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Restraint Position and Positional Asphyxia Reay, Donald T. M.D.; Howard, John D. M.D. Seattle, Washington (Reay) Tacoma, Washington (Howard)

To the Editor:

In the September issue of the *Journal*, Chan et al. published a review article on restraint position and positional asphyxia (1). This review resulted from their recent study which assessed ventilatory function in the prone, hog-tied position and its physiologic effects in healthy test subjects. We commend these clinical investigators for their work in giving us a better understanding of respiratory physiology, particularly as it applies to the hog-tied position. Our work in 1988 was an attempt to measure some physiologic effects of position and restraint. The methods employed were less sophisticated and noninvasive compared with those used by Chan et al. We acknowledge that the hog-tied position should be viewed as an inherently neutral position, and although there is a measurable change in ventilatory function, there is no significant physiologic consequence in normal people.

We still have concern regarding deaths that occur during restraint. From the work of Chan et al., we now know that the hog-tied position should not produce serious physiologic consequences. However, during street restraint maneuvers, the totality of events must be considered. In the process of rendering a person helpless to handcuff him or her in a prone position, the involved officers may be required to "pile on" the suspect, pinning the person to the ground with the partial or full weight of the officers and thus compressing and restricting ventilatory function. The physical condition of the person and the circumstances of restraint can make a difference, and each case must be evaluated with a careful reconstruction of events to identify respiratory interference during and after the "takedown" and before the person is restrained. One of us (D.T.R.) was witness to a take-down of an obese man whom the police were trying to control. While compressed on the ground, the man repeatedly complained that he was having difficulty breathing. He was handcuffed and sat upright and stopped complaining. We wonder what the outcome would have been if he had been held face down on the ground until he became quiet. We look forward to the planned research by the San Diego group in assessing the respiratory physiologic effects of weighted compression of the chest. Perhaps, this data will establish parameters regarding how much compression of the chest for how long is necessary to cause serious deleterious effects.

It is our sense that even after the appropriate research on respiratory effects by restraint is completed, there still will be restraint deaths that are not well understood. We are aware of a number of deaths that occurred during restraint which probably were not the result of respiratory interference. As such, causes of death are a matter of conjecture and cover the spectrum of catecholamine rush, neuroleptic malignant syndrome, psychogenic death (including exhaustive mania and excited delirium), and exercise-induced cardiac arrest. Because we have no anatomic markers to help us understand the death, it is important to have as much detail as possible of events surrounding the death. Luke and Reay emphasize this when writing on the perils of death in custody (2). In trying to understand these deaths and move the cause of death from the arena of conjecture into conclusions that are scientifically supportable, there is much similarity with the early investigation of sudden infant death syndrome (SIDS) in which the mechanism of sudden death was thought to be respiratory (3). Some of the early work concentrated on laryngeal spasm and interstitial pneumonia. Now, there is some evidence to suggest neuronal midbrain dysfunction with resulting apnea. Although the anatomic defect in SIDS remains elusive, we have some satisfaction in recognizing what SIDS is not rather than what it is. We now recognize that other factors are associated with SIDS (e.g., cosleeping, cigarette smoking in the residence, maternal drug use). Association does not necessarily indicate a cause, although there is good experimental evidence that the type of bedding on which an infant lays can create a situation for rebreathing of CO₂ and thus cause the death of an infant. Additionally, the association of SIDS with the prone sleeping position was initially viewed with some skepticism but has been confirmed by other carefully performed epidemiologic studies (4).

Using this SIDS experience, we may be able to identify multiple factors associated with restraint and death in custody. These factors include acute drug intoxication, psychosis, obesity, drugs, heart disease, kyphoscoliosis of the thoracic spine, and other preexisting but controlled natural diseases such as diabetes. We cannot summarily dismiss any of these as not playing a role, because there are some experimental animal models for restraint-related mortality. Rats subjected to cocaine and restraint had a 58% mortality compared with 17% for a group with cocaine exposure only (5). The specific physiologic changes may not be known, but the animal study is analogous to human death occurring during restraint and is an important reference point.

This does not solve the problem for the medical examiner charged with certifying the death in custody. Everyone seeks easy answers to complex issues, and this is particularly true in a police custody death. Likewise, we may be able to identify the various factors of a death in custody but not necessarily separate or assign percentages of causality even though something appears to be associated with the death; association does not necessarily indicate causality. The issue of establishing a cause of death is not new. There is a

frequent misconception that if a disease process or condition does not cause death entirely by itself, then it does not count. This restrictive form of reasoning has been argued for a long time and results in the oversimplification of the understanding of any particular death (6). It is more appropriate to make use of deductive reasoning and give consideration to multiple factors at work in causing death. This latter concept is well accepted and is incorporated into the standard death certificate. It is comforting to the pathologist to have a ruptured saccular aneurysm or a ruptured myocardial infarct to identify as cause of death. This is rarely the case in a custody death; during the investigation multiple factors emerge, all of which may have played some role in the death and none of which is convincing enough to stand alone as the undisputed cause of death. In such instances, it may be appropriate as previously recommended (2) to certify the death by a descriptive or situational statement that encompasses identified factors in the death. This seems reasonable. In the absence of any definitive lethal anatomic cause, multiple factors can be recognized and distinguished, but not necessarily separated; nor can a percentage of responsibility be assigned to each factor.

One of the difficulties in studying death in custody is that so few deaths occur in any jurisdiction that well-designed epidemiologic studies are not easily performed. We recommend that a national uniform data collection system be instituted for custody deaths to establish a collective experience that can be analyzed so that the risk factors associated with deaths and restraint can be identified. Until then, we will be left with many preconceived notions about what kills people when they are taken into custody and restrained. Fortunately, our clinical colleagues in San Diego have joined the challenge, and we look forward to their continued research in this vital area.

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