

COVER PAGE FOR the PDF file of

Chan TC, Clausen J, Neuman T, Eisele JW, Vilke GM.
Does weight force during physical restraint cause respiratory compromise?

A PAPER PRESENTATION ABSTRACT listed in the
Ann Emerg Med, October 2003;42(4),
ACEP Research Forum Supplement: pS17.

Because this Paper Presentation's abstract was so BRIEF,
I typed the whole thing out for its original
posting in my Restraint Asphyxia Library.

This PDF file contains:

A copy of the Supplement's Cover ...

A copy of the Supplement's page that contains the CITED ABSTRACT ...
AND, the pages I typed and posted of the Paper Presentation's abstract.

To read my 2005 REVIEW related to this article, go to:

<http://www.charlydmiller/LIB05/2005chasresearchreviewpart3.html>

“A Comprehensive Review of Frequently Misinterpreted
and Misrepresented Restraint Research” PART THREE”

Besides being “kinder” than the review I wrote in 2003,
the 2005 review is much more IN-DEPTH.

YOURS, CHAS
(Ms. Charly D. Miller)

<http://www.charlydmiller.com>

Restraint Asphyxia Library
<http://www.charlydmiller.com/RA/RAlibrary.html>

Supplement to

Annals

of

Emergency

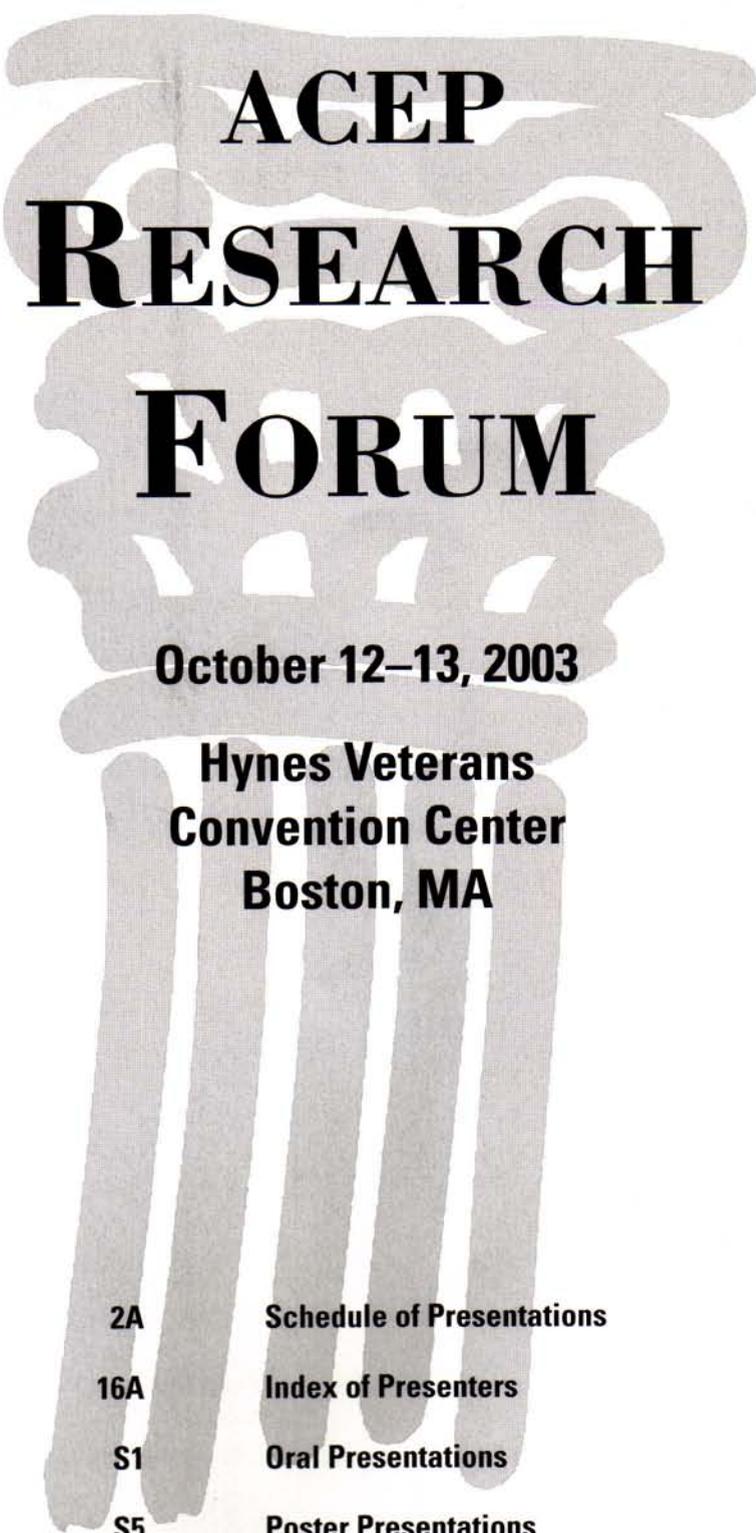
Medicine

www.mosby.com/AnnEmergMed

OCTOBER 2003

VOLUME 42, NUMBER 4

*Journal of the
American College of
Emergency Physicians*



ACEP RESEARCH FORUM

October 12–13, 2003

Hynes Veterans
Convention Center
Boston, MA

2A	Schedule of Presentations
16A	Index of Presenters
S1	Oral Presentations
S5	Poster Presentations

the gene encoding a galactosyltransferase necessary for O-antigen synthesis from *Klebsiella pneumoniae* 43816.

Results: Analysis of the mutant lipopolysaccharide (LPS) by sodium dodecylsulfate polyacrylamide gel electrophoresis confirmed the absence of O-antigen. In vitro, there were no detectable differences between wild-type *K pneumoniae* and the O-antigen deficient mutant in regard to avid binding by murine complement C3 or resistance to serum- or whole blood-mediated killing. Nevertheless, the 72-hour median lethal dose of the wild-type strain was 30-fold greater than that of the mutant (2×10^3 versus 6×10^4 colony-forming units) after intratracheal injection in ICR-strain mice. Despite being less lethal, the mutant organism exhibited comparable intrapulmonary proliferation at 24 hours compared with the wild type. Whole-lung chemokine expression (CCL3 and CXCL2) and bronchoalveolar inflammatory cell content was also similar between the 2 infections. However, whereas the wild-type organism produced bacteremia within 24 hours of infection in every instance, bacteremia was not seen in mutant-infected mice. Mutant and wild-type *K pneumoniae* were equally lethal when administered to C3 knockout mice, indicating a role for complement despite the lack of detectable in vitro differences.

Conclusion: These results suggest that during murine pneumonia with *K pneumoniae*, O-antigen contributes to lethality by increasing the propensity for bacteremia and not by significantly changing the early course of intrapulmonary infection.

58 Delayed Hypothermia Is Neuroprotective After Simulated Global Brain Ischemia

Lawrence EJ, Curtis KM, Neumar RW/University of Pennsylvania School of Medicine, Philadelphia, PA

Study objective: Clinical trials have demonstrated that therapeutic hypothermia improves the outcome of comatose cardiac arrest survivors. However, the therapeutic window for initiation of hypothermia remains unclear. This study evaluates the efficacy of delayed hypothermia after simulated brain ischemia in vitro.

Methods: Organotypic hippocampal slice cultures were prepared from P5 Wistar rat pups and cultured for 1 week before analysis. Ischemia was simulated by transferring slices to glucose-free buffer in a chamber purged with humidified 95% $N_2/5\%$ CO_2 at 37°C (98.6°F) for 30 minutes. Continuous hypothermia (33°C [91.4°F]) was induced at 0, 1, 2, or 4 hours after reoxygenation. Cell death was assayed at 24 hours by propidium iodide uptake. Propidium iodide fluorescence was quantified by computerized densitometry and compared by 1-way analysis of variance with Scheffe post hoc analysis.

Results: Simulated ischemia caused significant CA1 neuronal death compared with nonischemic controls. Hypothermia initiated 0, 1, 2, or 4 hours after reoxygenation reduced posts ischemic CA1 neuronal death by $47\% \pm 34\%$ ($P=.003$), $85\% \pm 4\%$ ($P<.001$), $88\% \pm 3\%$ ($P<.001$), and $89\% \pm 5\%$ ($P<.001$), respectively.

Conclusion: In this model of simulated global brain ischemia, delaying initiation of hypothermia for up to 4 hours after reoxygenation does not reduce therapeutic efficacy.

59 A Comparison of Artificial Turf in Tests of Impact Attenuation

Naunheim RS, Parrott H, Standeven J/Washington University, St. Louis, MO

Study objectives: The purpose of the study is to test a newer form of artificial turf used by a professional football team to determine whether its impact attenuation differs significantly from that of older types of artificial turf.

Methods: An instrumented computerized recording sphere, an impact recording device (Techmark, Inc., Lansing, MI), was dropped 20 times from a height of 48 inches on to 5 types of turf used by a professional football team: FieldTurf used on an inside field, outside grass at 24.5°C (76.1°F), outside grass at 0°C (32°F), AstroTurf used on an inside field, and AstroTurf used at a domed stadium. FieldTurf is an artificial surface composed of a subsurface of shredded tires. AstroTurf is a grass carpet over a subsurface of foam, which was tested with 2 thicknesses of foam. The surface at a domed stadium consisted of an AstroTurf carpet on top of 1.6 cm of padding over concrete. The second AstroTurf surface was an AstroTurf carpet on top of 2.5 cm of padding over concrete. All measurements were made in g, the acceleration caused by gravity.

Results: A 1-way analysis of variance was done to compare all 5 fields, which were found to be significantly different. ($F=220.02$, $df=4,95$, $P<.0001$). Duncan's range test shows that the outside grass at 24.5°C and the domed stadium are not different and the FieldTurf and AstroTurf with 2.5 cm of padding are not different. Although

there were no significant differences found in the AstroTurf field with 2.5 cm of padding and the FieldTurf field, there were significant differences in the FieldTurf surface at different sites on the field ($F=83.57$, $df=4,15$, $P<.0001$). The g force experienced on the FieldTurf field at the 30-yard line was 238, whereas on the old AstroTurf field, the same spot produced a g force of 188.

Conclusion: The indoor practice field used by a professional football team was changed from AstroTurf with 2.5 cm of padding to FieldTurf in an attempt to improve impact attenuation. Overall, there was no significant difference in impact attenuation; however, there were areas within the field that were significantly compacted, causing some spots on the field to be much harder than the AstroTurf surface, which is presumably caused by the compacting of the shredded rubber on which the field is based.

60 Does Weight Force During Physical Restraint Cause Respiratory Compromise?

Chan TC, Clausen J, Neuman T, Eisele JW, Vilke GM/University of California—San Diego, San Diego, CA; Forensic Pathology and Medicine, San Diego, CA

Background: Violent, combative patients often require physical restraint by emergency department, hospital, out-of-hospital, and law enforcement personnel. Concern has been raised that weight force, commonly applied during the restraining process, can compromise respiratory function, placing individuals at risk for asphyxiation.

Study objectives: We sought to determine the impact of weight force on pulmonary and respiratory function in a simulated restrained patient. We hypothesized that weight force would result in changes in pulmonary and respiratory characteristics.

Methods: Ten volunteers completed a randomized crossover, controlled trial in a pulmonary function laboratory. Subjects were placed in the sitting, prone restraint (PR), PR with 25-lb (11.25 kg) sandbag weight (PR+25), and PR with 50-lb (22.5 kg) sandbag weight (PR+50) placed on the back between the scapula. Subjects remained in each position for 5 minutes, during which spirometry was performed at 1 and 5 minutes, and transcutaneous oximetry (O_2 sat) and end-tidal CO_2 levels ($ETCO_2$) were monitored continuously. Data were collected on percentage of predicted forced vital capacity (%predFVC), percentage of predicted forced expiratory volume in 1 second (%predFEV₁), O_2 sat, and $ETCO_2$ and compared by repeated measures analysis of variance with post hoc testing ($P<.05$ significance, STATA software).

Results: At 1 minute, mean %predFVC decreased from 101% to 87.1% to 84.7% to 84.2% and mean %predFEV₁ decreased from 98.2% to 83.4% to 82.0% to 80.0% (sitting to PR to PR+25 to PR+50, respectively). Similar results were found at 5 minutes into each position (mean %predFVC decreased from 102% to 86.8% to 82.5% to 80.5%; mean %predFEV₁ decreased from 99.3% to 82.2% to 79.5% to 74.7%, respectively). However, mean O_2 sat remained above 95% and mean $ETCO_2$ levels remained below 45 mm Hg throughout the 5-minute period for all positions.

Conclusion: Prone restraint with weight force resulted in decrements in FVC and FEV₁ consistent with a restrictive pulmonary function pattern but did not result in evidence of hypoxia or hypercapnia.

61 Postextreme Endurance Syndrome in 2,113 Triathletes: Description of a New Clinical Syndrome

Collinsworth A, Kass L, Petersen HA, Carmack J, Lyle W, Akbar S, Schay J, Brenner BE/University of Arkansas for Medical Sciences, Little Rock, AR

Study objective: The acute medical implications of triathlon participation have not been well described. According to 2 recent triathlons, we report a new constellation of signs and symptoms termed the postextreme endurance syndrome (PEES). The purpose of this study was to describe the syndrome, risk factors, and recommendations for preparations by race medical personnel.

Methods: This was a prospective, observational study of the Ironman Triathlon in Panama City Beach, FL, in October 2001 and of the Ultramax Triathlon in Table Rock, MO, in September 2002. Demographic data and vital signs were collected on all racers before the race. Physical examination findings, treatments, and outcomes were collected prospectively on all individuals who presented for medical treatment. t Test, χ^2 , and 95% confidence intervals [CIs] are used as appropriate.

Results: Two thousand one hundred thirteen individuals (82% men, median age 37 years) participated in the 2 triathlons. Two hundred seventy-six (13%) racers sought medical attention for symptoms consistent with PEES, including muscle cramps (8%), nausea or vomiting (21%), relative hypothermia (temperature $<98^\circ F$

Does Weight Force During Physical Restraint Cause Respiratory Compromise?

CITATION:

Chan TC, Clausen J, Neuman T, Eisele JW, Vilke GM.
Does weight force during physical restraint cause respiratory compromise?
Ann Emerg Med, October 2003;42(4),
ACEP Research Forum Supplement: pS17.

Presenters/Abstract "Authors":

Chan TC, Clausen J, Neuman T, Eisele JW, Vilke GM.
University of California--San Diego, San Diego, CA; Forensic Pathology
and Medicine, San Diego, CA.

Background: Violent, combative patients often require physical restraint by emergency department, hospital, out-of-hospital, and law enforcement personnel. Concern has been raised that weight force, commonly applied during the restraining process, can compromise respiratory function, placing individuals at risk for asphyxiation.

Study objectives: We sought to determine the impact of weight force on pulmonary and respiratory function in a simulated restrained patient. We hypothesized that weight force would result in changes in pulmonary and respiratory characteristics.

Methods: Ten volunteers completed a randomized crossover, controlled trial in a pulmonary function laboratory. Subjects were placed in the sitting, prone restraint (PR), PR with 25-lb (11.25 kg) sandbag weight (PR+25), and PR with 50-lb (22.5 kg) sandbag weight (PR+50) placed on the back between the scapula. Subjects remained in each position for 5 minutes, during which spirometry was performed at 1 and 5 minutes, and transcutaneous oximetry ($O_2\text{sat}$) and end-tidal CO_2 levels ($ETCO_2$) were monitored continuously. Data were collected on percentage of predicted forced vital capacity (%predFVC), percentage of predicted forced expiratory volume in 1 second (%predFEV1), $O_2\text{sat}$, and $ETCO_2$, and compared by repeated measures analysis of variance with post hoc testing ($P<.05$ significance, SATA software).

Results: At 1 minute, mean %predFVC decreased from 101% to 87.1% to 84.7% to 84.2% and mean %predFEV1 decreased from 98.2% to 83.4% to 82.0% to 80.0% (sitting to PR to PR+25 to PR+50, respectively). Similar results were found at 5 minutes into each position (mean %predFVC decreased from 102% to 86.8% to 82.5% to 80.5%; mean %predFEV1 decreased from 99.3% to 82.2% to 79.5% to 74.7%, respectively). However, mean O₂sat remained above 95% and mean ETCO₂ levels remained below 45 mmHg throughout the 5-minute period for all positions.

Conclusions: Prone restraint with weight force resulted in decrements in FVC and FEV1 consistent with a restrictive pulmonary function pattern but did not result in evidence of hypoxia or hypercapnia.

Read CHAS' NEW (2005) Opinion Paper related to this article!:

"A Comprehensive Review of Frequently Misinterpreted and Misrepresented Restraint Research" PART THREE

(Besides being "kinder," the 2005 review is much more IN-DEPTH.)

<http://www.charlydmiller.com/LIB05/2005chasresearchreviewpart3.html>