mask), inspiratory air flow (via pneumotachograph), and genioglossal electromyography (EMG_{GG}) were recorded. P_{CRIT} was obtained in both

the passive condition, under conditions of decreased EMG_{GG} (passive P_{CRIT}), and in an active condition, whereas EMG_{GG} was increased (active P_{CRIT}). The difference between the active P_{CRIT} and passive P_{CRIT} ($P_{CRIT P-A}$) was calculated in each subject to determine the compensatory neuromuscular response.

RESULTS: The difference between the active P_{CRIT} and passive P_{CRIT} ($P_{CRIT A-P}$) was significantly greater in women than in men (4.6 ± 2.8 cm H₂O and 2.2 ± 1.7 cm H₂O, respectively; $P < 0.01$), suggesting greater compensatory neuromuscular response to upper airway obstruction independent of arousal.

CONCLUSION: We demonstrate that the arousal-independent compensatory neuromuscular responses to upper airway obstruction during midazolam anesthesia were partially maintained in women, and that gender may be a major determinant of the strength of compensatory responses during anesthesia.

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The Electrocortical Effects of Enflurane: Experiment and Theory

James W. Sleight, MD*, Jeannette A. Vizuete, BS, Logan Voss, PhD*, Alistair Steyn-Ross, PhD, Moira Steyn-Ross, PhD, Charles J. Marcuccilli, MD, PhD, and Anthony G. Hudetz, DBM, PhD

From the *Department of Anaesthesiology, Waikato Clinical School, University of Auckland, Auckland, New Zealand; Department of Anesthesiology, Medical College of Wisconsin, Milwaukee, Wisconsin; Department of Engineering, University of Waikato, Hamilton, New Zealand; and Department of Pediatrics, Medical College of Wisconsin, Milwaukee, Wisconsin.

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BACKGROUND: High concentrations of enflurane will induce a characteristic electroencephalogram pattern consisting of periods of suppression alternating with large short paroxysmal epileptiform discharges (PEDs). In this study, we compared a theoretical computer model of this activity with real local field potential (LFP) data obtained from anesthetized rats.

METHODS: After implantation of a high-density 8 x 8 electrode array in the visual cortex, the patterns of LFP and multiunit spike activity were recorded in rats during 0.5, 1.0, 1.5, and 2.0 minimum alveolar anesthetic concentration (MAC) enflurane anesthesia. These recordings were compared with computer simulations from a mean field model of neocortical dynamics. The neuronal effect of increasing enflurane concentration was simulated by prolonging the decay time constant of the inhibitory postsynaptic potential (IPSP). The amplitude of the excitatory postsynaptic potential (EPSP) was modulated, inverse to the neocortical firing rate.

RESULTS: In the anesthetized rats, increasing enflurane concentrations consistently caused the appearance of suppression pattern (>1.5 MAC) in the LFP recordings. The mean rate of multiunit spike activity decreased from 2.54/s (0.5 MAC) to 0.19/s (2.0 MAC). At high MAC, the majority of the multiunit action potential events became synchronous with the PED. In the theoretical model, prolongation of the IPSP decay time and activity-dependent EPSP modulation resulted in output that was similar in morphology to that obtained from the experimental data. The propensity for rhythmic seizure-like activity in the model could be determined by analysis of the eigenvalues of the equations.

CONCLUSION: It is possible to use a mean field theory of neocortical dynamics to replicate the PED pattern observed in LFPs in rats under enflurane anesthesia. This pattern requires a combination of a moderately increased total area under the IPSP, prolonged IPSP decay time, and also activity-dependent modulation of EPSP amplitude.

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An Evaluation of Perioperative Pregabalin for Prevention and Attenuation of Postoperative Shoulder Pain After Laparoscopic Cholecystectomy

Seong-Hwan Chang, PhD, MD*, Hae-Won Lee, MD*, Hae-Kyoung Kim, PhD, MD, Seong-Hyop Kim, PhD, MD, and Duk-Kyung Kim, PhD, MD

From the Departments of *Surgery, and Anesthesiology and Pain Medicine, Konkuk University School of Medicine, Seoul, South Korea.

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Postlaparoscopic shoulder pain (PLSP) frequently follows laparoscopic surgery. In this placebo-controlled study, we evaluated the efficacy of two perioperative doses of

injected twice, an intraneural injection (20 nmol/2 μ L) 30 min before PSL, and a perineural injection (20 nmol/10 μ L) on Day 1 after PSL. Thermal hyperalgesia and tactile allodynia induced by PSL were evaluated by the thermal paw withdrawal test and the von Frey test, respectively.

RESULTS: As measured by Western blotting, in sham-operated mice, the levels of pERK1/2 in sciatic nerve were constant and the same as those in naive mice across Days 1-14. In PSL-operated mice, a significant increase in pERK1/2 was observed on Day 1 after PSL and persisted until Day 3. As measured by immunohistochemistry, immunoreactivity of pERK1/2 in PSL-operated sciatic nerve was markedly increased in comparison with that in sham-operated sciatic nerve on Day 1 after PSL. In the sciatic nerve on Day 1 after PSL, as indicated by double immunostaining, the increased immunoreactivity of pERK1/2 was colocalized with glial fibrillary acidic protein (GFAP), a marker of Schwann cells, but not F4/80, a marker of macrophages. PSL-induced thermal hyperalgesia was significantly attenuated by treatment with U0126 on Days 3, 7, and 14 after PSL. The PSL-induced tactile allodynia was also significantly attenuated by treatment with U0126 on Days 7 and 14 after PSL.

CONCLUSION: Activation of ERK in Schwann cells of the injured peripheral nervous system may play an important role in the development of neuropathic pain. Our results suggest that pERK itself and ERK-related mediators are potential therapeutic targets for the treatment of neuropathic pain.

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Lipid Emulsion Improves Recovery from Bupivacaine-Induced Cardiac Arrest, but Not from Ropivacaine- or Mepivacaine-Induced Cardiac Arrest

York A. Zausig, MD, DEAA*, Wolfgang Zink, MD, DEAA*, Meike Keil, MS, Barbara Sinner, MD, DEAA*, Juergen Barwing, MD, DEAA, Christoph H. R. Wiese, MD*, and Bernhard M. Graf, MD, MSc*

From the *Department of Anaesthesiology, University of Regensburg, Regensburg; and Department of Anaesthesiology, Emergency and Intensive Care Medicine, University of Goettingen, Goettingen, Germany.

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BACKGROUND: Cardiac toxicity significantly correlates with the lipophilicity of local anesthetics (LAs). Recently, the infusion of lipid emulsions has been shown to be a promising approach to treat LA-induced cardiac arrest. As the postulated mechanism of action, the so-called "lipid sink" effect may depend on the lipophilicity of LAs. In this study, we investigated whether lipid effects differ with regard to the administered LAs.

METHODS: In the isolated rat heart, cardiac arrest was induced by administration of equipotent doses of bupivacaine, ropivacaine, and mepivacaine, respectively, followed by cardiac perfusion with or without lipid emulsion (0.25 mL · kg⁻¹ · min⁻¹). Subsequently, the times from the start of perfusion to return of first heart activity and to recovery of heart rate and rate-pressure product (to 90% of baseline values) were assessed.

RESULTS: In all groups, lipid infusion had no effects on the time to the return of any cardiac activity. However, recovery times of heart rate and rate-pressure product (to 90% of baseline values) were significantly shorter with the administration of lipids in bupivacaine-induced cardiac toxicity, but not in ropivacaine- or mepivacaine-induced cardiac toxicity.

CONCLUSIONS: These data show that the effects of lipid infusion on LA-induced cardiac arrest are strongly dependent on the administered LAs itself. We conclude that lipophilicity of LAs has a marked impact on the efficacy of lipid infusions to treat cardiac arrest induced by these drugs.

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Fractal Cardiovascular Dynamics and Baroreflex Sensitivity After Stellate Ganglion Block

Chikuni Taneyama, MD*, and Hiroshi Goto, MD

From the *Anesthesia and Pain Relief, Chikuni Taneyama Clinic, Shiojiri City, Nagano Prefecture, Japan; and Department of Anesthesiology, University of Kansas Medical Center, Kansas City, Kansas.

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K9H 1.26 ± 0.18)☒ 0.46 ± 0.08 bpm/mm Hg, P < 0.05 ☒1.17 ± 0.35

)☒ 0.51 ± 0.13 bpm/min, P < 0.01☒☒8fIn6H☒

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-1.62 ± 0.22 , $P < 0.01$ vs -1.30 ± 0.80 ($P < 0.05$)
 -2.40 ± 0.80 , $P < 0.05$ vs -2.13 ± 0.50 , $P < 0.05$

vs -1.20 ± 0.40 ($P < 0.05$)

vs -1.20 ± 0.40 ($P < 0.05$)

vs -1.20 ± 0.40 ($P < 0.05$)

vs -1.20 ± 0.40 ($P < 0.05$)

BACKGROUND: It has been shown that stellate ganglion block can attenuate baroreflex sensitivity. Our primary purpose in this study was to determine whether fractal dynamics (dynamic change of self-similar fluctuation patterns) of not only heart rate but also systolic blood pressure variability are involved in attenuation of baroreflex sensitivity after stellate ganglion block.

METHODS: Sixteen young, healthy volunteers entered the study. Spectral analysis of heart rate and systolic blood pressure variability was performed before and 30, 60, 90, and 120 min after either right or left stellate ganglion block, separated by a 1 to 1-min interval, with 6 mL of 1% mepivacaine. Shortly after each spectral analysis, baroreflex sensitivity was assessed with the head-up tilt test.

RESULTS: Baroreflex sensitivity, assessed by the head-up tilt test, was significantly attenuated at 30 min after either right or left stellate ganglion block (1.26 ± 0.18 to 0.46 ± 0.08 bpm/mm Hg, $P < 0.05$ and 1.17 ± 0.35 to 0.51 ± 0.13 bpm/min, $P < 0.01$, respectively). Fractal slopes reflecting the degree of self-similarity of fluctuations were significantly increased at 30 min after either right or left stellate ganglion block (right stellate ganglion block—heart rate; -1.08 ± 0.30 to -1.62 ± 0.22 , $P < 0.01$; right stellate ganglion block—systolic blood pressure; -1.30 ± 0.80 to -2.40 ± 0.80 , $P < 0.05$; left stellate ganglion block—systolic blood pressure; -1.20 ± 0.40 to -2.13 ± 0.50 , $P < 0.05$). Fractal slope did not change after left stellate ganglion block with heart rate variability analysis.

CONCLUSIONS: Loss of complexity (status of being complex behavior) of both heart rate and systolic blood pressure variability, indicated by increased fractal slopes, is one mechanism in attenuating baroreflex sensitivity after stellate ganglion block.