In collaboration with the Canadian Critical Care Society,
the Canadian Journal of Anesthesia
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(Adult and Pediatric)
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The King Award is presented by the CCCS for the best oral presentation by a resident. The award is named after Garner King who was Professor and Head of the Department of Medicine at the University of Alberta. Garner King trained many of the first generations of critical care physicians in Canada and also many from abroad. He was the second president of the CCCS (from 1978–1980).

The Spanier Award, presented for the 2nd best oral presentation by a resident, is named in memory of Allen Howard Spanier who died on April 27th, 1999, at the age of 52. He was Associate Professor in Surgery, Medicine and Anesthesia, McGill University and Senior Surgeon and Chairman, Department of Adult Critical Care, Sir MB Davis Jewish General Hospital (Montreal, Quebec). He was President of the CCCS from 1992–1994.

Additional awards are presented for outstanding resident posters. An award is presented for the best abstract submitted to the Scientific Poster Sessions. Finally, the judges highlighted posters presenting a particular scientific or practical merit.

1st prize and King Award

Resident

EPINEPHRINE, CARDIAC ARREST AND MORTALITY

Authors: Conan McCaul, Patrick McNamara, Doreen Engelberts, Gregory Wilson, Alex Romaschn, Andrew Redington, Brian Kavanagh.

Institution Affiliation: Hospital For Sick Children, Toronto, Ontario, Canada.

Background: Epinephrine is the recommended pharmacological agent of choice for cardiopulmonary resuscitation. It increases coronary and cerebral perfusion pressure during chest compressions in a dose-related manner. Despite successful resuscitation myocardial dysfunction occurs and many patients die because of recurrent tachyarrhythmias, heart failure and hypotension due not only to underlying illness but also to the detrimental effects of resuscitative agents.

Objectives: To characterize the effect of epinephrine and the additive effects of concomitant calcium channel blockade on post-resuscitation myocardial performance.

Design/Methods/Subjects: Sprague-Dawley rats were anesthetized and exposed to one-minute asphyxial cardiac arrest. Standardized resuscitation was attempted with mechanical ventilation (FiO₂ 1.0), chest compressions and *iv* medication.

Measurements: (Series 1 & 2): Serial transthoracic echocardiography, invasive systemic arterial pressure and blood gas analysis were carried out at baseline and at regular intervals up to two hours post-resuscitation. LV systolic and diastolic function was assessed using M-Mode derived shortening fraction (LVSF) and end-diastolic dimension (LVEDD). (Series 3) Left atrial pressure was measured with a directly placed transduced fluid filled catheter.

Design: Series 1(closed chest): Animals were randomized to 0, 10 or 30 µg·kg⁻¹ epinephrine. Series 2 (closed chest): Animals received 30 µg·kg⁻¹ *iv* epinephrine and were randomized to 0.1 mg·kg⁻¹ verapamil or 0.9% saline. Series 3 (open chest): animals were randomized to 0, 10 or 30 µg·kg⁻¹ epinephrine.

Results: (Series 1) epinephrine administration was associated with increased mortality (0, 38.5 and 71.4% for control, 10 and 30 µg·kg⁻¹ doses respectively, $P = 0.002$). All groups developed dose related hypertension ($P < 0.001$) and tachycardia immediately after resuscitation ($P = 0.004$). An early non-sustained dose-related reduction in LVEDD occurred in both epinephrine groups ($P < 0.05$). (Series 2) verapamil administration prevented all mortality associated with high dose epinephrine (0 *vs* 100%). In addition verapamil attenuated post-resuscitation hypertension ($p=0.001$) without systolic dysfunction during the recovery phase. Early diastolic dysfunction was prevented by verapamil. (Series 3) Animals receiving epinephrine developed significant but transient early left atrial hypertension.

Conclusions: Epinephrine administration in a setting of brief asphyxial cardiac arrest is associated with dose-dependent diastolic dysfunction, left atrial hypertension, systemic hypotension and increased mortality. These effects are attenuated by pretreatment with calcium channel blockade.

2nd prize and Spanier Award

Resident

A META-ANALYSIS OF NON-INVASIVE POSITIVE PRESSURE VENTILATION TO FACILITATE WEANING FROM MECHANICAL VENTILATION

Authors: Karen E. A. Burns MD FRCPC,¹ Neill Adhikari MD FRCPC,²
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Purpose: To summarize the evidence comparing noninvasive positive pressure ventilation (NPPV) and invasive positive pressure ventilation (IPPV) weaning on clinical outcomes in intubated adults with respiratory failure.

Data sources: We searched MEDLINE (1966 to 2003), EMBASE (1980 to 2003) and the Cochrane Central Register of Controlled Trials (The Cochrane Library, Issue 2, 2003) for randomized controlled trials comparing NPPV and IPPV weaning. Additional data sources included personal files, conference proceedings and author contact.

Selection criteria: We selected randomized or quasi-randomized studies that 1) included adults, with respiratory failure, invasively ventilated for at least 24 hr; 2) compared extubation with immediate application of NPPV to weaning using IPPV; and 3) reported at least one of mortality, ventilator associated pneumonia (VAP), weaning failures, intensive care unit or hospital length of stay (LOS), total duration of mechanical ventilation, duration of endotracheal mechanical ventilation (ETMV), duration of mechanical ventilation related to weaning, adverse events or quality of life.

Data extraction: Two reviewers independently assessed trial quality and abstracted data.

Data synthesis: Compared to IPPV, the NPPV strategy decreased mortality (relative risk, 0.41 [95% CI 0.22 to 0.76]), VAP (relative risk, 0.28 [95% CI 0.09 to 0.85]), intensive care unit LOS (weighted mean difference, -6.88 days [95% CI -12.60 to -1.15 days]), hospital LOS (weighted mean difference, -7.33 days [95% CI -14.05 to -0.61 days]), total duration of mechanical ventilation (weighted mean difference, -7.33 days [95% CI -11.45 to -3.22 days]) and duration of ETMV (weighted mean difference, -6.79 days [95% CI -11.70 to -1.87 days]). There was no effect of NPPV on weaning failures or the duration of mechanical ventilation related to weaning and insufficient data to pool adverse events or quality of life.

Conclusions: Summary estimates from five studies of moderate to good quality demonstrated a consistent positive effect on overall mortality. Although the risks and consequences of this approach have not been fully elucidated, the NPPV weaning strategy is associated with promising evidence of net clinical benefit.

Outstanding Resident Poster #1

A RANDOMIZED PILOT STUDY OF THE USE OF N-ACETYL CYSTEINE TO PREVENT RENAL FAILURE IN TRAUMA PATIENTS AT RISK

Authors: G. Scott Milencoff, Christian Rabbat, Zakorka Povosky, Alison E. Fox-Robichaud.

Institution Affiliation: Regional Critical Care Programme and Dept of Medicine, McMaster University, Hamilton, Ontario, Canada.

Background: A recent study has shown the use of a drug, N-acetyl cysteine (NAC), prevents the development of contrast-induced renal failure in some patients. NAC has also been shown experimentally to limit ischemia-reperfusion injury. Our hypothesis is that N-acetyl cysteine given to trauma patients prior to *iv* contrast may preserve renal function.

Methods: We are conducting a blinded randomized controlled study, in which patients are randomly assigned to receive either NAC ($150 \text{ mg} \cdot \text{kg}^{-1}$ q 12 hr for 72 hr), or placebo (equal volume per weight of 0.9% saline). The first dose of drug is initially administered in the trauma suite during ongoing resuscitation of the patient and the second prior to the patient leaving the trauma suite for radiological investigations. Patients are followed for one week with prospective collection of creatinine and note of complications such as sepsis.

Results: There are complete data on 18 patients to date. The mean age of the patients was 34.6 ± 17.7 yr in Group A and 47.7 ± 15.0 yr in Group B ($P = 0.125$). The patients were also matched for weight (76.8 ± 14.2 kg *vs* 88.3 ± 18.8 kg, $P = 0.16$), Injury severity score (23.8 ± 6.4 *vs* 21.1 ± 5.0 , $P = 0.36$) and admission creatinine ($84.0 \pm 25.6 \mu\text{mol} \cdot \text{L}^{-1}$ for Group A *vs* $96.4 \pm 36.2 \mu\text{mol} \cdot \text{L}^{-1}$ for Group B) ($P = 0.4$). Both groups of patients received equal volume of *iv* contrast. To date there is no difference between the patients with respect to highest creatinine, change in creatinine clearance, or septic complications.

Conclusions: It is possible to carry out a randomized placebo-controlled study on trauma patients as they arrive in the emergency department. Thus far, the two groups are balanced. It is too early to determine if N-Acetyl cysteine has a protective effect on renal function in this patient population.

This study was funded by a grant from the Hamilton Health Sciences Research Development Fund.

Outstanding Resident Poster #2

NUMBER AND VOLUME OF PHLEBOTOMY IN A MULTIDISCIPLINARY INTENSIVE CARE UNIT: TIME TO ACT**Authors:** François Marquis MD, David Bracco MD, Stéphane Lamarre RN, Rédouane Bouali MD.**Institution Affiliation:** Critical Care Unit. Montréal University Hospital, Campus Hôtel-Dieu, Montréal, Québec, Canada.

Phlebotomy is a major cause of anemia in the intensive care unit (ICU). Many strategies have been developed to decrease phlebotomies, such as closed-circuit lines. In our study, we designed an educational program to stop unnecessary blood drawing.

Our setting is a 12-bed unit in a tertiary care medical facility, with cardiac and major vascular surgery.

All samplings (volume and frequency) were recorded for ten weeks before and two weeks after the intervention. The intervention was a tutorial given to physicians, residents, nurses and RTs. The points stressed were decreasing routine ABGs, the frequency of samplings, stopping useless samplings and grouping samplings.

Posters showing the volumes of blood drawn during the control period were placed in the unit.

Our intervention resulted in a 50% decrease in the number of ABGs drawn. The amount of wasted blood was also reduced. However, the delay between samplings was not increased nor was the number of requested laboratory tests decreased. The increase in glucose sampling is due to the implementation of tight glycemic control. The overall 31% reduction in samplings represents a decrease of 79 L of blood drawn per year.

The intervention had a significant effect on reducing the "ABG reflex". However, there is an urgent need to change routine samplings. Analyses should be grouped and requested according to clinical context. Repeated analyses should be avoided. The implementation of a tight glucose control strategy increased the number of measurements but had no significant effect on the volume of blood drawn.

Education is useful to decrease the blood volume sampled in the ICU but improvements are needed to decrease sampling frequency and the number of test panels requested.

Analyses per ICU day	Before	After
ABG	3.5	1.8**
Cap. Gluc	0.84	2.07*
CBC	1.39	1.49
Coag	1.16	0.97
Biochemistry	1.79	1.84
Lactate	0.28	0.08
Blood cultures	0.13	0.17
Total panel	9.1	8.5
Discarded vol. per 24 hr	18.9	8.5**
Total vol. per 24 hr	62.9	44.1**
Interval between sampling	7.20	7.29
	* $P < 0.5$	** $P < 0.01$

Best Scientific Poster Sessions Abstract

ALTERNATIVE COMPLEMENT CASCADE FACTOR B REGULATION IN SEPSIS IN HUMANS

Authors: Brent W. Winston md frcpc, Yong Huang, Connie Mowat.

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Complement factor B (Bf) is a component of the alternative pathway of complement activation. Bf is induced during the acute phase response of sepsis resulting in alternative complement cascade activation. Complement activation is an important component of the innate immune response to invading microorganisms, however, complement activation may also be an important contributor to shock in sepsis. The main mediators of acute phase proteins are inflammatory cytokines including TNF alpha and IFN gamma and bacterial products such as LPS. We have previously mapped the IFN gamma-responsiveness of the Bf promoter to consist of an ISRE site (at -130 bp) and a GAS site (at -90 bp) in murine macrophages. We have also identified a TNF alpha and a LPS responsive element consisting of a NF-kappa B cis binding site between -423 and -433 bp of the Bf promoter. We now question the mechanism of Bf gene regulation by TNF alpha, IFN gamma and LPS in human peripheral blood monocytes (PBMC) and whether PBMC's from patients with septic shock induce Bf.

Methods and results: (1) We investigated Bf mRNA expression by RT-PCR. Like murine macrophages, TNF alpha stimulation induced a low level of Bf mRNA expression by itself but TNF alpha acted synergistically with IFN gamma to induce Bf mRNA expression in PBMC's. (2) Co-stimulation with IFN gamma and TNF alpha and stimulation with LPS demonstrated time- and dose-dependent induction of Bf expression in PBMC's from human volunteers. (3) PBMC's isolated from ICU patients with septic shock showed increased Bf mRNA expression by RT-PCR when compared to control patients in the ICU. (4) We have cloned the human Bf promoter and show identical potential ISRE and GAS cis-binding sites as well as two putative NF-kappa B cis-binding sites similar to the murine promoter. (5) Using a number of human Bf promoter luciferase-reporter constructs to map the human Bf promoter, we show IFN gamma promoter responsiveness at the GAS cis element (-102 - -94) only. TNF alpha and LPS promoter.

Highlighted Poster #1

HYPERGLYCEMIA AND BRAIN TISSUE PH FOLLOWING TRAUMATIC BRAIN INJURY

Authors: David A. Zygun, L.A. Steiner, A.J. Johnston, Peter J. Hutchinson, P.G. Al-Rawi, D. Chatfield, P.J. Kirkpatrick, David K. Menon, Arun K. Gupta.

Institution Affiliations: University of Calgary, Calgary, Alberta, Canada; University of Cambridge, London, UK.

Introduction: Hyperglycemia is thought to exacerbate ischemic and traumatic neuronal injury via the accumulation of lactate and subsequent development of tissue acidosis. We sought to test the hypothesis that blood glucose levels are associated with brain tissue pH and that correction of hyperglycemia would result in an improvement in brain tissue pH.

Methods: A retrospective analysis of 34 patients with traumatic brain injury admitted to a tertiary neuroscience critical care unit was conducted. Continuous monitoring of brain pH was performed using a Neurotrend parenchymal sensor. Blood glucose levels were collected at the bedside as per the clinical protocol.

Results: A total of 428 glucose measurements were recorded during pH monitoring. Mean glucose was 7.1 mM (range 2.8 – 21.7 mM). The median (IQR) brain tissue pH associated with these glucose measurements was 7.11 (7.00, 7.19). A linear generalized estimating equation model was created with brain tissue pH as the dependent and blood glucose as the independent variable. This model predicted that for each 1 mM increase in blood glucose, brain tissue pH changed by -0.011 (95% CI: -0.016, -0.005, $P < 0.001$). Individual patient linear regression models confirmed this result (mean slope: -0.009; 95% CI: -0.017, -0.001). This relationship remained significant in a multivariable model that included cerebral perfusion pressure, brain tissue oxygen and carbon dioxide tension and brain temperature. Twenty-one episodes of significant hyperglycemia (≤ 11.1 mM) treated with *iv* insulin were identified. Insulin therapy significantly reduced blood glucose concentration from median (IQR) 11.9 (11.4, 13.6) to 8.8 (7.3, 9.6) ($P < 0.001$). Baseline brain tissue pH was not significantly different from brain tissue pH associated with the subsequent glucose reading < 11.1 mM ($P = 0.29$, but there was a suggestion of improvement if the change in blood glucose was large.

Conclusions: Blood glucose is associated with brain acidosis in patients with major head injury. Prospective studies are required to confirm these results and determine whether treatment of hyperglycemia improves outcome.

Highlighted Poster #2

EVALUATION OF TWO NOVEL STRATEGIES FOR POST-PYLORIC PLACEMENT OF ENTERAL FEEDING TUBES**Authors:** Andrew J.W. Samis, Daren Heyland, John Drover.**Institution Affiliation:** Critical Care Program, Queen's University and Kingston General Hospital, Kingston, Ontario, Canada.

Introduction: Enteral nutrition has many advantages for critically ill patients but can be complicated by volume intolerance in those who are gastrically fed. Post pyloric placement of the distal end of the feeding tube is considered more advantageous because: 1) there is better success at reaching nutritional targets and they are reached sooner when feeding into duodenum *vs* gastric feeding, and 2) there is a decreased incidence of pneumonia. We evaluated two different strategies for post-pyloric placement of feeding tubes. In the first method tube placement was assisted by an external magnet (MagnaFlow Tube, Arrow International Inc.). The second method is frictional placement, where small plastic tabs on the external surface of the tube allow the tube to be captured by peristalsis and carried passively (Tiger Tube, Cook Inc).

Methods: We prospectively evaluated the success in placing 15 MagnaFlow tubes and 10 Tiger Tubes. Success was determined by a radiograph showing the tip of the tube beyond the pylorus.

Results: Ten of 15 MagnaFlow tubes (67%) and nine of 10 Tiger Tubes (90%) were successfully placed past the pylorus. The average physician time required to place each feeding tube was less with the Tiger Tube (< 10 min for all tubes) *vs* the MagnaFlow tube (26 min, range 5 min to 60 min). The only complication encountered with either tube was minor nasal bleeding.

Conclusions: The frictional feeding tube achieves a high success rate in post-pyloric placement and may offer advantages to blind bedside placement. The magnetically-assisted tube requires more time to place and is less reliable.