

## Positional Asphyxia During Law Enforcement Transport

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Three cases of positional asphyxia are described that occurred while victims were in a prone position in rear compartments of police patrol cars. These deaths are attributed to positional asphyxia. Autopsy findings and specific scene and circumstantial correlations of the investigation are discussed with emphasis placed on the limitations of interpretation of the anatomic changes at autopsy.

**Key Words:** Asphyxia—Hog-tied restraint—Positional asphyxia—Death in custody.

We have previously reported our concerns about the deleterious physiologic effects of a restraint maneuver that employs a "hog-tied" prone position (1). This method is commonly employed by law enforcement to incapacitate a suspect and occasionally is used during transport. We have had occasion to study three deaths where this method of restraint was employed by law enforcement while transporting suspects. Autopsy findings in these deaths did not demonstrate anatomic or toxicologic findings sufficient to explain death. Additionally, historical information indicated that the victims were thought to be alive at the time they were placed in the rear seat of police vehicles. Following several minutes of transport, the suspects became quiet and inactive. When they arrived at their destination, it was not immediately determined that each was experiencing respiratory difficulty although each remained motionless. After a few minutes, it was determined that the victims were not breathing and the alarm of a cardiac arrest was sounded. All three deaths are strikingly similar in circumstances and findings. Because of this, we reason that deaths in each instance were the result of adverse physiologic effects created by a semiprone and hog-tied position in a confined space. We report these deaths to alert law enforcement agencies to the potential consequences of this restraint during transport and to share with other death investigators the critical importance of historical information and the mechanics of events necessary to understand this type of respiratory death.

### CASE REPORTS

#### Case 1

The first victim was a 28-year-old white man who, 4 years prior to his death, had been seen for a nonclassified psychiatric illness for which psy-

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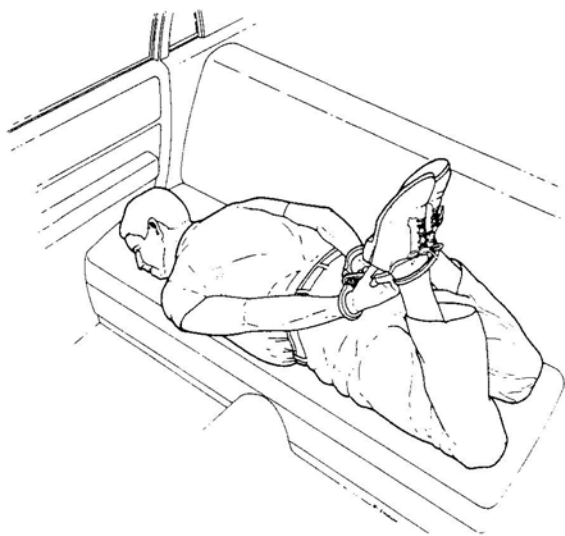


FIG. 1. A sketch of the resting position of the first victim during transport to hospital.

chotropic medications had been prescribed, leading to improvement in his behavior. One month prior to his death, he became more agitated and was again seen for psychiatric evaluation. He was diagnosed as manic depressive. All medications were stopped and he was started on lithium therapy. On the day of his death in the early morning hours, the victim became extremely agitated, and assaulted and threatened to kill his wife. His mother-in-law intervened, but was choked and escaped to a neighbor's house and called for police assistance. Two officers responded to the scene where a violent confrontation ensued. One police officer was choked. The victim was forced to release his hold only after the officer's partner struck him with a nightstick and choked him. The victim subsequently ran from his house and was cornered in the yard when he became trapped between dense bramble bushes and the front end of a law enforcement patrol car. Once cornered, he was overpowered by several police officers who placed him facedown on the ground and handcuffed him in a hog-tied fashion. Flexcuffs immobilized the ankles while handcuffs were used on the wrists. During the restraining maneuver, he continued to resist; once subdued, he became more relaxed, but was still responsive. At no time was it witnessed that a neck hold was applied during the restraining maneuver. The victim was then loaded onto the rear seat of a mid-sized patrol sedan for transport (Fig. 1).

Because of the two nightstick blows to the head that caused lacerations and bleeding, and because of multiple scratches over the body surface sus-

tained when the victim contacted the bramble bushes, a police officer was directed to rush the victim to a local hospital. A high-speed transit ensued that was estimated to have taken 5–7 min. The victim, although not monitored during this period of time, did not show noticeable activity.

Upon arrival at the emergency room, he remained prone when he was removed from the vehicle and transferred to an emergency room stretcher. Nursing personnel reported a faint, slow carotid pulse and spontaneous respirations, but the emergency room physician who saw him within a minute of arrival reported no respiration, poor facial color, and no blood pressure. Restraints were removed and the victim was placed on his back. Vital signs and cardiac telemetry demonstrated cardiopulmonary arrest with asystole. Resuscitation was initiated, which resulted in ventricular fibrillation, eventually converting to a sinus tachycardia. Administration of lidocaine and a dopamine drip resulted in maintenance of this rhythm with a blood pressure of 90–100 mm Hg. His blood lithium levels sampled during this period were determined to be subtherapeutic. The victim remained totally unresponsive during this period, with fixed and dilated pupils. Because there was concern that he might have a head injury, he was transferred to a larger medical center in a comatose, unresponsive condition and was admitted with a diagnosis of anoxic encephalopathy. Cerebral flow studies showed no perfusion and his condition rapidly deteriorated. He died 5 h after admission. The autopsy was performed 68 h after death.

The body at the time of autopsy measured 73 in. (1.85 m) in length and weighed 267 lb (121.36 kg). A noteworthy feature was the abundant abdominal panniculus. A proper assessment of the body habitus was limited because of early postmortem gas distention of soft tissues along with early skin slippage, the result of inadequate refrigeration. The surface examination of the body showed multiple bruises of the extremities and back and there were two gaping lacerations of the occipitoparietal scalp. In addition, multiple scratches were present over the surface of the extremities. All of these findings corroborated the historical information that this man had been struck with nightsticks and had become entangled in bramble bushes. Other noteworthy findings at the time of autopsy included a few petechial hemorrhages in the conjunctiva of the right upper eyelid with a single flame-shaped hemorrhage in the inner canthus of the left lower eyelid. Internal examination showed that there was no injury to the cranial cavity and brain except for subga-

leal hemorrhages attributed to impacts to the head. The brain further demonstrated some features suggestive of cerebral edema. Layered dissection of the neck was essentially free of any injury except for a small 1.3 cm area of bruising in one submandibular gland. There was mild to moderate focal coronary arteriosclerosis and a heart weight of 415 g. Moderate fatty change of the liver was likewise noted.

Toxicologic examination of antemortem blood showed the presence of 0.2 mEq/L of lithium (therapeutic level, 0.8–1.2 mEq/L). Lidocaine and caffeine were also detected, but no other drugs were demonstrated. Death was attributed to positional asphyxia.

### Case 2

The second victim was a 28-year-old healthy white man who was house sitting and drinking beer with his brother most of one afternoon. A small group of men arrived and tried to enter the house. A verbal confrontation took place and the group left. Later, the brothers went out to their van for a trip to the store for more beer. They noticed their van had been tampered with and began shouting at each other about the problem. Neighbors assumed they were fighting and called police.

Two police officers arrived and tried to calm the brothers and get them to go into their house. During this time, a records search found outstanding traffic warrants on the victim. The victim would not quiet down and became increasingly agitated. When faced with the option of going into the house or being arrested, he ran. A pursuit and struggle ensued. He was struck several times with nightsticks, once to the head. After the victim was partially subdued prone on the ground, a witness to the event ran out had held the victim's legs. Several officers arrived to help restrain and hog tie the victim. The witness heard the victim say during the struggle on the ground "gimme some air, gimme some air." The pursuit and struggle lasted ~7 min. There is no evidence that a neck hold was used at any time during the confrontation.

When the victim was at last restrained, and while still resisting and complaining, he was placed in a prone position in the back of a patrol car on a narrow, molded plastic, one-piece seat. The transporting officer left as soon as the victim was in the car. An officer at the scene called for an aid unit to meet them at the jail in order to treat the head injury.

While en route to the jail, the victim slipped down and became wedged between the front and back

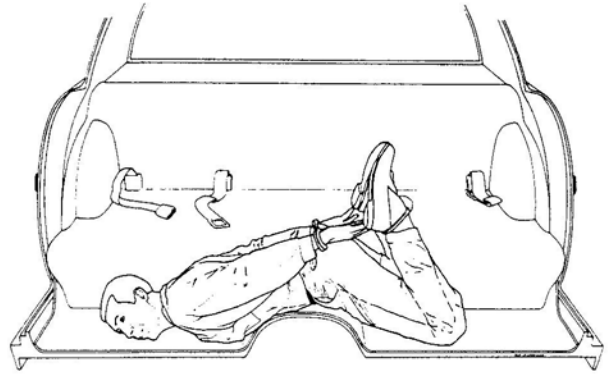


FIG. 2. A sketch of the final resting position of the second victim on the floor of the police vehicle used for transport to hospital.

seats with his left shoulder partway up the back of the front seat and his right shoulder against the bottom panel and foot well of the back seat (Fig 2). The transporting officer recalled that the victim had been "rustling" around a little bit and that his breathing had been raspy. About 3 min later, his breathing was "gurgly" and the transporting officer called a Code 3 upgrade to paramedics. Medics arrived at the jail about the same time as the officer and victim. Approximately 4 min had elapsed during the trip from the scene to the jail. The victim was unresponsive when removed from the patrol car. Despite all efforts, he never regained any vital signs. He was pronounced dead 26 min after CPR had been initiated.

At autopsy, the body measured 70 in. (1.78 m) and weighed 220 lb (99 kg). Deep neck muscle hemorrhage was present adjacent to the left carotid artery and structures of the neck. There was some vomitus in the airway. Other injuries included laceration and contusion to the right parietal scalp attributed to a nightstick blow. Other abrasions and soft tissue contusions to the body surface and extremities were also present and attributed to multiple nightstick blows. Also noted were minor circumferential abrasions and contusions to both wrists and one ankle from restraints. No petechial hemorrhages of the conjunctivae were noted. Findings in all thoracic and abdominal organs were within normal limits without significant anatomic change to account for death.

Toxicologic tests found a blood alcohol level of 0.12 g/100 ml, LSD blood level of 3.2 ng/ml, THC blood level of 4.1 ng/ml, and THC blood metabolite level of 108 ng/ml. No other drugs or chemical findings of note were present. Death was attributed to positional asphyxia.



FIG. 3. Arresting officer demonstrating approximate position of decedent in back seat of patrol car. Note that the lower portion of the body still remains on the seat. The legs have not been tied and flexed as occurred during the actual transport of the victim.

### Case 3

The third victim was a 34-year-old black man with a long psychiatric history of undifferentiated schizophrenia, who had stopped taking his medication on his own sometime prior to the described incident. On the day of his death, he began to show bizarre behavior in the home of his grandmother, including wandering about talking to himself and sticking his head in an oven and attempting to turn on the gas. The grandmother called the police. The victim apparently became quite agitated upon the arrival of the police officer and a verbal confrontation, followed shortly thereafter by a physical confrontation, took place. A backup patrol car was summoned. A scuffle took place inside the grandmother's home with one police officer admitting to several fistful blows to the decedent's chest and abdomen, but not to use of a baton or a neck hold. The victim was wrestled to the ground, whereupon his arms were placed behind his back, two sets of handcuffs were applied, and leg straps were placed around his ankles. He was taken to the waiting patrol car and placed into the back seat with his head, shoulders, and chest in the foot well behind the driver's seat and his right flank over the drive shaft hump. His legs were in a flexed position on the rear bench seat behind the front passenger seat (Fig. 3). The arresting officer got in the back and rode with the victim to the city jail, which was ~5–7 min away. The officer stated that the victim became quiet, but nothing was noted to be wrong until their arrival at the receiving dock at the city jail, at which time the arresting officer said to his partner, "I think he's dead." The victim was removed from

the vehicle and emergency response personnel pronounced him dead at the scene.

Upon the arrival of the Medical Examiner (A.D.S.) at the scene, the deceased was lying on his side on the concrete floor with his hands cuffed behind his back by two sets of handcuffs. The legs were tied together at the ankles. The body was immediately removed for a postmortem examination.

At postmortem examination, the body measured 68 in. (1.73 m) in length and weighed 150 lb (67.5 kg). Insignificant injury to the head consisted of two small abrasions to the skin of the left side of the face and a small abrasion of the right side of the nose. Two minor contusions of the scalp were noted. Petechiae were noted in both eyes and on the pleural surfaces of both lungs. No abnormalities other than congestion were noted. Complete microscopic examination showed no histologic abnormalities. Results of toxicologic analysis of body fluids including vitreous fluid were within normal postmortem limits. Death was attributed to positional asphyxia.

### AUTOPSY FINDINGS AND PHYSIOLOGIC CONSIDERATIONS

The conclusions drawn from these investigations were based primarily on the reconstruction of events surrounding each death. Complete detailed autopsies showed limited findings that did not enable any anatomic cause of death to be established. In two of the victims, cutaneous injuries were compatible with impacts by nightsticks. There were scattered bruises over the bodies and lacerations of the scalp. In the first victim, considerable attention was given to the neck dissection since conjunctival petechial hemorrhages were present and there was evidence that the victim had been choked early in the confrontation. However, historical accounts of the choking episode indicate that it occurred 15–20 min before the victim was subdued. Multiple witnesses denied that choking occurred during the "take-down" and placement of restraints. Additionally, the victim had received vigorous chest compression during resuscitation. The second victim had no physical or historical evidence of cervical compression. Conjunctivae were congested, but free of petechiae. Neck compression, although considered to have been possible, could not be supported by historical accounts of witnesses. Hemorrhage in the soft tissues of the left side of the neck was probably impact injury from a nightstick. The third victim showed minimal cutaneous abrasions, but did show

conjunctival petechiae. Again no cervical injuries were present and no historical information to suggest choking was elicited. The presence of conjunctival petechiae appears to have resulted from the head placed at a lower level than the rest of the body. This would increase venous pressure in the head and promote petechiae. In all three victims, no significant natural disease process was present that could account for an independent "cardiac event."

Toxicologic analyses of the body fluids of two of the victims demonstrated subtherapeutic levels of lithium in one, while in the other there was evidence of alcohol, LSD, and THC. These findings were considered significant only in that the erratic and violent behavior of those two victims had some explanation. Subtherapeutic lithium levels in the manic-depressive illness of the first victim could account for an emergence of manic behavior. In the second victim, the presence of alcohol, LSD, and THC provides a reasonable explanation for irrational, violent, and uncontrollable behavior. In these two instances, toxicologic findings shed some light on the decedents' behavior, but do not explain their deaths.

Positional asphyxia occurs when the position of the body interferes with respiration, resulting in asphyxia. The deleterious positional effect may result either from interference with the muscular or mechanical component of respiration, from compromise of the airway, or from some combination of these. In addition, in all cases of positional asphyxia, one or more contributory factors provide an explanation for the inability of the victim to correct the deleterious and potentially lethal position, for example, alcohol/drug intoxication, concussive head injury, entrapment, restraint, or physical disability. In our view, the application of hog-tied-type restraints and subsequent positioning of the victim in a confined space are the critical factors that led to the respiratory compromise that caused these men's deaths. Hence, all three men died as a result of *positional asphyxia*. This conclusion is drawn from the totality of the investigation and depends on historical information and reconstruction of events. These deaths can only be properly evaluated with knowledge of the dynamics of the events preceding and surrounding death. In the first victim, obesity with a large abdominal panniculus interfered with respiration in the prone position in a confined space, whereas, in the other two deaths, mechanical displacement of the abdomen by the convex contour of the floor and the edge of the car seat accounted for loss of effective diaphragmatic

excursions of respiration. In each instance, the final fatal event was hypoxia.

## DISCUSSION

Respiration depends on three critical elements: the gas exchange function of the lungs, the patency of the airway, and the muscular pump or bellows that ventilates the lungs. All three are vital to life. The parenchyma of the lung, if severely diseased or damaged by injury, results in failure of the gas exchange function and hypoxemia. If the airway is obstructed at any level, a similar consequence will ensue. The same result occurs in pump or bellows failure where the lung may be healthy and the airway patent, but the mechanical muscular bellows of the chest fails. Pathologic causes of respiratory bellows failure are detailed in the medical literature, but respiratory failure attributed to adverse body positioning has not been scrutinized (2). The three deaths in this report are the result of disturbed physiology because of failure of the respiratory bellows produced by an adverse position in a confined space.

The respiratory bellows or pump depends upon the output drive of central nervous respiratory centers that control respiratory muscle activity. This neural impulse center may not be capable of responding to oxygen demand. Such failure occurs when these respiratory centers are chemically depressed as in drug intoxications, for example with barbiturates or opiates. In these instances, the central nervous system drive is attenuated or dampened and fails to respond to biochemical demands of the body. Unless life is artificially supported, death occurs rapidly. Since the higher centers of cerebration are also affected by drug intoxication, the intoxicated person may be unaware of impending death. Only careful blood-gas monitoring and artificial support of respiration can overcome the deficits in central nervous system drive to maintain respiration and life.

Failure of the bellows or the pump function of respiration can result from a mechanical abnormality of the thorax that impedes a proper bellows action. A flail chest or severe chest deformity can cause bellows failure and, ultimately, respiratory failure and death. A nonfunctioning or poorly synchronized respiratory bellows causes severe biochemical disturbances. Since such anatomic abnormalities can be readily identified, the pathophysiology leading to hypoxia, hypercapnia, and death is easily understood and generally accepted.

Bellows failure can also occur from respiratory

muscular failure. Muscles of respiration may be unable to contract and generate the motion of respiration even though the central nervous system drive is functioning and the thorax is structurally intact. Physiologic inspiratory muscle fatigue can occur (3). The exact cause of inspiratory muscle fatigue has yet to be clearly defined, but there are other recognizable clinical conditions such as myasthenia gravis and other neuromuscular disorders causing muscle dysfunction, in which respiratory muscle fatigue results in hypercapnia and its attendant biochemical consequences (4). This is rarely a cause of sudden death, since the process, although insidious, is generally clinically recognizable and treatable.

There is an additional cause of bellows failure that has not been cited in the clinical or forensic literature, but is illustrated in the three deaths that form the basis of this report. Here the restrained position of the victims in a confined space creates a circumstance for bellows restriction and failure.

The chest wall depends on the interaction of the diaphragm with the musculature both of the rib cage and the abdomen. Breathing takes place by displacement of either the abdomen or the rib cage (5). The total volume displaced in the abdomen by the rib cage bellows action is the total volume of change produced during inspiration in the lungs. When the muscle of the diaphragm contracts, the dome of the diaphragm displaces abdominal viscera downward. Muscles of the diaphragm, in addition to displacing abdominal contents, also act on the costal attachments to lift the rib cage in a respiratory movement. Contraction of the intercostal muscles during inspiration expands the rib cage. If respiration depends totally on the intercostal and accessory muscles of the neck, as when the phrenic nerve is severed and the diaphragm is paralyzed, the bellows action of the thorax displaces the abdomen inward so that abdominal volume is displaced upward and reduced. Any increase in the lung volume is offset by abdominal displacement due to inward volume change. Roussos and Macklem detailed these observations in their report on the respiratory muscles, an excellent review of respiratory muscle physiology to which the reader is referred (6). In the normal supine person, breathing is primarily the result of diaphragm muscle activity. The excursion of the chest during respiration in a supine position is the result of diaphragm muscle contraction. On the other hand, a normal person in the erect position uses both the intercostal chest muscles and the diaphragm. Consider the adverse effects of the prone position in breathing, particularly where the abdominal panniculus is so large

that it displaces the abdominal volume or when the abdominal volume is displaced by an object. The consequence is that the effective abdominal excursions produced by contraction of the diaphragm are reduced and the tidal volume of respiration is substantially reduced. If uncorrected, this leads to hypercapnia and hypoxemia.

Roussos and Macklem point out that, as the work of breathing becomes very difficult, many muscles in the arms and trunk and neck are recruited and contribute to the total oxygen utilization of breathing. The specific role of the hog-tied restraint posture and its biochemical effects on those muscles is unclear at this time, although it is obvious that any restraint that prevents a change of position could restrict breathing further by preventing those muscles from assisting in respiration.

Another, more ill-defined, factor that is likely to augment respiratory muscle fatigue is related to prior violent muscular activity. Weakness of muscles due to hypercatabolic states occurs with sepsis or when long surgical procedures cause increased energy demands (7). During the violent confrontations that ensued in these instances that we report, the expenditure of muscular energy by each victim was probably substantial. Energy that is expended by the contractile muscle machinery of the body is subtracted from the respiratory muscle needs. Muscle fatigue may induce the central nervous system to shunt energy to contracting muscles. A deficit in energy supply to respiratory muscles can influence their performance. A decrease in chemical energy supply to respiratory muscles will hasten their failure as well as the failure of other muscle groups (8). All three victims were rendered more vulnerable to respiratory muscle fatigue through deficits of energy created by violent muscle activity before they were placed in their final restrained positions.

#### OTHER CONSIDERATIONS

The diagnosis of positional asphyxia should be considered when (a) circumstances surrounding death indicate a body position that could interfere with respiration; (b) historical information indicates "difficulty in breathing" or unusual physical respiratory signs such as cyanosis, gurgling, gasping, or any other physical manifestations that could be interpreted as evidence of respiratory distress; (c) there is absence of significant or catastrophic anatomic pathologic changes at autopsy that would conclusively account for death, including such catastrophic events as intracerebral hemorrhage or ruptured myocardial infarct; and (d) clearly toxic or

fatal levels of drugs or chemicals that are ordinarily incompatible with life (including carbon monoxide, cyanide, and lethal levels of therapeutic and abused drugs) are absent from body fluids.

During the evaluation of deaths such as those reported here, sudden cardiac death without pathomorphologic changes must be considered. A relationship between stress and death is known to exist, but there is a dearth of factual data because of the unpredictability and suddenness of such deaths (9). Certainly primary sudden cardiac death without pathomorphologic changes is a well-recognized entity. However, in view of the pathophysiology of the respiratory disturbance created by respiratory restriction, it is likely that any fatal cardiac event would be precipitated by the adverse respiratory physiology produced by respiratory restriction. Since it is known that biobehavioral stresses can augment cardiac susceptibility to ventricular fibrillation (10), one might contend that these deaths were caused by a malignant ventricular arrhythmia triggered by the psychological events and is unrelated to and independent of the position of the victim. Ventricular fibrillation has been reported spontaneously and has been triggered by psychiatric interviews, by stressful emotions, and during REM sleep stages (11). However, recognizing that biobehavioral factors predispose to sudden cardiac death does not allow for the conclusion that the deaths reported here are the result of such factors. There are no pathomorphologic changes that enable differentiation between death induced by psychobehavioral factors and the pathophysiologic disturbance created by body position. Until such time as we have a means of differentiating between these two physiologic events, it is our view that the best explanation for such deaths is positional asphyxia, which is supported by circumstantial events.

Another possible cause of death that needs to be considered, especially when sudden death occurs in psychiatric patients, is the so-called neuroleptic malignant syndrome (NMS). In the late 1800s, the diagnosis of "acute exhaustive mania" was given to sudden death of psychotic patients from apparently natural but undetermined cause. This condition is poorly understood and may be related to a "cardiac event" due to psychological stress as described above. NMS is a hyperpyretic syndrome that is frequently fatal and occurs in people who are taking antipsychotic agents, which include phenothiazines, butyrophenones, thioxanthenes, and other drugs (12). A drug's potential for inducing this syndrome appears to parallel its antidopaminergic potency. Additional predisposing factors include physical exhaustion, dehydration, organic

brain disease, and the use of long-acting depot neuroleptics (13). Classic features of NMS are hyperthermia, hypotonicity of skeletal muscles, and fluctuating consciousness along with instability of the autonomic nervous system. NMS has been seen in psychiatric and medical patients given neuroleptic drugs and is not related to previous exposure to the drug or to toxic overdoses. However, NMS may also occur in the absence of the administration of neuroleptic-type drugs and appears to be indistinguishable from the poorly understood "acute exhaustive mania" described prior to use of neuroleptics (14). Sudden phenothiazine-related deaths that lack the other distinguishing features seen in the typical NMS death have also been described (15). A history of neuroleptic drug use or the other clinical features of NMS in an unexpected death might suggest that this syndrome is the most likely cause of death. Because neither NMS nor positional asphyxia show definitive pathologic/toxicologic findings, positional asphyxia and neuroleptic malignant syndrome are not mutually exclusive and may coexist. Each death must be investigated not only by examining the morphologic features at autopsy and the toxicologic results, but also by evaluating the victim's medical history in conjunction with the scene and circumstances surrounding death. Without a consideration of all these factors, it is likely that some deaths due to positional asphyxia will go unrecognized.

There will be deaths, particularly those where cocaine, methamphetamine, and high levels of alcohol are present, in which it becomes tempting to assign the cause of death solely to these intoxicants, and to ignore or discount the final position in which the victim was found dead. In a previous report on cocaine deaths, Wetli and Fishbain (16) cite instances where victims were found dead in the back seat of police cars and in other unusual circumstances where there was the potential for the position of the body to adversely affect respiration. Whether any of these deaths had an unrecognized pathologic respiratory contribution because of body position is unknown. Such assessments must take into account the total death investigation and not focus on anatomic findings and toxicologic values without due consideration of the resting position of the victim.

The same dilemma occurs in deaths where significant, but not necessarily fatal, natural disease exists. Finding advanced coronary artery disease, cirrhosis, and even diabetic ketoacidosis in a person who is restrained in a position that can compromise respiration may tempt the death investigator to attribute death solely to the natural disease process and discount any contribution to death by the po-

sition of the body. Again, we do not know of a clear or direct way to settle such issues. Because the positional asphyxia diagnosis is primarily based on scene and historical information, it is not often an unequivocal diagnosis in any given death investigation. Nevertheless, our view is that positional asphyxia should be seriously considered whenever unusual positional stress has been placed on respiration. Until postmortem methods are developed to better assess physiologic disturbances, the ultimate judgment will be in the hands of the examining pathologist. Because of the complexity of the issues involved, there certainly will be a spectrum of opinions and no clear answer. The ultimate and difficult assessment as to cause of death is in the hands of the certifying physician who is left to make a judgment that incorporates the bias and prejudice inherent in each of us, but is also based on the important principle of forensic pathology which requires that each death be evaluated using all available information, especially historical and scene investigative material, rather than relying solely on anatomic-toxicologic information. □

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