

CASE REPORT**PSYCHIATRY & BEHAVIORAL SCIENCES**

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Physical Restraint and Near Death of a Psychiatric Patient

ABSTRACT: Physical restraint is used as a last resort emergency measure to calm and safeguard agitated and/or aggressive psychiatric patients. This can sometimes cause injuries, and rare fatalities have occurred. One mechanism of injury and death while in physical restraint is that of severe asphyxiation. We present the case of a hospitalized man in his mid-30s, suffering from schizophrenia. The patient was obese. He became aggressive and had to be manually restrained with a “takedown.” After having been put in the prone position on the floor with a significant weight load on his body, he lost respiration and consciousness. Subsequently, he was given CPR. He regained consciousness and respiration, while the cyanosis receded in 1–2 min. Psychiatrists and pathologists should be aware that physically restraining a patient in the prone position with a significant weight load on the torso can, in rare cases, lead to asphyxiation.

KEYWORDS: forensic science, forensic psychiatry, physical restraint, prone position, asphyxiation, schizophrenia

Physical restraint is an emergency measure that sometimes has to be resorted to in order to calm and safeguard an agitated or aggressive patient (1,2). This intervention can have adverse psychological and potentially deleterious physical effects (3,4). The most tragic outcome is the death of the patient (1–3). The suggested mechanism is thought to be asphyxiation (5,6). This is described in the literature variously as restraint asphyxia, mechanical asphyxia, positional asphyxia, and postural asphyxia (7,8). Some have disputed the concept of “positional asphyxia” and argue that factors other than body positioning appear to be more probable determinants for sudden deaths in persons in the prone “hogtie” custody restraint position (9–11). Among the factors that have been suggested to be more important are: illicit drug use, physiological stress, hyperactivity, hyperthermia, catecholamine hyperstimulation, and trauma from struggle.

We present a restraint episode in a psychiatric patient that came close to a fatal end and where the survival of the patient has resulted in information that could shed new light on the topic of asphyxiation during restraint.

The study was approved by the Hospital’s Data Protection Officer and by the Director of the Division of Addiction and Specialized Psychiatry. Written informed voluntary consent was obtained from the patient for publication of this case report.

Case Report

A Caucasian man, in his mid-30s, with a diagnosis of paranoid schizophrenia was being treated at a medium security unit at a

Norwegian University Hospital. From his teens, he had abused various illegal drugs, including amphetamine, cocaine, cannabis, lysergic acid diethylamide, ecstasy (MDMA or 3,4-methylenedioxymethamphetamine), anabolic steroids, and alcohol. He ended school prematurely in order to commit himself to the martial arts and had become proficient. He first came to psychiatric treatment 6 years before the reported incident. The last few years he had gained substantial weight, now being 120 kg. His body mass index was 42. The abdomen was large and protruding. He had dyspepsia and possibly bronchial asthma. The last few months before the incident, his psychotic condition gradually deteriorated. He had persecutory delusions, visual and auditory hallucinations, and anxiety. The regular medication was clozapine 600 mg/day (200 + 400 mg), chlorpromazine 100 mg TID, escitalopram 20 mg QD. On an “as needed” basis he used chlorpromazine 50–100 mg p.r.n. (max 300 mg per day), levomepromazine mixture 100 mg p.r.n. (max 200 mg per day), esomeprazole 20 mg p.r.n., and ipratropium bromide inhalation aerosol 20 µg/dose, 1–2 inhalations maximum three times per day.

Several attempts to reverse his psychotic decompensation, both psychopharmacologically and with regard to the milieu interventions, failed. At the time of the incident, he was in a severe psychotic state with visual and auditory hallucinations, persecutory delusions, anxiety, restlessness, agitation, and thought blocking. Having delusional ideas about another patient in the unit, he walked into this patient’s room in an aggressive and combative state. The staff at the unit immediately intervened whereupon a struggle started. Owing to the patient’s physical strength and vigorous struggling, the “takedown” turned out to be extremely difficult requiring at least 10 persons to keep him under control. The patient had to be placed in the prone “spread-eagle” fashion on the floor in order to prevent harm to others. A member of staff was supporting the patient’s head rotated to the side while simultaneously talking to him and observing his level of consciousness. Two persons held each of his arms and legs, and two had to lie with almost full body weight (about 80 kg

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each) on his torso. After about 2 min from the start of the “take-down,” staff noticed facial cyanosis, arrested respiration, and loss of consciousness, and spontaneous urination. Immediately, the restraint was released. He was turned around and placed in the supine position. CPR, that is, external cardiac massage and mouth-to-mouth breathing was started whereupon the patient after 1–2 min regained consciousness and respiration. The facial cyanosis was gradually replaced with normal skin color. There was a scant amount of vomit on his face. The episode lasted around 3–4 min from being put in the prone position until he had resumed respiration and normal skin color. In the turmoil, staff did not examine the peripheral pulse. The patient was now placed in recovery position, given oxygen on a mask, and pads for a heart-starter were attached to his chest. After about 10 min, paramedics arrived. Examination revealed the patient to be awake but obtunded, respiration was superficial (respiration rate 24 breaths/min). The skin was dry and warm. According to the registration on the heart-starter, the patient had sinus tachycardia with a heart rate of 120/min. The circulation was stable. Auscultation of lungs and heart was normal. Oxygen saturation measured with pulse oximetry in the ambulance was 94%. The patient was brought to the Cardiology Department for further observation and treatment. On arrival, his blood pressure was 140/85 mmHg. The peripheral pulse rate was regular, 110/min. The oxygen saturation was now 95%. ECG showed a sinus rhythm with heart rate 113/min, that is, sinus tachycardia. The QTc interval was 415 msec (normal < 440 msec). Chest X-ray was normal. Blood tests did not give any indication of myocardial infarction or any other physical disease. Blood-lactate was 2.4 mM (normal range 0.7–1.8 mM) indicating hyperlactatemia. Serum clozapine was 2870 nM (estimated normal range 300–2500 nM). The total amount of chlorpromazine equivalents (12) administered the last 24 h, regular and on demand, was 1500 mg. He had an uneventful recovery and was readmitted to the Psychiatric Department the next day. He suffered no physical sequelae.

When interviewed later, the patient said that he could recall all events up to the moment immediately before losing consciousness and, after that, he remembered being transported in the ambulance. He could also recall that while being restrained he had difficulty in breathing and think he said, “I can’t breathe” to the restrainers. The staff taking part in the restraining could not recall hearing this, however. Curiously, the patient denied being afraid or panicking during the episode. A thorough review of his medical history could not substantiate the suspect bronchial asthma. In retrospect, an alternative explanation for his occasional subjective respiratory problems could be anxiety, a symptom that is sometimes wrongly diagnosed as asthma.

Discussion

We have presented a case where the patient was physically restrained in the prone position, with a heavy weight load on his back, and where he subsequently lost respiration and consciousness. The suggestion that the prone position in some circumstances can interfere with respiration causing asphyxia and even death is not new (5,13,14). The term *restraint asphyxiation* was first proposed in 1993 by O’Halloran and Lewman (6). It expanded the concept of positional asphyxia to include the process of subduing and either physically or mechanically restraining an individual and was later suggested reserved for “...deaths during restraint that appear to be the result of chest compression or hogtying” (15, p. 50). Stratton et al. (14) were the first to

report sudden, unexpected deaths in the prone hobble (synonymous with “hogtied”) restraint while being transported by medical staff. Three criteria for defining postural or positional asphyxia have been outlined (14,16). First, the person has to be in a position that interferes with pulmonary gas exchange. This could be caused by the obstruction of the airways or by the restriction of the movements of the chest case and diaphragm. Second, the person must have been unable to escape the position. And last, in case of death, other causes, both natural and unnatural, must be excluded with reasonable degree of certainty. This requires a thorough autopsy.

There are some experimental studies of the effects of prone positioning (17–19). Roeggla et al. (17) found a significant impairment of cardiopulmonary parameters (reduced forced expiratory volume, reduced forced vital capacity, end-tidal carbon dioxide increase, heart rate decrease, systolic blood pressure decrease, diastolic blood pressure decrease, and mean cardiac output decrease) after 3 min in the prone hobble restraint position. However, there was no change in oxygen saturation as measured by pulse oximetry. Chan et al. (18) found a restrictive pulmonary function pattern, but no clinically relevant changes in oxygenation or ventilation, concluding that there was no evidence of ventilatory failure, significant hypoventilation, or asphyxiation as a result of body positioning in the restraint position. However, Chan et al. (18) suggested that individuals with large abdominal girths and body mass index > 30 might be at greater risk of development of a restrictive pulmonary function pattern because of abdominal compression from body positioning. A review by the same group concluded that there was no evidence to suggest that body position alone caused hypoventilation, respiratory compromise, or positional asphyxia in the “hogtie” custody restraint position (10). Responding, Howard and Reay emphasized that “... multiple factors, rather than one single cause, play a role in deaths where restraint has been applied” (20, p. 117), and they later brought attention to the importance of the weight force applied by staff during restraint (21). An experimental study with 50 pounds placed on the subject’s back did not lower forced vital capacity or forced expiratory volume in 1 sec significantly or result in hypoxia or hypoventilation (22). Adding 102.3 kg on the back of the subjects significantly lowered mean voluntary ventilation but was described as clinically insignificant (9). Parkes and Carson (19) placed participants face down and applied the body weight of the restraining persons on the upper torso, with and without a flexed restraint position (“Figure four leg lock” which is similar to the prone “hogtied” restraint position), concluding that some, but not all, prone restraint positions significantly restricted lung function.

In 2000, O’Halloran and Frank (15) reported 21 cases of asphyxial deaths during prone restraint. One of the common elements was pressure applied to the upper torso. Following a discussion regarding the scientific basis of the concept of restraint asphyxia (23,24), a case report described the death of a patient manually restrained in the prone position with a heavy weight applied to his back (25). The autopsy disclosed minor external injuries and old inferior frontal cerebral contusion scars, but no cardiovascular or other organ injuries that could explain death. Miyaishi et al. (26) described the death of a prisoner restrained in the prone position. The autopsy disclosed typical findings of thorax compression with intramuscular hemorrhages on the back and multiple fractures of the ribs. No evidence of neck compression/smothering was found, and it was suggested that the cause of death was compression of the thorax. In sum, the restraint asphyxia theory has been debated, most have sug-

gested that the prone position alone is not sufficient to cause significant asphyxia but have believed it to be a contributing factor. The mechanism of compression asphyxia seems to be less controversial.

Our case is somewhat different from most of the previously published cases. As there were several trained mental health workers present, among them one medical doctor, and the patient survived, we were able to recollect and describe the episode fairly detailed. This gives us a unique basis of information in discussing the different hypothetical causes for the near death occurrence. A range of different diagnoses can be considered in this case. The treating clinicians considered the patient's condition to represent an exacerbation of his paranoid schizophrenia. He was very psychotic with anxiety and agitation, but he did not have fever, fluctuating confusion, neuromuscular symptoms (tremor, myoclonic jerks, and muscular rigidity), or typical catatonic behavior. Although they should be considered, on the basis of the information now available, we feel the following differential diagnoses to be unlikely in this case; acute exhaustive mania, excited delirium, Bell's mania, catatonia, sympathomimetic poisoning, neuroleptic malignant syndrome, central anticholinergic syndrome, and serotonin syndrome.

The immediate cause for the patient's cyanosis and loss of consciousness must have been either asphyxiation (i.e., respiratory arrest), cardiac dysfunction (i.e., cardiac arrhythmia or asystole with reduced cardiac output), or a combination of the two.

Clinical information about his heart function during the episode could have clarified the issue. Understandably, ECG-monitoring during the episode would not have been feasible. Examination of peripheral pulse could have contributed with relevant data. However, carotid pulse examination is an inaccurate method of confirming the presence or absence of circulation (27). There was no indication of heart disease or hypertension prior to the episode. The patient was hemodynamically stable shortly after regaining consciousness. From the limited information available, there were no positive findings indicating a cardiac etiology. Still, we have to consider the possibility of arrhythmia. The patient used four different psychotropic drugs, three antipsychotics (clozapine, chlorpromazine, and levomepromazine) and one antidepressant (escitalopram). Sudden unexpected death in psychiatric patients treated with antipsychotics has been a concern for several decades (28). The cause is believed to be ventricular fibrillation (28). A prolonged rate-corrected QT interval on the ECG (QTc) is a surrogate marker for a predisposition to polymorphic ventricular arrhythmia (torsade de pointes) which can progress to ventricular fibrillation (29). Virtually all antipsychotics can cause QTc interval prolongation (28). Our patient did not have QTc interval prolongation, but that does not completely exclude the possibility of a cardiotoxic effect causing arrhythmias as these may in some cases develop without QTc prolongation (30). The use of several antipsychotics simultaneously in the same patient is far from optimal and ought to be avoided. Nevertheless, this treatment strategy is occasionally used to help nonresponding or poorly responding patients, especially when there is a need to sedate the patient (12). Our patient was on three antipsychotics with relatively strong anticholinergic effects. This could have decreased parasympathetic tone, thereby giving preponderance to the patient's sympathetic tone. Autonomic dysregulation as a result of sympathetic hyperactivity and/or parasympathetic hypoactivity has been associated with arrhythmic effects (31). None of the drugs had direct sympathomimetic effects, but the patient's psychological stress might have caused a catecholamine rush thereby adding to the total

sympathetic tone acting on the heart. Even though we lack information about the patient's cardiac function during the episode, drawing on the data that is available, we consider cardiac arrest or arrhythmia to be less likely.

It is a central question to what degree the patient's respiration was compromised. He was brought flat on the floor in the "spread-eagle" position with two staff members (body weight about 80 kg each) lying on his torso, thereby compressing both his chest and abdomen to the ground. The patient remembered having difficulty breathing just before losing consciousness. Two pathophysiological mechanisms had the potential for causing significant respiratory embarrassment. First, restriction of thoracic cage expansion resulting from the fixed body position with a heavy weight on his torso (19). Second, impaired diaphragmatic movement due to increased intra-abdominal pressure. The patient was obese with a protuberant abdomen. Obesity is considered a possible risk factor for asphyxiation during restraint (24), particularly as it may compromise diaphragmatic motion in the prone position because of excessive fat adding to the increased intra-abdominal pressure. The vomit on the patient's face that was noticed after CPR was terminated indicated regurgitation of gastric contents. This is in accordance with an assumption of a significantly increased intra-abdominal pressure.

Another consequence of increased intra-abdominal pressure could be compression of the inferior vena cava causing reduced venous blood return to the heart and subsequently an impaired cardiac output. This mechanism, the inferior vena cava syndrome, has been suggested as a cause of sudden death during restraint with prolonged external compression of the lower torso (32). An experimental study on healthy, athletic young men demonstrated a negative correlation between the amount of weight applied (gradually increased from 5 to 25 kg) to the lower torso and the diameter as well as the maximum blood flow in the inferior vena cava (32). We cannot exclude the possibility of this mechanism, although ill-defined, having been of importance to the patient in our case.

There are additional factors that might have contributed to the patient's inability to maintain adequate respiration; He was exhausted and fatigued after several days, being driven with a high level of physical activity and minimal sleep. Moreover, the total dosage of antipsychotic medication given was high, probably adding to his fatigue.

There was no clear indication of upper respiratory tract obstruction. The patient's head was placed in the proper position, that is, rotated to the side, while a member of staff kept visual and verbal contact with him until he became unconscious and cyanotic. Neither do we have any reason to believe drug abuse to be a contributing factor. The strict management and surveillance at the unit would have revealed such abuse. However, a toxicological analysis would have been conclusive.

From the available data, we assume that the patient developed asphyxia secondary to the physical restraint. The compression of the torso (i.e., the thorax and the abdomen) was probably more important than the prone position itself in causing the asphyxia. Experimental studies (9,18,22) have suggested that restraint in the prone position alone causes a restrictive pulmonary function pattern, but no clinically relevant changes in oxygenation or ventilation.

The American Psychiatric Association Committee on Patient Safety (33) has warned against restraining a patient in the prone position on the assumption that it can cause "suffocation" (p. 10). The National Institute for Clinical Excellence (NICE) in Great Britain (34) also warned against restraint, but chose not

to present one position as safer than another because “the evidence base surrounding the dangers of positional restraint is weak” (p. 98). The NICE Guideline Development Group believed there were dangers related to restraint in any position and therefore discouraged restraint for prolonged periods in any position.

There have been several reports of sudden, unexpected deaths occurring in restraint situations in the last two decades (2,3,5,6,35,36). This case illustrates a situation where physical restraint in the prone position seemed unavoidable, and almost ended fatally. Close monitoring of the patient’s clinical status, rapid release of the restraint when the patient showed signs of asphyxiation, and immediate implementation of CPR prevented a more negative outcome. Actually, the mere release of the restraint might in itself have been sufficient. In retrospect, it is natural to ask if the episode could have been prevented. We cannot exclude the possibility that using different psychotropic medications and/or having a more strict isolation regime with even more staff present in the immediate surroundings of the patient could have been prophylactic with regard to the patient’s aggression.

A further consideration is monitoring once restraint has been implemented. Masters and Wandless (37) and Masters (38) have advocated the use of portable pulse oximeters to monitor oxygenation during restraint, and it is possible that using such equipment in the present case could have been clinically useful. Our hospital has local guidelines for handling aggression and violence. While there are no national Norwegian guidelines, protocols and guidelines appear to be relatively similar throughout the country, and the main principles appear to be in line with current U.K. and U.S. guidelines (33,34). Although there is little Norwegian legal precedent, we believe that if the patient had died, the manner of death would most probably have been listed as an accident. This case report, we hope, can be a relevant contribution to the discussion of the hazards of physical restraint and the possible causes of fatal outcomes.

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