CORRESPONDENCE

Metabolic Acidosis in Restraint-associated Cardiac Arrest

We read with interest the recent article by Dr. Hick et al. about metabolic acidosis in restraint-associated cardiac arrest.[1] We commend the authors for their case series, which addresses the important issue of sudden death in association with vigorous physical activity and restraint, and the possible role of severe metabolic acidosis. We would, however, recommend caution in interpreting the observations made in this series of five patients.

The contribution of restraint struggle and body position to the profound acidosis reported in these patients remains unclear. First, a good portion of the metabolic acidosis may have simply been due to cardiopulmonary arrest, as blood gas sampling seems to have taken place after the initial arrest and resuscitation efforts on these patients.[2,3]

Second, it is unlikely fully restrained individuals, even with struggling, are capable of generating oxygen consumptions (VO_2) sufficient to generate a profound acidosis. Generally, exercise involving antigravity muscles plus movement of the extremities produces the highest VO_2 levels.[4] All subjects described in this series were in a state of heavy physical exertion from fighting or fleeing immediately prior to being physically restrained. Generation of a portion of the observed metabolic acidosis from high VO_2 and anaerobic metabolism more likely occurred during this exertion rather than struggle after being restrained.

Third, the role of cocaine in these cases cannot be overstated. All five patients had evidence of cocaine on toxicologic screening, and their behavior (which ultimately led to their physical restraint) was consistent with sympathomimetic intoxication or even what has been termed "toxic" or "excited delirium."[5,6] In states of catecholamine excess (endogenous and exogenously administered), impaired oxidative phosphorylation leading to metabolic acidosis has been demonstrated.[7,8] As the authors note themselves, stimulant drug use, particularly when combined with physical exertion, can lead to profound metabolic acidosis without physical restraint.[1,9]

Regardless of the etiology of the acidosis, there is currently no evidence to support the authors' speculation that prone and hobble body positioning "may significantly impact . . . [the] ability to develop a compensatory respiratory alkalosis" as evidenced by a "20%" drop in "maximal ventilatory volume" [sic] referenced to our study.[10] We exercised subjects well beyond anaerobic threshold to a mean heart rate of 169 beats/min and pH 7.28. During 15-minute rest periods in the sitting and hobble positions, there were no differences in heart rate recovery, $PaCO_2$ (as reported), or pH levels (7.33 vs 7.34, respectively).[10]

Moreover, none of the five victims reported in this case series were in the hobble position. Four were restrained prone, a position in which we found a much smaller (15%) drop in maximal voluntary ventilation, or MVV (and only slightly more than the 10% drop seen in the supine position).[10] The remaining fifth victim was "on his side" when he suffered a sudden, ultimately fatal cardiac arrest. This case casts doubt on the authors' recommendation that "emphasizing side rather than prone positioning may eliminate some of the problems."[1]

Finally, there are several errors in the content of a number of citations. The authors' claim that Pudiak and Bozarth placed cocaine-injected rats in restraint cylinders such that they did not have "freedom to turn around" is misleading. In that study, the rats had enough room to move and reverse their position, a less restrictive condition that "might better be described as confinement stress."[11] A small subgroup of rats (n = 5, outside the study's main methodology and for which no statistical data were provided) were placed in more restrictive confines, but even in that group, "ample space was provided for normal respiration."[11] The high mortality rate seen in both groups of rats suggests changes in respiratory function do not play a role in the pathophysiology of restraint deaths. The authors' reference to the article by Bell et al. attributing deaths of subjects in a "hobbled position" to positional asphyxiation is also in error. No victim in that case review was described as being restrained in the hobble or hog-tie position.[12]

In summary, there is little evidence that respiratory changes associated with specific body positions significantly contribute to the metabolic derangements and pathophysiology of sudden death in restrained individuals. More likely, as shown in this case series, a multitude of other factors may explain both the profound metabolic acidosis and sudden deaths seen in these individuals.—THEODORE C. CHAN, MD, TOM NEUMAN, MD, and GARY M. VILKE, MD, *Department of Emergency Medicine,* JACK CLAUSEN, MD, *Division of Pulmonary Medicine, Department of Internal Medicine,* and RICHARD F. CLARK, MD, *Division of Toxicology, Department of Emergency Medicine, University of California—San Diego, San Diego, CA*

Key words. metabolic acidosis; restraints; cardiac arrest; acidosis; sudden death.

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Cardiorespiratory Consequences of the Hobble Restraint

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We read with great interest the article by Dr. Hick et al. on metabolic acidosis in restraint-associated cardiac arrest.[1] The authors offer several factors as possible explanations for acidosis while the patient is in the hobble restraint: exacerbation of exercise-induced lactic acidosis, drug-induced factors, and respiratory impairment. We have investigated cardiopulmonary response to the hobble restraint in the upright and prone positions in six male volunteers in a randomized crossover trial.[2] No change was observed in the investigated cardiopulmonary parameters after use of the hobble restraint in the upright position. After use of the hobble restraint in the prone position, mean forced vital capacity decreased by 39.6%, mean forced expiratory volume decreased by 41%, mean end-tidal carbon dioxide increased by 14.7%, mean heart rate decreased by 21.3%, mean systolic blood pressure decreased by 32.3%, mean diastolic blood pressure decreased 26.1% and mean cardiac output decreased by 37.4% (p for all reported changes <0.01). The hobble restraint in the prone position clearly leads to a dramatic impairment of hemodynamics and respiration. In amendment to the authors' explanations for the development of severe acidosis during use of the hobble restraint, we think that the dramatic hemodynamic deterioration should be considered as a main contributing factor.—GEORG ROEGGLA, MD, *Department of Internal Medicine, Municipal Hospital of Neunkirchen, Austria,* HANNELORE ROEGGLA, MD, *Department of Medical Psychology, University of Vienna, Austria,* BERTHOLD MOSER, MD, *Department of Internal Medicine, Municipal Hospital of Neunkirchen, Austria,* and MARTIN ROEGGLA, MD, *Department of Emergency Medicine, University of Vienna, Austria*

Key words. restraints; hobble restraint; acidosis; metabolic acidosis; cardiorespiratory effects.

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In reply:—We thank our colleagues for their thoughtful contributions and for the opportunity to clarify some well-made points. It is gratifying to see that there is great interest in the area of restraint use and restraint-associated death.

In reporting our case series, we hoped to draw the attention of public safety and emergency medical personnel to the association observed between cardiopulmonary arrest occurring in the setting of restraint use, and metabolic acidemia significantly greater than that usually observed in routine cardiac arrest management. We do not have adequate information to assert the end cause of death, nor can we do much besides speculate on contributing causes and hope that future research will be able to better control the myriad variables that make restraint-associated deaths so difficult to study.

We agree with Dr. Chan and colleagues that the acidosis observed in our patients almost certainly had its genesis in the profound physical exertion before and during their restraint process, and concur that the restraint position itself is unlikely to generate a profound acidosis. Additionally, cocaine was likely pivotal in the development of severe metabolic acidosis in our series, but without serum evidence of concurrent cocaine intoxication in two of our five cases of cardiopulmonary arrest, we did not wish to overstate this association. Of note, all of our nonarrest cases (Table 1 in our case series report[1]) admitted to crack cocaine use. Though excited delirium from cocaine is probably a major contributor to death in these circumstances, this distinction is of limited benefit when confronted with a struggling patient, as excited delirium may be due to causes other than a sympathetic toxidrome.

It is doubtful that the levels of acidosis shown by our patients were due simply to the cardiac arrest state. Our cases in Table 1 never arrested, yet had moderate to profound metabolic acidosis. In arrested patients, the large series by Dybvik et al. and that of McGill and Ruiz support that our patients were far more acidotic than those in routine cardiac arrest circumstances.[2,3] Fillmore et al.'s series of 14 patients, cited by Dr. Chan et al., also noted minimal pre-intubation acidemia (pH 7.31 on average).[4] Interestingly, in Chazan et al.'s series of patients (also cited by Chan et al.), the most acidotic patients were those with respiratory compromise, and "superimposed hypercapnia appeared to be the critical factor leading to the development of profound acidosis."[5] We believe this supports our recommendation to minimize positional restrictions on the respiratory abilities of these patients.

We do not wish to overstate our conclusions regarding positioning of patients during restraint. There does not seem to be any debate over the fact that prone or hobble positioning impedes respiratory function, and appreciate the further contributions of Dr. Roeggla and colleagues.[6] The controversy continues over whether this positional decrease in ventilatory ability has any clinical effect. A healthy patient

model who does not continue to struggle against restraints apparently suffers no ill effects from moderate reductions in ventilatory capacity.[7] The detriment to a struggling, profoundly acidotic patient whose life may depend on the ability to develop a respiratory alkalosis has not been studied, and thus we agree with previously expressed concerns that this reduction cannot be inferred to be "not clinically relevant."[8]

We believe it is reasonable to recommend to our public safety agencies that patients be placed in a position that least compromises their ventilatory efforts, so long as it is safe to do so. However, we suffer under no delusions that position alone is lifesaving. This indeed is evidenced in case 5, where restraint position made far too small of a difference to salvage this patient. Though prompt emergency medical services (EMS) response and aggressive correction of acidemia did result in return of spontaneous circulation, the patient died. Whatever position may be used is doubtless of far less clinical significance than pre-arrest recognition and management of these patients' behavioral and metabolic derangements.

The study by Pudiak and Bozarth is not connected with respiratory effects of restraint position, as the study was carefully designed to allow the subject to breathe unhindered. Rather, it is an intriguing example of how cocaine may contribute substantially to death in the setting of restraint stress, in concordance with Dr. Chan and colleagues' comments on the subject. When a 320–375-g rat is confined in a 3-inch (7.6-cm) tube, we think that it is accurate to say that the rat lacked the *freedom* to turn around, though perhaps we could have clarified this with the addition of "in a normal fashion," or added additional specifics about the experiment model that might have been more satisfactory. The fact that some experimental subjects were able to achieve a position reversal in the tube during the restraint interval "indicated that confinement in the restraint cylinder did not prohibit all movement of the subject."[9]

Dr. Chan et al. are correct that the large series by Bell et al., which described patients who placed *themselves* into positions that compromised their ventilatory ability (and apparently contributed to their deaths), did not involve any patients in hobble restraints.[10] Language that more clearly differentiated the patient groups involved in the Bell et al.[10] and Reay et al.[11] articles was changed in a draft revision. The primary author apologizes for the error, and appreciates the opportunity to correct the citation. We hope that other readers were not misled, as it was certainly not our intent.

We appreciate Dr. Roeggla and colleagues' bringing their work to our attention.[6] The levels of cardiorespiratory embarrassment they have documented are striking, and we look forward to additional studies that will confirm these results.

There is no doubt that restraint death is a multifactorial process and that no single mechanism explains this phenomenon. What we bring to this conversation is not a position on positions, if you will, but an awareness via our case series that profound metabolic acidosis may contribute significantly to restraint-associated deaths.

Hopefully, the close attention being given to this subject will prompt widespread recognition that patients who continue to struggle against maximal restraint techniques are at high risk for complications and death. Early involvement of EMS personnel, and aggressive management of these patients, with rapid detection and correction of metabolic derangements, can lead to good outcomes in these difficult situations.—JOHN L. HICK, MD, STEPHEN W. SMITH, MD, and MICHAEL T. LYNCH, MD, *Department of Emergency Medicine, Hennepin County Medical Center, Minneapolis, MN*

Key words. metabolic acidosis; acidosis; restraints.

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