

Response to: Prone restraint cardiac arrest – A comprehensive review of the scientific literature and an explanation of the physiology

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We read with interest the recent review article by Steinberg titled 'Prone restraint cardiac arrest: A comprehensive review of the scientific literature and an explanation of the physiology'.¹ We appreciate the author highlighting our work in a separate section, as well as reviewing the scientific studies of many of our colleagues, but urge caution in the author's conclusion these sudden deaths in agitated individuals restrained in the prone position are 'most likely due to metabolic acidosis exacerbated by inadequate ventilation and a decrease in CO (cardiac output)'.

Steinberg hypothesises the ventilatory changes seen with the prone position 'limits' ventilation and the ability to generate a compensatory 'respiratory alkalosis' by lowering arterial CO₂ in an individual with an already preexisting severe metabolic acidosis. In our original study, the decreases in pulmonary function testing in the prone position were similar to those in the supine position and moreover, there was no evidence of elevated CO₂ levels to suggest an inability to ventilate adequately, nor differences in pH levels with prone restraint.^{2,3}

Steinberg appears to equate a decrease in maximal voluntary ventilation (MVV) as a reduction in ventilation during restraint. MVV is not equivalent to the amount of ventilation needed for a given degree of oxygen consumption (VO₂). As VO₂ increases, ventilation increases and consequently the additional CO₂ generated by aerobic metabolism is excreted. Until the anaerobic threshold is achieved, P_aCO₂ and F_ECO₂ remain normal (~40 mm Hg and 4%, respectively). The earliest sign of ventilatory insufficiency is an elevated CO₂ level. In ours and other studies such as by Roeggla et al.⁴ cited by the author, there were no abnormal elevations of F_ECO₂, indicating even with a reduced MVV, ventilation was appropriate to that degree of exercise and at a level appropriate to maintain a normal pH.

Steinberg cites Parkes' work showing 'significant' lung function changes, but fails to note these investigators concluded 'it remains open to debate whether these constitute clinically significant and potentially fatal restrictions'.⁵ Steinberg partially cites Cary's work noting 'reductions in ventilatory capacity', however, the author appears to

disregard Cary's complete conclusion that restraint 'did not impair cardiorespiratory function'.⁶ In Cary's experiment, individuals were exercised to 85% of the age predicted maximum heartrate and were then placed in a prone restrained position with 165 lbs. on their backs while at the same time their abdomen was compressed over a rolled up piece of carpet ~9" in diameter. They reported F_ECO₂ was the same as in individuals who were exercised and then placed in a seated position. This clearly demonstrates the alveolar ventilation in this group was normal and the same compared to those in a seated position after heavy exercise. In essence, the reported changes in MVV of the degree noted in this and other studies is simply insufficient to cause any change in the amount of ventilation performed to support a given VO₂.

In terms of CO during restraint, Steinberg cited our echocardiographic study demonstrating no significant changes in CO, measured using echocardiogram parameters, in prone restraint with and without weight force applied.⁷ The author cited Roeggla's finding of significant decreases in CO, but in that study of only six subjects, measurements were performed with a peripheral finger device known to have variable reliability leading to internal data inconsistencies.^{4,8} The author also cited Ho's work demonstrating a reduction in IVC diameter, but no CO measures were obtained and vital signs, including blood pressure and heartrate, remained normal and unremarkable indicating if there was a reduction in CO, it had little to no impact on overall hemodynamic function.⁹ In Krauskopf's study (also cited), investigators reported similar decreases in IVC parameters, but only 'fair' correlation for CO and

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cardiac index and ‘no significant difference between measurements performed with and without weight force application for any of the cardiovascular parameters’.¹⁰

It should be noted sudden deaths in agitated individuals who are restrained have been reported in the medical literature not only in the prone position, but also in supine, sitting and side restraint positions, further casting doubt on Steinberg’s assertion the cause is ‘most likely’ due to acidosis exacerbated by inadequate ventilation and a decrease CO as a result of prone positioning.^{3,11} Well-performed, large epidemiologic studies including some cited by Steinberg have failed to demonstrate an inherent increased risk of sudden death with prone restraint.^{11–13}

We appreciate Dr. Steinberg’s attempt to review the investigative work of others and the medical literature on this important topic. As noted by the author, ‘the actual physiologic cause of death in these circumstances remains uncertain’ and each case is different with many involved variables, requiring specific assessment and review. Any hypotheses or speculative thoughts must be carefully reviewed with clear scientific analysis before drawing broad or generalised conclusions, however Dr. Steinberg’s conclusion cannot be supported by the available data.

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