

COVER PAGE FOR
the PDF file of

Eisele JW, Chan TC, Vilke GM, Clausen J.

Comparison of respiratory function
in the prone maximal restraint position
with and without
additional weight force on the back.

The abstract of an UNPUBLISHED paper presented at the annual meeting
of the **American Academy of Forensic Science**,
in Reno, Nevada: February 21-26, 2000.

Because I was FAXed a poor copy of the conference book page containing this
Paper Presentation's abstract, I typed the whole thing out for its original
posting in my Restraint Asphyxia Library.

This PDF file of the abstract contains:
A copy of the FAXed page ... and
the Paper Presentation's abstract, as I typed and posted it.

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 2002,
the 2005 review is much more IN-DEPTH.

YOURS, CHAS
(Ms. Charly D. Miller)

in organs in need of large amount oxygen such as the cerebrum, lungs, liver and kidneys. PEH, such as in this case, can be negative as PEH quickly disappears from body fluids. Autopsy and clinical findings pointed to poisoning, and and fustoria tablets were found in the wheat

poisoning must (caulis) living in phosphorus con- to sudden infant

From the annual meeting of the American Academy of Forensic Science, in Reno, NV; February 21-26, 2000.

G69 Comparison of Respiratory Function in the Prone Maximal Restraint Position With and Without Additional Weight Force on the Back

John W. Eisele, MD, Department of Pathology, Theodore Chan, MD, Gary Wilke, MD, Tom Neuman, MD, Department of Emergency Medicine, and Jack Clausen, MD, Department of Medicine, University of California at San Diego Medical School, 9500 Gilman Drive, La Jolla, CA*

After attending this presentation, the participant will understand the effects on respiratory function of being placed in the prone, maximal restraint position ("hogtying" or "hobble restraint") without weight on the back and with varying weights placed on the upper back.

Although the technique of prone maximum restraint ("hogtying" or "hobble restraint") is widely used in controlling violent or irrational individuals and is generally considered safe, there are several case reports of deaths occurring during this procedure. Some of the fatalities have in addition had weight applied to their back during the restraint. A previous study from this laboratory has presented data regarding the effects on respiratory function during maximum restraint. This study is an extension of the previous investigation in which the additional factor of weight applied to the back is included.

Ten healthy volunteers were recruited and informed of the procedure, and gave verbal and written consent. They were screened for pulmonary function in the sitting position and rejected if baseline forced vital capacity (FVC) or forced expiratory volume in one second (FEV1) were outside of acceptable limits. They were then randomly placed in one of four positions and subsequently crossed-over into the three other positions. These were: (1) sitting position, (2) prone maximal restraint position with no weight on the back, (3) prone maximal restraint position with 25 pounds placed between the shoulder blades, and (4) prone maximal restraint position with 50 pounds placed between the shoulder blades. After being placed into position, a baseline blood pressure was determined and this was repeated after 4 minutes in the position. At 1 and 5 minutes after the being placed in each position, spirometric measurements including FVC and FEV1 were determined. During the procedures the subjects were monitored continuously with sensors for transcutaneous oximetry (O2sat) and end-tidal CO2 (etCO2), and readings were recorded every 30 seconds.

Results for the first subjects indicate that FVC showed the anticipated decrease to approximately 82% of expected between sitting and prone restraint. After 25 pounds was added there was a decrease to approximately 79% of expected and after 50 pounds a decrease to approximately 75%. FEV1 showed a similar progressive decrease, averaging 81% of expected after prone restraint, 76% of expected with 25 pounds and 72% of expected with 50 pounds. O2 saturation showed a slight decrease, and etCO2 showed minimal and inconsistent changes. Blood pressure showed no significant change. None of the parameters showed major changes during the 5 minute period of observation.

These results indicate that weight placed on the back during maximal restraint does cause a decrease in spirometry parameters. Data collected to date, however, does not indicate a significant effect on oxygen saturation or end tidal CO2. The statistical and clinical significance of these findings will be presented and discussed. Although body size and habitus are matters of concern in evaluating the effects of restraint, the subjects in this study were intentionally kept within an average range; because of the small number of subjects it was felt that including extremes of body size would decrease the statistical significance of the results. Likewise, the amount of weight in this study was limited, but the authors' lack of specific knowledge of the effects of this procedure made this a matter of safety for the volunteers. These are clearly matters of concern for future studies.

This study was supported by a grant from the Pathology/Biology Section Research Committee of the American Academy of Forensic Sciences.

Maximal Restraint, Hogtying, Pulmonary Function

G70 Airway Obstruction Due to Blunt Trauma—A Swell Case

Adrienne A. Szgovia, MD, and Aldo J. Fusaro, DO, Office of the Medical Examiner, County of Cook, Chicago, IL; John Latall, MD, University of Illinois Medical Center, Chicago, IL; and Mitra B. Kuletkar, MD, Office of the Medical Examiner, County of Cook, Chicago, IL*

After attending this presentation, the participant will be able to recognize the relevant clinical information, the physical findings, and the laboratory findings characteristic of Hereditary Angioedema (HAE), and become familiar with the genetics, pathogenesis, and precipitating causes of HAE.

The authors describe a previously undiagnosed case of hereditary angioedema type I in a 38-year-old white female with no family history. The subject was struck once in the right parietal area by her boyfriend during an altercation. Within two days, the subject developed massive edema of the face, head, and neck, requiring intubation. Decreased serum C1 esterase inhibitor (C1EI), C4, total complement, and C3 levels confirmed the diagnosis of type I hereditary angioedema. She suffered a progressively worsening hospital course, which included the development of renal failure due to rhabdomyolysis, liver failure, subclavian line sepsis, and disseminated intravascular coagulation. She expired 19 days after admission.

Since its first description by Quinke in 1882, the amount of information known about hereditary angioedema (HAE) has increased considerably. HAE is due to either decreased synthesis of the normal complement protein C1EI or synthesis of an abnormal (nonfunctional) protein. There are two heritable subtypes of HAE, both of which have autosomal dominant inheritance patterns. Type I account for 85% of cases. These patients have markedly reduced levels of C1EI due to decreased synthesis of the inhibitor protein. Type II accounts for the remaining 15%. These patients have synthesis of an abnormal inhibitor protein. Angioedema may be acquired. These patients have normal synthesis of C1EI, but its catabolism is markedly increased. Most cases are the result of lymphoproliferative disorders.

C1EI is located on chromosome 11 in the region p11.2 — q13. C1EI belongs to a group of inhibitors that includes antithrombin III and alpha-1-antitrypsin. It is a circulating alpha2-globulin which blocks the activation of C1s (a complement component which cleaves C4). Deficiency of C1EI results in the activation by trauma of the classical complement cascade, with recurrent and incapacitating episodes of localized edema. The events leading to swelling are uncertain. One possibility is that trauma triggers the formation of plasmin from plasminogen which then activates C1 esterase. Deficiency of C1EI leads to

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"PAPER PRESENTERS": John W. Eisele MD, Department of pathology; Theodore Chan MD, Gary Vilke MD, Department of Emergency Medicine; and Jack Clausen MD, Department of Medicine; University of California at San Diego Medical School, 9500 Gilman Drive, La Jolla, CA.

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* Since *I* had to look it up to be sure I understood its use;

here is the 17th ed. of Taber's Medical Dictionary's definition of *habitus* =

1. A physical appearance, body build, or attitude.
2. A physical appearance that indicates a tendency for a person to develop a specific disease.

However, as used in this context, it seems clear that the abstract authors' use of the term, "habitus," primarily refers the WEIGHT of someone's body.